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JULY 1968
VOLUME 3, NO. 7
5666–6665

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Volume 3, No. 7 Abstract and Citation Nos. 5666-6665

GASTROENTEROLOGY ABSTRACTS AND CITATIONS

A monthly publication of the
National Institute of Arthritis and Metabolic Diseases

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis and Metabolic Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication of GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available, free to qualified interested investigators and practitioners, citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology has accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis and Metabolic Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

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Use of Funds for printing this publication
approved by the Director of the Bureau of
the Budget on October 7, 1968.

For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402
Subscription price per year in the United States, Canada, and Mexico, $8.50; foreign $2.25 additional, $1.00 per single copy. Payment is required in advance, and check or money order should be made payable to the Superintendent of Documents.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans
Ar.	Arabic
Bul.	Bulgarian
Ch.	Chinese
Cz.	Czech
Dan.	Danish
Dut.	Dutch
E.	English
Eston.	Estonian
Fin.	Finnish
Fl.	Flemish
Fr.	French
Ger.	German
Gr.	Greek
Heb.	Hebrew
Hun.	Hungarian
Ic.	Icelandic
In.	Indonesian
It.	Italian
Jap.	Japanese
Kor.	Korean
Latv.	Latvian
Lith.	Lithuanian
Nor.	Norwegian
Pol.	Polish
Por.	Portuguese
Rum.	Rumanian
Rus.	Russian
Ser.	Serbo-Croatian
Sl.	Slovak
Sp.	Spanish
Sw.	Swedish
Th.	Thai
Turk.	Turkish
Uk.	Ukrainian
Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone
ADP	adenosine diphosphate
AMP	adenosine monophosphate
ATP	adenosine triphosphate
BSP	sulfobromophthalein
C	degrees centigrade
cm	centimeter(s)
CNS	central nervous system
cpm	counts per minute
DNA	deoxyribonucleic acid
e.g.	for example
g	gram(s)
μf	microgram(s)
hr	hour(s)
i.m.	intramuscular
i.p.	intraperitoneal
IU	international unit(s)
i.v.	intravenous
kg	kilogram(s)
LD$_{50}$	median lethal dose(s)
LDH	lactic acid dehydrogenase
m	meter(s)
M	molar
mEq	milliequivalent(s)
mM	millimolar
μM	micromolar
mC, μC	milli-, microcurie(s)
mg	milligram(s)
min	minute(s)
ml	milliliter(s)
mm	millimeter(s)
MTD	maximum tolerated dose
ng	nanogram (10^{-9})
pg	picogram (10^{-12})
p.o.	orally
ppm	parts per million
r	Roentgen
RBC	red blood cells (erythrocytes), red blood count
resp.	respectively
Rev.	review (only in citations)
RNA	ribonucleic acid
s.c.	subcutaneous
sec	second(s)
SGOT	serum glutamic-oxalacetic transaminase
SGPT	serum glutamic-pyruvic transaminase
U	unit(s)
UV	ultraviolet
WBC	white blood cells (leukocytes), white blood count
yr	year(s)

PRE-CLINICAL SCIENCES

GENERAL

5666 INTESTINAL PHOSPHATASE AND FAT ABSORPTION. *(E.)* Warnock, M. (Dept. Path., U. Chicago, Ill.). *Proc Soc Exp Biol Med* 129(3):768-772, 1968.

After administration of a meal containing 56 g of fat, serum alkaline phosphatase of intestinal origin was demonstrated electrophoretically in 14 healthy subjects (blood type A or nonsecretors of ABH) who were lacking it in the fasting state. Other subjects ingesting skim milk or glucose did not show the same response. A dilution technique is presented which showed that 2-3 mU/ml of phosphatase activity can be demonstrated electrophoretically in types of subjects in whom phosphatase has not been found by other authors. The most important variables in this technique are the buffer used to make the gel (Tris-borate buffer) and the conditions employed during staining. The importance of a controlled diet in studies of intestinal phosphatase is emphasized.

5667 STUDIES ON GASTRIC BLOOD FLOW IN RABBITS USING THE RADIOACTIVE CLEARANCE METHOD. I. BASIC STUDIES. *(Jap.)* Shimizu, M. (Tokyo Med. Coll., Japan). *Tokyo Ika Daig Z* 26(1/2):77-84, 1968.

Gastric mucosal blood flow was measured in white male rabbits by a modification of Kety's Na ^{131}I clearance technique (*Amer Heart J* 38:321, 1949). The proportion of the radioactivity cleared was measured 10 and 20 min after the injection of 0.1 ml of Na ^{131}I solution (10-20 µC) into the subserosal layer of the stomach wall. In nonanesthetized animals, the clearance rates were 47.5 ± 5.1% (10 min) and 65.8 ± 3.8% (20 min). In animals anesthetized with urethan (1 g/kg), the clearance rates and body temperature both decreased so significantly that control values could not be obtained. Animals anesthetized with phenobarbital (30 mg/kg) had clearance rates of 44.4 ± 3.8% and 62.3 ± 4.2%, resp. at 10 and 20 minutes. In these animals, clamping the portal vein and the vena coronaria epigastrica significantly decreased the clearance in the initial 10 min, while clamping of the splenic vein produced no change, indicating that a large proportion of the gastric blood flows in the former veins. Clearance rates for the pyloric region were 55.5 ± 6.3% and 66.4 ± 6.3%, resp. at 10 and 20 min, which were significantly higher than those for the corpus.

5668 STUDIES ON GASTRIC BLOOD FLOW IN RABBITS USING THE RADIOACTIVE CLEARANCE METHOD. II. EFFECTS OF VARIOUS AGENTS AND OF THE AUTONOMIC NERVOUS SYSTEM ON GASTRIC BLOOD FLOW. *(Jap.)* Shimizu, M. (Tokyo Med. Coll., Japan). *Tokyo Ika Daig Z* 26(1/2):85-92, 1968.

The Na^{131}I clearance technique (S. S. Kety, *Amer Heart J* 38:321, 1949) was used to evaluate the effect of epinephrine (10 µg/kg), norepinephrine (10 µg/kg), neostigmine (75 µg/kg), histamine (1 mg/kg), chlorpromazine (1 mg/kg), hyoscine-N-butylbromide (1 mg/kg), prednisolone (5 mg/kg), ACTH (5 U/kg), vagal stimulation (4 volts for 10 seconds), vagotomy, splanchnic nerve stimulation (4 volts for 10 seconds) or splanchnicectomy on gastric mucosal blood flow in anesthetized rabbits (30 mg/kg phenobarbital). Whereas significant increases in gastric blood flow were observed after epinephrine, chlorpromazine and hyoscine-N-butylbromide, significant decreases were seen after norepinephrine, histamine, ACTH or stimulation of the splanchnic nerve. Slight increases were produced by either vagotomy or vagal stimulation, and a slight decrease was produced by neostigmine. There was no change in gastric blood flow after prednisolone or section of the splanchnic nerves, and histamine produced no change in pyloric blood flow.

5669 AN ELECTRON MICROSCOPIC STUDY OF THE LOCALIZATION, IN MOUSE LIVER, OF POLYVINYLPYRIDINE-N-OXIDE AND POLY-p-DIMETHYLAMINOSTYRENE-N-OXIDE, SYNTHETIC POLYMERS WITH A PROTECTIVE EFFECT AGAINST SILICOSIS. *(It.)* Bairati, A. (L. Devoto Clin., U. Milan, Italy) and P. Castano. *Med Lavoro* 59(2):81-104, 1968.

When 2 synthetic polymers which are known to protect macrophages against the cytotoxic effects of silica (poly-p-dimethylaminostyrene-N-oxide and polyvinylpyridine-N-oxide) were administered to Swiss-Albino mice, either at 1.5 mg/week s.c. to a total of 30 mg in 5 months or at single doses of 2 mg, electron microscopy of ultrathin liver sections prepared 1-30 days after administration of the polymers failed to reveal any isolated polymer molecules. However, the Kupffer cells contained numerous granular vacuoles, not seen in control livers, which are interpreted as phagosomes or segregating vacuoles containing varying amounts of the polymers. No such vacuoles were ever seen in the hepatocytes, but electron-opaque granular material was also seen in the intercellular spaces. There were no submicroscopic alterations in cellular morphology in either the Kupffer cells or the hepatocytes. The granular material in the vacuoles and intercellular spaces had the same electron opacity and staining properties as the ribosomes, although there were small differences in the ultrastructure of the vacuoles depending on which polymer had been administered. It is concluded that polymer molecules are pinocytosed by the Kupffer cells and stored in the phagosomes, where they aggregate to form a granular material. Contact between silica and the polymers probably takes place both in the phagosomes and in the intercellular spaces, where the polymers prevent the action of silica on the external cellular membrane.

5670 EXTRAVASCULAR SHUNTING OF OXYGEN IN THE SMALL INTESTINE OF THE CAT. *(E.)* Kampp, M. (Dept. Clin. Physiol., U. Gotëborg, Sweden), O. Lundgren and N. J. Nilsson. *Acta Physiol Scand* 72(4):396-403, 1968.

An attempt was made to differentiate between intravascular and extravascular shunting by comparing, at various levels of vascular smooth muscle tone, the venous appearance time of oxygen with that of red blood cells after injection into the superior mesenteric artery of 8 cats. Under resting conditions, intra-arterially injected red blood cells labelled with methemoglobin appeared in the venous circulation 1-2 sec later than did oxygen administered in a similar manner. This is taken to indicate extravascular shunting (countercurrent exchange) of oxygen between the ascending and descending limbs of the mucosal vascular loops, which are located mainly in the villi. No difference between the venous appearance time of red blood cells and oxygen could be demonstrated during either vasodilation induced by isopropylnorepinephrine or vasoconstriction induced by stimulation of the regional sympathetic fibers, indicating that extravascular shunting of O_2 is abolished under these conditions.

5671 **ACUTE PORTAL HEMOCONCENTRATION IN ENDOTOXIN SHOCK: SEARCH FOR AN EXPERIMENTAL SHOCK MODEL.** *(E.)* Boruchow, I. B. (U. Pennsylvania Med. Sch., Philadelphia) and R. M. Abel. *Arch Surg* 97(4):666-671, 1968.

In order to elucidate the role of the spleen and of portal hypertension in canine endotoxin shock, intact or splenectomized dogs were given a median lethal dose of *Escherichia coli* endotoxin, after which portal venous pressure changes and hematocrit values were compared with those in the arterial circulation. Acute portal hemoconcentration was noted in both intact and splenectomized dogs, but the effect was less in the latter. This phenomenon is related both to epinephrine-mediated contraction of the splenic red-blood-cell reservoir and to the transudation of fluid from the portal circulation during acute portal hypertension. The first mechanism is abolished by splenectomy, while the 2nd is abolished by splenocaval shunting. These data support the view that the spleen does not serve the same reservoir function in the human as in the dog, and that splenectomy should probably be included as part of the canine experimental shock model.

5672 **AGE-RELATED TRENDS OF THE AMYLASE, GLYCOPROTEIN, LIPOPROTEIN AND SERUM PROTEIN IN HUMAN BLOOD.** *(E.)* Das, B. C. (Biometry Res. Unit, Indian Statist. Inst., Calcutta). *Exp Geront* 3(2): 159-170, 1968.

The effects of age, height and weight on serum protein, glycoprotein, lipoprotein and amylase levels were investigated by multiple regression analysis of the results of electrophoretic mobility studies on blood samples from 317 adult males and 97 females, 17-76 yr of age. The protein components identified electrophoretically were: α-, β_1-, β_2- and γ-amylase; α-, β- and γ-glycoprotein; α- and β-lipoprotein; albumin, and α_1-, α_2-, β_1-, β_2- and γ-globulin. Significant age-related trends were observed for α_1- and γ-serum globulin, β-lipoprotein and γ-amylase, and the levels of both α- and γ-amylase were found to vary with the body weight. There were no significant effects due to age or weight on the glycoprotein components.

5673 **THE ROLE OF GASTRIC MUCOSAL MAST CELLS DURING TRITON WR-1339 LIPEMIA OF RATS.** *(E.)* Haikonen M. (2nd Dept. Pathol., U. Helsinki, Finland), T. Räsänen and E. Taskinen. *Acta Physiol Scand* 74(3):368-371, 1968.

In experiments involving 36 male Sprague-Dawley rats, i.p. administration of Triton WR-1339 (either 1 x 50 mg or 3 x 50 mg at 12-hr intervals) produced a steep increase in both esterified fatty acids (900% and 2600%, resp.) and a decrease in plasma proteins. In the gastric and jejunal walls of rats sacrificed 4-5 hr after Triton administration, the mucosal mast cell count and the metachromatic granulation of these cells increased significantly, after an initial decrease. The lipemic effect of Triton WR-1339 is attributed to inhibition of heparin liberation from the mast cells and consequently of lipoprotein lipase activity, resulted in an accelerated conversion of fatty acids to the protein-bound form.

5674 **STUDIES ON THE IMMUNOBIOLOGICAL SIGNIFICANCE OF THE SOLUBLE LIVER PROTEINS.** *(Ger.)* Meyer zum Büschenfelde, K. H. (2nd Med. Clin., U. Mainz, Germany). *Z Ges Exp Med* 148(2):131-163, 1968.

5675 **RELATIONSHIP BETWEEN LIVER SIZE AND BODY SIZE.** *(E.)* DeLand, F. H. (Johns Hopkins Med. Inst., Baltimore, Md.) and W. A. North. *Radiology* 91(6): 1195-1198, 1968.

5676 **INTESTINAL ABSORPTION AND DIGESTION OF PROTEINS AND LIPIDS WITH PARTICULAR ATTENTION TO THE DUODENUM: AN ENDOLUMINAL STUDY DURING TRANSIT OF A TEST MEAL IN A NORMAL SUBJECT.** *(It.)* Azzaroli, P. (Inst. Spec. Med. Path., U. Bologna, Italy), G. Fontana, G. A. Lanfranchi and S. Ferri. *Arch Pat Clin Med* 44(6):491-507, 1968.

5677 **REGULATION OF FEED INTAKE IN RUMINANTS.** *(E.)* Baile, C. A. (Harvard Sch. Publ. Hlth., Boston, Mass.). *Fed Proc* 27(6):1361-1366, 1968.

5678 **HYPOTHALAMIC AND LIMBIC INFLUENCES ON FOOD INTAKE.** *(E.)* Grossman, S. P. (U. Chicago, Ill.). *Fed Proc* 27(6):1349-1360, 1968.

5679 **RELATIONSHIP BETWEEN THE HYPOTHALAMUS AND STOMACH.** *(Rev.) (Fr.)* Martin, M. S. (Ed. Herriot Hosp., Lyon, France), R. Lambert and F Martin. *Path Biol (Paris)* 16(23/24):1099-1113, 1968.

5680 SIZE DISTRIBUTION AND COUNTING OF LIVER NUCLEI BY AN ELECTRONIC PARTICLE COUNTER. *(E.)* Wannemacher, R. W. Jr. (Dept. Nutr., Rutgers U., New Brunswick, N. J.), K. Muramatsu and W. K. Cooper. *Proc Soc Exp Biol Med* 129(3):899-901, 1968.

Liver slices from male Wistar rats of various ages were specially prepared (method of Blobel and Potter) in order to determine the size distribution and number of liver nuclei by means of an electronic particle counter. The number of nuclei counted with the electronic counter closely paralleled the results obtained visually with a microscope and hemocytometer. The electronic counter required less than a minute to count the sample as compared to 5-10 min for the hemocytometer. There was a good correlation between the degree of liver polyploidy and nuclear size. The DNA content and the nuclear size both increased with the age of the animal. A clear separation of the various degrees of polyploidy according to precise nuclear volumes was not possible.

5681 IRON CONTENT OF BRUNNER'S GLANDS IN ACQUIRED HEMOSIDEROSIS. *(E.)* Astaldi, G. (Tortona Hosp., Italy), P. L. Taverna, G. Meardi and R. Airo. *Amer J Dig Dis* 13(11):935-940, 1968.

Histochemical studies of the iron content (hemosiderin) within sections of Brunner's glands in duodenal biopsy specimens from 10 healthy subjects showed that practically no iron is normally found within the glands. In contrast, in 7 patients with aplastic anemia and marked transfusional hemosiderosis, the cuboidal cells of the same glands showed a high hemosiderin content. In the most severe cases, 100% of the tubules showed a positive reaction to Perl's test. The intensity of the iron-positive reaction within the duodenal glands varied according to the severity of the hemosiderosis. These results are in agreement with the interpretation that, in hemosiderosis, the desquamation of iron-loaded epithelial cells of Brunner's glands contributes to the removal of iron from the body. The cells have this function in common with the epithelium of other mucus-secreting glands and with macrophages.

5682 BIOCHEMICAL AND HISTOCHEMICAL OBSERVATIONS ON β-GLUCURONIDASE IN THE MAMMALIAN GALLBLADDER. *(E.)* Ballantyne, B. (Sch. Med., U. Leeds, England) and W. G. Wood. *Amer J Dig Dis* 13(6):551-557, 1968.

Beta-glucuronidase was determined quantitatively and localized histochemically in gallbladders removed from 6 male and 6 female adult Flemish giant rabbits and the same number of male and female guinea pigs after they had been killed by an overdose of 6% (w/v) sodium pentobarbitone. The gallbladders from male guinea pigs contained 25% more β-glucuronidase than the female, and that of the male rabbits 56% more than the female. Male guinea pigs had 3.8 times more β-glucuronidase than male rabbits, and female guinea pigs 4.8 times more than female rabbits. Most of the enzyme was found in the epithelial cells of both rabbit and guinea pig gallbladders with much lower concentrations in smooth muscle and connective tissue cells of the lamina propria. Beta-glucuronidase was found in the cytoplasm of the epithelial cells and was concentrated near the cell membrane. The possible role of this enzyme in Na^+ transport, mitosis in the gallbladder epithelium, and bilirubin absorption is discussed.

5683 APICAL LOCALIZATION OF GLYCOPROTEINS IN GASTRIC MUCOUS CELLS. *(E.)* Lambert, R. (Ed. Herriot Hosp., Lyon, France), F. Martin, C. André, L. Descos and G. Vouillon. *Amer J Dig Dis* 13(11):941-951, 1968.

The staining characteristics and distribution of glycoproteins in the apical pole of gastric fundic mucosal cells was studied in the pig, rabbit, guinea pig, mouse, rat, dog, bat, monkey and man. The ultrastructure of rat gastric mucosa was also studied by electron microscopy. In most of the species studied, a mucous coat was observed at the apical pole of the cells, in the epithelium, and in the deep part of the gastric pits. This coat, which was rich in sulfated glycoproteins, probably plays a role in the resistance of the mucosa to peptic digestion. It is suggested that glycoprotein molecules may inhibit proteolysis by the formation of a resistant complex with the protein of the mucosal cells.

5684 ELECTRON MICROSCOPIC MEASUREMENTS OF THE THICKNESS OF VARIOUS MEMBRANES IN OXYNTIC CELLS FROM FROG STOMACHS. *(E.)* Lillibridge, C. B. (Dept. Med., U. Washington, Seattle). *J Ultrastruct Res* 23(3/4):243-259, 1968.

Gastric mucosa obtained from fasting leopard frogs *(Rana pipiens)* was fixed in glutaraldehyde (1.6%), acrolein (0.5-16%), permanganate (1.8%) or osmium tetroxide (3.3%) and subjected to electron microscopy on an RCA-EMU 2C microscope. The average spacing between the centers of the two dense components in the unit membranes composing the vesicotubules of the parietal cells was 51-65 Å (± 7), and was thus the same as the spacing in the plasma membrane at the glandular lumen (51-69 Å). The spacing in the membranes of mitochondria was 41-50 Å (± 10) and that in ribosome-coated endoplasmic reticulum was 44-47 Å (± 7), both of which were thus consistently smaller than the spacing in the plasma membrane or the vesicotubules. Except in the case of the endoplasmic reticulum, the spacing in most membranes was larger after fixation in aldehydes than after fixation in aldehydes than after fixation in inorganic media. The sources of error in electron microscopic measurements of this type are analyzed in detail, and concluded to total about ± 7.5 Å if all errors were in the same direction. Based on the assumption that subcellular elements having narrower spacing are associated with internal membranes while those with wider spacing are associated with the plasma membrane, it is concluded that the vesicotubules in frog parietal cells probably communicate directly with the cell exterior; they are therefore not part of the endoplasmic reticulum.

5685 INCREASED NUMBERS OF ARGENTAFFIN CELLS IN THE GUINEA PIG DUODENUM FOLLOWING INTRAPERITONEAL INJECTION OF ANGIOTENSIN. *(Ger.)* Huber, R. (Inst. Path. Anat., U. Lausanne, Switzerland), E. Weber and C. Hedinger. *Klin Wschr* 46(21):1168-1169, 1968.

The duodenal crypts of guinea pigs sacrificed 1 hr after i.p. injection of angiotensin (1 μg) showed approximately 422 argentaffin cells/100 crypts compared to 196 in controls given saline. Further studies demonstrated that the number of argentaffin cells rises rapidly to a maximum in about 30-60 min and begins to decrease slowly after 2-4 hr, normal values being reached 24 hr after injection of angiotensin. When guinea pigs were exposed to stress (being transported 200 km) before administration of either angiotensin or saline, the number of argentaffin cells per 100 duodenal crypts was 449-450 in both groups, indicating maximal stimulation in response to stress alone.

5686 QUANTITATIVE HISTOCHEMISTRY OF HISTAMINE AND HISTIDINE DECARBOXYLASE IN THE STOMACH OF RAT, PIG, AND DOG IN VARIOUS FUNCTIONAL STATES. *(E.)* Kim, Y. S. (Stanford U. Med. Sch., Palo Alto, Calif.) and D. Glick. *Gastroenterology* 55(6): 657-664, 1968.

Fluorometric studies on specimens from freshly killed animals showed that histamine and histidine decarboxylase are localized primarily to the chief cell zone in the body of the stomach of fed rats. Enzyme activity was negligible in the rat antrum and in all regions of the pig and dog stomach. Although no enzyme activity could be demonstrated in the rat stomach after a 48-hr fast, the histamine concentration and its histological distribution remained virtually unchanged. Sodium pentothal treatment decreased enzyme activity in fed rats, while bethanechol and ACTH had little effect; however, the histamine concentration was reduced by bethanechol and ACTH, but not by pentothal. Injected histamine reduced enzyme activity, but not the histological distribution or concentration of histamine in the gastric mucosa. The histamine concentration appeared relatively high in the parietal cell, chief cell, and submucosal regions of the fasted dog stomach. Atropine reduced the histamine concentration in the chief cell and submucosal areas. Histamine was also demonstrated in the glandular portion of the dog antrum. Pig stomach showed a histamine distribution similar to that in the rat, but submucosal levels were high. The data suggest a feedback control of histamine synthesis; assuming such a control mechanism, histidine decarboxylase activity should increase on tissue histamine depletion. To reconcile the observation that decarboxylase activity decreased after histamine injections with the observation that ACTH or bethanechol reduced the histamine concentration but had little effect on the enzyme activity, it is pointed out that the histamine concentration may not have been reduced drastically enough or that the time interval between drug administration and death may not have been sufficient to observe the effect.

5687 FINE STRUCTURAL LOCALIZATION OF THREE LYSOSOMAL ENZYMES AND NONSPECIFIC ALKALINE PHOSPHATASE IN THE VILLUS OF THE HUMAN DUODENUM. *(E.)* Hugon, J. S. (Fac. Med., U. Sherbrooke, Quebec, Canada) and M. Borgers. *Gastroenterology* 55(5):608-618, 1968.

The ultrastructural distribution of the lysosomal enzymes acid phosphatase, arylsulfatase, and E-600 resistant esterase and of a nonspecific alkaline phosphatase were studied in the absorbing cells of the duodenal villi of 4 patients (3 men and 1 woman) operated on for peptic ulcers. Cytochemical findings were compared with the morphological aspects of the cells. The lysosomal enzymes were found mainly in the various forms of duodenal lysosomes. Alkaline phosphatase activity was observed on the microvilli, in some smooth endoplasmic reticulum profiles, and on all of the dense bodies. It has not been proven that alkaline phosphatase is functional in the lysosome; however, it can be reactivated after total inhibition by ethylenediaminetetraacetic acid. The enzyme may play a role in fat, protein, amino acid, and sugar absorption.

5688 GANGLION CELL DISTRIBUTION IN THE NORMAL RECTUM AND ANAL CANAL: A BASIS FOR THE DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE BY ANORECTAL BIOPSY. *(E.)* Aldridge, R. T. (Royal Child. Hosp., Melbourne, Australia) and P. E. Campbell. *J Pediat Surg* 3(4):475-490, 1968.

The normal morphology and distribution of ganglion cells in the lower rectum and anal canal were studied at autopsy in 22 infants and children who died of unrelated diseases and had no history of constipation. Ganglion cells were found to be distributed in the myenteric, deep submucous, and superficial submucous plexus. Depending on age, a hypoganglionic zone was generally present which extended upwards an average of 4 mm (range 0-14 mm) in the myenteric plexus and 7-10 mm (range 2-18 mm) in the submucous plexus from the anal valve line. In rare cases, there was as much as an additional 1.5 cm of bowel where such cells were sufficiently sparse to be missed in random sections. Beyond this point, however, ganglion cells were found in their normal closely-packed distribution and could easily have been identified in suction-biopsy specimens of rectal mucosa. The ganglion cells of Auerbach's plexus were found to extend beyond those of the submucous plexus. It is concluded that any biopsy which includes the plexus over 3 cm above the anal valve will suffice for the diagnosis of Hirschsprung's disease, provided the sections are stained with hematoxylin and eosin after paraffin processing. The presence of ganglion cells in specimens obtained from the hypoganglionic zone or a point 3 cm above the anal valve excludes a diagnosis of Hirschsprung's disease. The absence of ganglion cells in the hypoganglionic zone is relatively meaningless, but their absence at a point 3 cm or more above the anal valve line is diagnostic of Hirschsprung's disease.

5689 HISTOCHEMICAL STUDIES ON THE ENZYMES IN HUMAN LIVER DURING FETAL DEVELOPMENT. *(Fr.)* Roman, I. C. (Fac. Med., Bucharest, Romania). *Ann Histochim* 13(3):155-163, 1968.

A histochemical study was made of the enzymes in liver slices from human embryos aged 2, 3, 4, and 5 months. The livers of embryos up to 4 months of age contained small amounts of ATPases, phosphomonoesterases, succinic dehydrogenase, and isocitric dehydrogenase-NAD; larger amounts of pentose shunt enzymes; and very large amounts of enzymes involved in anaerobic glycolysis. During this period no 20 β-hydroxysteroid dehydrogenase, β-hydroxybutyrate dehydrogenase, or monoamine oxidase was detected. Beginning with the 4th month enzyme activity was characterized by a reduction in the activities of enzymes participating in anaerobic glycolysis and the pentose shunt and an increase in those belonging to the Krebs cycle, lipoic dehydrogenase, diaphorases, and ATPases. Monoamine oxidase, 20β-hydroxysteroid dehydrogenase, and β-hydroxybutyrate dehydrogenase were detected for the first time at the beginning of the 4th month.

5690 COLONIC HISTIOCYTOSIS: CLINICAL AND PATHOLOGICAL EVALUATION. *(E.)* Ekuan, J. H. (U. Colorado Med. Ctr., Denver) and R. B. Hill Jr. *Gastroenterology* 55(5):619-625, 1968.

Colonic histiocytosis (the presence of PAS-positive histiocytes, often in small clusters in the lamina propria or subjacent to the crypts) was found in 14 out of 136 consecutive colorectal biopsies. However, no significant changes in the mucosal epithelium or crypts were found in any case, and the inclusions of moderately osmiophilic material were completely different from the inclusions characteristic of Whipple's disease. These 14 patients (8 males and 6 females, 13-72 yr old) had a wide range of presenting complaints and included 2 with no gastrointestinal symptoms. Only 2 had significant intestinal disease. These findings indicate that colonic histiocytosis is only a morphological entity with no clinical significance and is not related to Whipple's disease.

5691 SUBMICROSCOPIC FEATURES OF THE DUODENUM AND JEJUNUM AFTER INTRALUMINAL ELECTRICAL STIMULATION. *(It.)* Labo, G. (Inst. Spec. Med. Path., U. Bologna, Italy), T. Posteli, G. Gasbarrini, F. Faggioli and N. Melchionda. *Arch Ital Mal Appar Dig* 34(5):433-472, 1967.

After bipolar, intraluminal, electrical stimulation with a direct current for different periods, biopsies of the intestinal mucosa were taken with a Crosby-Kugler capsule from 5 normal female subjects, aged 23-65 yr, and longitudinal sections of the second part of the duodenum or the first jejunal loop were obtained from female Sprague-Dawley rats. Electron microscopy showed that, in contrast to the parallel position seen under basal conditions, protofilaments in the cytoplasm of the covering epithelium were oriented perpendicular to the major cell axis. Fine granular structures were found inside the microvilli, and filaments seemed to project outside the double membrane of the covering in a direction transverse to the core of the microvilli, no matter whether stimulation was applied for 30 sec or 5 min. The mitochondria became much rounder and had a mean transverse diameter 4 times that found under basal conditions. The most pronounced mitochondrial changes were seen after prolonged stimulation (5 min). Ergastoplasmatic canals in the middle of the cells were markedly dilated and filled with small masses of osmophilic material with regular contours and without a covering membrane. Material of the same type was present in the Golgi apparatus and in small intercellular spaces. Microfilaments of smooth muscle cells were oriented parallel to the major cell axis and to the basal epithelial membrane. The direction of cytoplasmic filaments in the intramural nerve plexus close to a small cluster of nonmyelinated fibers was changed to that they were oriented toward the nerve tissue. Possible interpretations of these findings are discussed.

5692 AGE DEPENDENT IMPAIRMENT OF CELLULAR PROLIFERATION IN REGENERATING RAT LIVER. *(Ger.)* Klinge, O. (Inst. Path., U. Würzburg, Germany). *Virchow Arch Abt B Zellpath* 1(4):342-345, 1968.

A histological study was made of regenerating livers in 36 male Sprague-Dawley rats following 2/3 hepatectomy at the age of 4 weeks (young rats), 6 months (adults), and 2 yr (old animals). Animals were killed and livers removed 24, 28, 32, and 36 hr after the operation. The maximum rate of mitosis was lower in old animals than in young ones and occurred at 36 hr compared to 28 hr for the young animals. Disturbances in mitosis occurred in 27% of cells examined in young rats, 38% in adults, and 65.6% in old animals. In young and adult rats these disturbances generally occurred in the later stages of mitosis and were usually due to disturbances in spindle formation and the failure of cytokinesis to occur, resulting in the formation of cells with 2 nuclei. In old animals most mitotic disturbances occurred during prophase and were usually due to the failure of spindles to form or spindle anomalies and incomplete migration of the centriole.

5693 STUDIES ON THE RETICULIN NETWORK OF HUMAN LIVER. *(E.)* Velican, C. (Inst. Intern. Med., Acad. Sci., Bucharest, Romania) and D. Velican. *Virchow Arch Abt B Zellpath* 1(4):297-316, 1968.

The topochemical significance of argyrophilia, its variations in intensity, and the mechanism of "collagenization" of the hepatic reticulin network were investigated in biopsy specimens from 293 patients (179 males and 114 females, most 40-70 yr old) with chronic diseases of the liver (mostly hepatitis and cirrhosis). The occurrence of argyrophilia in the human liver reticulin network was found to require the presence of macromolecular complexes rich in hydroxyl groups, which are capable of being converted into carbonyl groups, and probably some lipids possessing aldehydic functions. Enzymatic removal of sialic acid and hexosamines increased the intensity of the argentic impregnation, suggesting that the material involved in the reaction is composed primarily of hexoses. Lipozyme digestion of glucosamine apparently makes some hydroxyl and/or carbonyl radicals available, allowing more intense and uniform silver staining; glucosamine is a component of the perifibrillary sheath of some reticulum fibers and is included in a complex of cement substances connecting network components to the subendothelial space. Collagenization of the reticulum fibers results from a progressive increase in the number of collagenous microfibrils and fibrils. Fuchsinophilia occurs when the reactivity of hexose hydroxyl radicals is associated with an adequate concentration of hydroxyl groups of collagen which binds van Gieson's picrofuchsin. This collagen shows a macromolecular hypostability which is reflected in an increased susceptibility to collagenase digestion, an increased solubility in alkali solution and acidic buffers, and the breakage of hydrogen and electrostatic bonds.

5694 AGE CHANGES IN SIZE AND NUMBER OF MITOCHONDRIA OF HUMAN HEPATIC CELLS. *(E.)* Tauchi, H. (Nagoya U. Sch. Med., Japan) and T. Sato. *J Geront* 25(4):454-461, 1968.

Electron microscopy of liver biopsy specimens taken from 52 patients (21-79 yr old) without liver pathology revealed a decrease in number and increase in size of the mitochondria with age, especially in those patients over 60 yr old. While the average number per 5000 μ^2 decreased from 1893.8 in those under 49 yr to 1200.4 in those over 70, the mean square root of the planimetric area increased from 0.79 to 1.15 and the mean circumference increased from 3.29 to 4.73 μ in the same age groups. Histochemical studies of tissue specimens revealed no age-related alterations in succinic dehydrogenase activity. Osmiophilic granules and other inclusion bodies, however, increased in both size and number with age, particularly in the giant mitochondria.

5695 LOCALIZATION OF GASTRIN ACTIVITY IN THE GASTRIC ANTRUM.

(E.) Broomé, A. (Cent. Hosp., Boras, Sweden), B. Fyrö and L. Olbe. *Acta Physiol Scand* 74(3):331-339, 1968.

Using conscious gastric-fistula cats and sham-fed antrectomized Pavlov-pouch dogs for the assay of extracts prepared by the method of Komarov *(Proc. Soc Exp. Biol.* 38:514-516, 1938), gastrin activity was determined in sections (cut parallel to the surface) of the antral mucosa and submucosa of cats and dogs, and in the aboral 2 cm of corpus mucosa of dogs and man. The maximum gastrin activity in the antrum was found in the basal two-thirds of the gland crypt region of the mucosa. The superficial third of the antral mucosa of the cat contained no gastrin activity while in the dog traces were found there. Extracts from the submucosa of the cat antrum contained no gastrin activity but low activity was found in one dog out of three. Gastrin cell may thus be localized predominantly in the middle and basal parts of the gland crypts of the antral mucosa in the dog and cat. Using the aboral 2 cm of mucosa, above the corpus-antrum boundary, no gastrin activity was found in 3 dogs or in 10 of 14 duodenal ulcer patients. Gastrin cells thus seem to be absent from the corpus mucosa of dog and man, but the occasional finding of gastrin in the most aboral part of the acid-secreting gastric mucosa of man suggests a dentate, or sometimes deeply notched, antrum-corpus boundary.

5696 GASTRIN-LIKE ACTIVITY IN DIFFERENT PARTS OF THE GASTRO-INTESTINAL TRACT OF THE CAT.

(E.) Emås, S. (Dept. Pharmacol., Karolinska Inst., Stockholm, Sweden) and B. Fyrö. *Acta Physiol Scand* 74(3):359-367, 1968.

Specimens of mucosa from various portions of the cat digestive tract were extracted by the method of Komarov *(Proc. Soc. Exp. Biol.* 38:514-516, 1938) and, in the case of the antrum and proximal duodenum, by a modification of the method of Gregory and Tracy *(Gut* 5:103-117, 1964). The extracts were assayed for gastrin activity on nonanesthetized gastric-fistula cats. Gastrin-like activity was found in the mucosa of the antrum and the proximal duodenum using either method of extraction. Gastrin-like activity was also found in most extracts from the cardiac region of the stomach. However, extracts from the cardiac region and from the proximal duodenum had only 5-10% of the activity of those from the antrum. The acid-secreting portion of the stomach, the middle and distal parts of the duodenum, and the jejunum, colon and pancreas had no gastrin-like activity. Since cholecystokinin-pancreozymin produced a significant acid response only at doses of 16-32 μ/kg/hr i.v., it is unlikely that the duodenal activity can be ascribed to this hormone.

5697 GASTROINTESTINAL DISTRIBUTION OF GLUCURONIDE SYNTHESIS AND THE RELEVANT ENZYMES IN THE RAT.

(E.) Hänninen O. (Dept. Physiol., U. Turku, Finland), A. Aitio and K. Hartiala. *Scand J Gastroent* 3(5):461-464, 1968.

In studies on tissue slices from adult male Wistar rats, the rate of o-aminophenol glucuronide synthesis in the small intestine was found to decrease from the oral to the aboral end, where it was 15% of the duodenal level. The rates in slices of the glandular stomach, cecum and colon were 34, 15 and 30%, respectively, of the duodenal value. The decrease in uridine diphosphate-glucuronyltransferase activity (to p-nitrophenol) in the mucosal extracts of the small intestine was almost linear and amounted to 66% from the oral to the aboral end. The activities in the glandular stomach, cecum and colon were 63, 32 and 83%, respectively, of the duodenal value. In contrast, the β-glucuronidase activity increased linearly by 41% between the oral and the aboral end of the small intestine. The activities in the glandular stomach, cecum and colon were 82,221 and 162%, resp., of the duodenal value. The distribution pattern of glucuronide synthesis in tissue slices of the gastrointestinal tract and the relevant enzyme activities in the extracts thus differ. It is concluded that there must be additional limiting factors in intact cells.

5698 LYMPH DRAINAGE FROM THE ESOPHAGUS IN RELATION TO THE SPREAD OF CANCER: AN EXPERIMENTAL STUDY.

(It.) Brunetti, E. (Inst. Gen. Clin. Surg., U. Rome, Italy), L. Forlivesi and F. Corneli. *Arch Chir Torac Cardiovasc* 22(2):169-181, 1968.

To clarify the different routes by which esophageal cancer can metastasize through the lymphatic system, 0.5 ml of a 10% India India ink solution was injected into the submucosal or muscular layer of the esophageal wall of 24 healthy mongrel dogs at the level of the cervical, thoracic, and abdominal segments. Dogs were killed at various intervals 24-48 hr after injection and lymph drainage was studied. Lymph flow in the submucosal lymph plexus differed depending upon the segment of the esophagus into which the ink was injected, while that in the muscular lymph plexus occurred in the same way regardless of the site of injection. The rate of lymph flow from the muscular lymph plexus was considerably faster than that from the submucosal. For the most part, the flow of lymph from collectors to regional lymph nodes occurred perpendicular to the esophageal wall and reached lymph glands located far away. Lymph drainage from the thoracic segment of the esophagus and the thoracic duct almost always involved the mediastinal lymph nodes. On the basis of these results, it is recommended that esophagectomies for cancer be combined with removal of tributary lymph nodes in order to prevent metastasis.

5699 PROLONGED FIXATION OF LIVER TISSUE IN GLUTARALDEHYDE AND ITS USE IN ELECTRON MICROSCOPY.

(Por.) Sampaio de Lacerda, P. R. (Inst. Path., U. Dusseldorf, Germany). *Hospital (Rio)* 74(2):379-402, 1968.

Pieces of liver tissue measuring about 1 x 0.5 x 0.3 cm were obtained from black male and female mice and fixed in 6.25% glutaraldehyde in 0.1 M pH 7.3 Sorenson buffer containing

0.2 M sucrose. Material was then stored for 10 or 20 days at 4C or room temperature (20-24C). No morphological changes were seen under the light microscope in any of the specimens. Electron microscope studies were made after tissue had been washed in buffer for 30 min, fixed for 2 hr in OsO_4 buffered with pH 7.4 Palade solution, sectioned in an ultramicrotome, and contrasted with 0.5% uranium acetate and lead acetate. No changes were seen in samples stored for 10 days at 4C. Changes in the cell membranes, particularly in the sinusoids, were found in samples stored longer under refrigeration or at room temperature. These changes in ultrastructure are attributed to changes in osmolarity. No evidence of necrosis or autolysis was found.

5700 **HEMATOPOIETIC TISSUE IN THE LIVER OF THE FETAL RAT AT THE END OF GESTATION. II. EFFECT OF MATERNAL LAPAROTOMY AND ADRENALECTOMY.** *(Fr.)* Nagel, J. (Fac. Sci., Reims, France) and R. Jacquot. *Arch Anat Micr Morph Exp* 57(2):99-105, 1968.

In experiments on Wistar CF rats, maternal laparotomy on the 15th day of gestation produced a significant reduction in the amount of hematopoietic tissue in the livers of 17-21 day-old fetuses. Sham adrenalectomies performed on the 15th day had the same effect on 17-day-old fetuses. Bilateral adrenalectomies performed on mothers on the 14th-15th day of gestation had no significant effect on hematopoietic tissue in the 17-day-old fetus but produced a significant reduction in 18- and 19-day-old fetuses; this reduction was not as great as that seen after laparotomy alone. No differences were found between fetuses whose mothers had undergone adrenalectomy on the 14th day and laparotomy on the 15th and those whose mothers had only been adrenalectomized. These results suggest that a large increase occurs in fetal adrenal activity and circulating maternal steroids 18 days after copulation; hematopoietic tissue then disappears rapidly from the fetal liver. Maternal adrenalectomy produces adrenal hyperactivity in the fetus to compensate for loss of maternal steroids; this results in a significantly more rapid reduction in hematopoietic tissue than in control fetuses. The reduction in fetal hematopoiesis occurring after maternal laparotomy or sham adrenalectomy is probably due to an increase in maternal steroid secretion.

5701 **HYDROLYTIC ENZYMES IN SPLENIC AND HEPATIC MEGAKARYOCYTES OF THE RAT.** *(E.)* McFadden, K. D. (Sch. Med., U. Alberta, Edmonton, Canada) and G. J. Marien. *Growth* 32(3):189-198, 1968.

Histochemical tests on liver and spleen tissue of rats (Sprague-Dawley, 100 male and female) of various ages (fetal to 1 yr) confirmed the presence of hydrolytic enzymes within the cytoplasm of the megakaryocyte cell line. These cells appear in the liver from the fetal period until the 10th postnatal day, and in the spleen, largely after the 10th postnatal day. All megakaryocytes observed in both liver and spleen contained the 4 enzymes studied (acid phosphatase, non-specific esterase, β-glucuronidase and leucine aminopeptidase) regardless of the age of the animal. Activity was high for all except leucine aminopeptidase in both organs. In contrast to the constant level of activity in the mature megakaryocytes, other cells (splenic marginal metalophils, free macrophages) showed increasing enzyme activity with increasing age. The granules signifying the presence of enzymes were fewer in immature megakaryocytes than in mature cells, which may be related to differences in cell volume or to changes associated with maturation.

5702 **A TABULAR SYNOPSIS OF THE ANTIGENIC SUBSTANCES IN THE HUMAN GASTRIC MUCOSA.** *(Ger.)* Rapp, W. (Med. Clin., U. Heidelberg, Germany). *Wiederbeleb Organersatz* 5(2):102-107, 1968.

5703 **ASPECTS OF THE FINE STRUCTURE OF LIGHT AND DARK CELLS IN THE INTRAHEPATIC BILE DUCT EPITHELIUM OF THE MOUSE.** *(E.)* Yamada, K. (Nagoya U. Sch. Med., Japan). *Nagoya J Med Sci* 30(3):309-318, 1967.

5704 **RADIOAUTOGRAPHIC AND ELECTRON MICROSCOPIC STUDIES OF THE DARK CELLS OF THE LIVER.** *(Rus.)* Krymskii, L. D. (A. V. Vishnevskii Inst. Surg., Acad. Med. Sci. USSR, Moscow), B. V. Vtiurin, K. V. Botsmanov, A. K. Badikova and K. I. Dzarakhokhov. *Biull Eksp Biol Med* 66(9):105-108, 1968.

5705 **THE LYMPHOID TISSUE OF THE HUMAN GASTROINTESTINAL MUCOSA. II. ITS ROLE.** *(Fr.)* Crabbé, P. A. *Presse Med* 76(39):1875-1878, 1968.

5706 **INITIAL RESULTS OF HISTOCHEMICAL FLUORESCENCE STUDIES ON THE CATECHOLAMINE AND 5-HYDROXYTRYPTAMINE CONTENT IN THE HUMAN INTESTINAL WALL.** *(It.)* Capurso, L. (Inst. Spec. Med. Path., U. Rome. Italy) and C. A. Friedman. *Arch Ital Mal Appar Dig* 34(6):577-580, 1967.

5707 **ULTRASTRUCTURAL ASPECTS OF THE ENDOCRINE SECRETION OF THE DUODENUM.** *(It.)* Labó, G. (Inst. Spec. Med. Path., U. Bologna, Italy), G. Gasbarrini, V. Freyrie, N. Melchionda and F. Benfenati. *Arch Ital Mal Appar Dig* 34(4):375-384, 1967.

5708 **METACHROMATIC REACTION OF PANCREATIC B CELLS TO TOLUIDINE BLUE; INFLUENCE OF pH ON STAINING.** *(E.)* Fujita, T. (Okayama U. Med. Sch., Japan) and K. Takaya. *Stain Techn* 43(6):329-333, 1968.

See also: 6148, 6160, 6218, 6466, 6534, 6551, 6630

5709 **VARIATION IN INTESTINAL TRANSPORT OF L-VALINE IN RELATION TO AGE.** *(E.)* Ning, M. (Indiana U. Sch. Med., Indianapolis), S. Reiser and P. A. Christiansen. *Proc Soc Exp Biol Med* 129(3):799-803, 1968.

Weanling male Wistar rats were fed *ad libitum* and everted intestinal sacs were used to measure various parameters of valine transport at 4, 8, 12, 18, 24 and 40 weeks of age. The rats gained weight at the rate of 25 g/week until 18 weeks of age, after which a weight gain of 9 and 4.5 g/week was observed at 24 and 40 weeks, resp. The serosal/mucosal valine ratio remained relatively constant during the first 8 weeks, after which it fell off sharply and was only 1/6 of its initial level at 40 weeks of age. Net transport of valine fell off linearly between 4 and 18 weeks of age, after which it remained at a relatively constant level. At the end of 40 weeks, net transport was 1/7 of its initial value. The tissue uptake of valine was reduced by 33% during the experimental period. The decrease in the transport parameters generally followed the increase in animal weight. The results show the necessity of standardizing the weight or age of the experimental animals used for intestinal transport studies.

5710 **COMPARISON OF HISTAMINE AND CARBACHOL EFFECT ON VITAMIN B_{12} ABSORPTION IN MAN.** *(E.)* Kanaghinis, T. (Evangelismos Hosp., Athens, Greece), A. Papayannis, D. Ikkos and P. Gatsou. *Amer J Dig Dis* 13(11):952-958, 1968.

The Schilling test (urinary recovery of orally administered ^{58}Co-cobalamin) was used to compare the effects of histamine (1 mg s.c.) and carbachol (0.25 mg i.m.) on vitamin B_{12} absorption in 61 patients (57 males and 4 females, 14-75 yr old, including several with pernicious anemia or gastric cancer). The results showed that histamine and carbachol are equally potent in increasing the intestinal absorption of radiocobalamin. While it is recognized that histamine increases vitamin B_{12} absorption via stimulation of intrinsic factor secretion in the gastric mucosa, this mechanism is unlikely for carbachol. It is therefore suggested that both drugs may also have a direct effect on the small intestine which acts to increase B_{12} absorption by another, still unknown, mechanism.

5711 **THE PHENOMENON OF PERSORPTION.** *(E.)* Volkheimer, G. (Charité Hosp., Humboldt U., Berlin, Germany) and F. H. Schulz. *Digestion* 1(4):213-218, 1968.

Human volunteers were used to make a quantitative analysis of the phenomenon of intestinal persorption, i.e., the passage of large, solid, undissolved food particles from the intestinal lumen into the venous blood. Subjects were given 100-300 g of uncooked starch suspended in milk or water p.o. and the number of starch granules in the blood was determined at intervals during the next 8 hours. After administration of either corn starch (240 million granules/g with a particle size of 7-22 microns) or potato starch (12 million granules/g with a particle size of 8-110 microns), the number of particles in the venous blood reached a peak in approximately 10 minutes and a 2nd peak after 90 minutes. After 8 hours most of the granules had disappeared. All subjects had been fed a starch-free diet for 3 days and no granules were found in the blood before the test. Up to a point, the number of granules persorbed varied directly with the amount of starch ingested, but there was little difference between the numbers persorbed after equal doses of the 2 types of starch, and the number was no longer increased by doses above 300-400 g.

5712 **MORPHOLOGICAL ASPECTS WHICH SUGGEST THE MECHANISM OF TRANSFER (ABSORPTION) OF CERTAIN PROTEINS IN THE INTESTINE OF THE ADULT RAT.** *(Fr.)* Chisiu, N. S. (Dr. I. Cantacuzino Hosp., Bucharest, Romania). *Rev Roum Med Intern* 5(1):65-69, 1968.

Histochemical and immunofluorescence techniques were used to study the intestinal absorption of protein macromolecules in normal, adult male, Wistar rats. Human serum albumin labeled with fluorescein isothiocyanate could be demonstrated in the conjunctivovascular axes of the jejunal villi following transparietal injection into the duodenum. Penetration of the nonlabeled albumin molecule into the jejunal mucosa following intraduodenal injection was also confirmed by the fluorescence observed after treatment of the jejunum with rabbit antisera prepared against human serum albumin and labeled with fluorescein isothiocyanate. These findings demonstrate that undigested protein molecules can penetrate the intact intestinal epithelium. It is suggested that absorption of such antigenic material in states of protease deficiency may play a role in the development of autoimmune diseases.

5713 **EFFECT OF BACTERIAL FLORA ON IRON ABSORPTION IN THE RAT.** *(E.)* Geever, E. F. (Albert Einstein Coll. Med., Bronx, N. Y.), D. Kan and S. M. Levenson. *Gastroenterology* 55(6):690-694, 1968.

At 21 days of age, half of a group of 20 male Fischer littermate germ-free rats were "conventionalized" by deliberate contamination with normal cecal contents. When the animals had reached a weight of about 275 g, iron absorption was compared in the 2 groups. Measurement of whole-body reactivity for 7 days after gastric instillation of a measured dose of ^{59}Fe sulfate (0.22-0.23 μC) revealed consistently greater values in the germ-free animals. The reason for the increased iron absorption in the germ-free rats remains unclear. However, the importance of the intestinal flora and coprophagy in experimental gastrointestinal absorption studies are discussed.

5714 **BIOCHEMICAL STUDY OF THE MECHANISM OF THE INTESTINAL ABSORPTION: EFFECT OF BLOOD GLUCOSE LEVELS ON GLUCOSE ABSORPTION BY THE INTESTINE *IN VITRO*.** *(E.)* Casey, M. G. (U. Med. Clin., Cantonal Hosp., Lausanne, Switzerland), J. P. Felber and A. Vannotti. *Digestion* 1(4):233-237, 1968.

In rats made diabetic by i.p. injection of alloxan (80 mg/kg), a direct proportionality was discovered between the blood sugar (50-400 mg%) and the ^{14}C-glucose absorbed by loops of excised intestine incubated in Krebs-Ringer bicarbonate buffer (0.25-1.35 micromoles/100 mg in 20 min). Glucose absorption by intestinal loops *in vitro* could also be increased by prior i.v. administration of glucose (2 g/kg) to normal rats, reaching a maximum 60 min after administration. This effect, however, was almost completely counteracted by simultaneous administration of puromycin, indicating a probable role of protein synthesis since there was no effect on intestinal O_2 consumption. It is concluded that the increased glucose absorption in diabetes is a mechanism of compensating for the high blood sugar levels and involves an increase in the number of cellular transport systems.

5715 INHIBITORY EFFECTS OF ALCOHOL ON INTESTINAL AMINO ACID TRANSPORT *IN VIVO* AND *IN VITRO*.

(E.) Israel, Y. (Dept. Biochem., Santiago, Chile), I. Salazar and E. Rosenmann. *J Nutr* 96(4): 499-504, 1968.

The effect of ethanol on the active transport of ^3H-labeled amino acids in the small intestine was studied *in vitro* and *in vivo* in male albino rats on a balanced diet. It was shown that 0.5 and 2.0% ethanol inhibited the active transport of L-phenylalanine in everted jejunal sacs by 60 and 84%, resp. The latter concentration completely abolished the active transport of L-methionine, although it reduced tissue respiration by only 19%. *In vivo,* 250 mg alcohol/100 g body weight given by stomach tube inhibited the intestinal absorption of L-phenylalanine by about 50% and did not affect the absorption of D-phenylalanine, which is not actively transported. These findings may be significant in relation to the nutritional deficiencies and fatty liver that occur in chronic alcoholics, even when on balanced diets.

5716 A MODE OF INTESTINAL ABSORPTION UNUSUALLY DEPENDENT ON PHYSIOLOGICAL STATE.

(E.) Antonioli, J. A. (Dept. Biol. Chem., U. Michigan, Ann Arbor) and H. N. Christensen. *Amer J Physiol* 215(4): 951-958, 1968.

Male Sprague-Dawley rats, weighing 250-350 g and fasted 24 hr before experiments, received intragastric doses of α,α-diethyl- and α,α-dicyclopropylglycine to determine the contribution of a nonspecific pathway of absorption which is presumably masked by the mediated transport of oridinary amino acids. Because of the presence of two hydrocarbon groups on the alpha carbon atom, these 2 amino acids appear not to be taken up into various cells and tissue systems that mediate the transport of ordinary amino acids. Except when energy production is interfered with or desquamation occurs, these amino acids enter and traverse rat intestinal segments slowly and are very poorly absorbed when introduced directly into the intestine by laparotomy under continuous anesthesia. Nevertheless, when ^{14}C-labeled diethyl- or dicyclopropylglycin were given by stomach tube to intact conscious rats, 40-50% of the dose appeared in the urine in 4 hr. Since these amino acids are not metabolized and are cleared at the rate of glomerular filtration, their urinary excretion is assumed to be a measure of their absorption. The intestinal absorption of diethylglycine in the intact animal was not interfered with by simultaneous administration of methionine and was directly proportional to the dose. These findings indicate that diethyl- and dicyclopropylglycine are absorbed by a diffusionlike process which is not chemically mediated but is hampered by intestinal ligation. The precise nature of this process remains to be elucidated.

5717 ABSORPTION OF BILE ACIDS FROM THE LARGE BOWEL IN MAN.

(E.) Samuel, P. (Long Island Jewish Hosp.-Queens Hosp. Ctr. Affiliation, Jamaica, N. Y.), G. M. Saypol, E. Meilman, E. H. Mosbach and M. Chafizadeh. *J Clin Invest* 47(9):2070-2078, 1968.

Bile acid absorption from the large intestine was studied in 8 patients (4 men and 4 women, 31-79 yr old) following cholecystectomy for cholecystitis and cholelithiasis. Only 2 of these patients were clinically jaundized, with SGOT elevation, although a 3rd had an elevated serum alkaline phosphatase. During surgery, 9.6-22 μC of ^{14}C-cholic acid were injected into the lumen of either the cecum, the hepatic flexure of the colon, or the transverse colon in 7 patients. A T-tube was placed in the common hepatic duct of 6 of these patients and bile was collected daily for 5 days, while in the 7th patient bile was aspirated from the terminal ileum. Significant amounts of radioactivity, 43.6-84.6% of the administered dose, appeared in the T-tube bile, mostly within 24 hr. When ^{14}C-cholic acid was given in enema form to an 8th patient 5 days postoperatively, 30.8% was excreted through the T-tube in 48 hr. Thin-layer chromatographic analysis of bile samples indicated that the fraction of the radioactivity recovered as desoxycholate increased during the postoperative period. Daily total bile acid excretion increased significantly from the first to the fifth experimental days. The amount of cholate excreted increased markedly with time, that of chenodeoxycholate increased moderately, and that of deoxycholate decreased sharply during the 5-day period. Marker studies in 3 patients revealed no regurgitation of cholic acid into the small bowel, and aspirates from the terminal ileum in an additional patient contained no radioactivity. Neither the addition of unlabeled cholic acid nor previous treatment with sulfisoxazole or chloramphenicol had any effect on bile acid absorption. The findings indicate that cholic acid is converted to deoxycholic acid in the colon and that both are absorbed in significant amounts from the human large intestine.

5718 ABSORPTION OF FREE AND CONJUGATED BILE ACIDS IN THE SMALL INTESTINE OF RATS *IN VITRO* AND *IN VIVO*.

(Ger.) Forth, W. (Inst. Pharm. Toxicol., U. Saar, Hamburg, Germany) and H. Glasner. *Naunyn Schmiedeberg Arch Pharm Exp Path* 261(4):314-328, 1968.

Isolated, 8-cm-long segments of rat small intestine, from the duodenojejunal flexure to the ileocecal valve, were perfused with Tyrode solution containing 10^{-4}M/liter of 3α,7α,12α-trihydroxycholanic acid; 3α,12α-dihydroxycholanic acid; or their taurine or glycine conjugates. With the exception of dihydroxycholanic acid, these concentrations of bile acids produced little if any inhibition of water and electrolyte absorption. In all cases bile acid absorption was highest in the lower ileum with much more conjugate being absorbed than free bile acid. No evidence was found that free bile acids were converted into conjugates or conjugates into free bile acids during absorption except for conversion of the glycine conjugate of trihydroxycholanic acid into free trihydroxycholanic acid; this is attributed to bacterial hydrolysis. Determinations of bile acid in the wall of intestines perfused with trihydroxycholanic acid, its glycine conjugate, and dihydroxycholanic acid showed that dihydroxycholanic acid concentrations were highest in all parts of the intestine from the upper jejunum to the lower ileum; trihydroxycholanic acid concentrations were higher than those of its conjugates in all sections. In contrast to these *in vitro* studies, *in vivo* studies with ileal loops of rats showed that both bile acids and their conjugates were absorbed very rapidly, and the differences in their rates of absorption were small. The *in vitro* differences are ascribed to the method.

5719 INTESTINAL GLUCOSE ABSORPTION DURING THE LIFE-SPAN OF A COLONY OF RATS. *(E.)* Klimas, J. E. (Biol. Dept., Fairfield U., Conn.). *J Geront* 23(4):529-532, 1968.

The effect of aging on glucose absorption from the small intestine was studied during the life-span of an isolated colony of 100 healthy male Charles River CD strain rats. Body weight and intestinal length and weight increased most rapidly during the first 5 months of growth; however, this increase was accompanied by a decrease in the intestinal length/body weight and intestinal weight/body weight ratios. Glucose absorption, when expressed as mg/hr or mg/cm of intestine per hr, increased significantly during the first 10 months and then remained relatively stable over the remainder of the animal's life. When glucose absorption was expressed as mg/100 g body weight per hr, it decreased significantly during the first 10 months and remained fairly stable as the animal grew older. Glucose absorption thus seems to depend primarily on the relative length of the small intestine.

5720 CALCULATION OF CALCIUM ABSORPTION RATE FROM PLASMA RADIOACTIVITY. *(E.)* Nordin, B. E. C. (Gen. Infirm., Leeds, England), M. M. Young, C. Oxby and L. Bulusu. *Clin Sci* 35(2):177-182, 1968.

In a study of calcium absorption rates, serum ^{47}Ca or ^{45}Ca activity was measured 30, 60 and 120 min after the oral administration of 5 μC of radioisotope to 6 normal elderly subjects, 5 normal young adults, 4 patients on corticosteroid therapy, 6 patients with renal disease, 7 with osteoporosis, 3 with osteomalacia, 4 with Paget's disease, 26 with renal stones with or without hypercalciuria, and 6 with primary hyperparathyroidism. Calcium malabsorption was detected after 30 min in the elderly, in patients on steroid therapy, and in some of those with renal disease or osteomalacia. High absorption rates were observed in renal disease patients with hypercalciuria and in those with primary hyperparathyroidism. Diagnostic discrimination was greatest at 30 min and decreased with time. Mathematical analysis showed that the discrimination can be improved by making an approximate correction to allow for the removal rate of the isotope from the plasma.

5721 EFFECT OF AGE ON THE INTESTINAL ABSORPTION ON RADIOMETHIONINE. *(Hun.)* Pénzes, L. (U. Med. Sci., Budapest, Hungary), G. Simon and M. Winter. *Kiserl Orvostud* 20(5):494-499, 1968.

Tracer studies with dl-^{35}S-methionine showed that the percentage of an administered dose (0.56-36.0 mg/100 g body weight) absorbed in 20 min by a ligated segment of small intestine was inversely proportional to the dosage and was significantly higher in 26-month-old Wistar female rats than in 6-month-old rats. Thus, when 0.85 mg/100 g was administered, the percent absorption was 39.1 in 6-month-old rats, 42.7 in 14-month-old rats and 48.0 in 26-month-old rats, even though the body weights were all approximately the same (229-255 ± 32 g). A similar effect of age was observed when the specific activity of the liver proteins was determined, although age had no significant effect on either the percentage of administered radioactivity incorporated into the liver or the radioactivity of the blood. At an administered dose of 3.25 mg/100 g body weight, the activity of the liver proteins (counts/min/10 mg) increased from 94 in 6-month-old rats to 103 in 14-month-old rats and 140 in 26-month-old rats. The increased utilization of administered methionine in old age is ascribed to the increased requirements for this essential amino acid.

5722 PHYSIOLOGIC ALTERATIONS IN SMALL BOWEL FUNCTION WITH AGE: THE ABSORPTION OF D-XYLOSE. *(E.)* Guth, P. H. (Orange County Med. Ctr., Orange, Calif.). *Amer J Dig Dis* 13(6):565-571, 1968.

The 5 g xylose tolerance test was given to 98 patients, aged 10 to 96 yr, and the 25 g xylose tolerance test to 29 patients, aged 36 to 95 yr. No patients had any gastrointestinal or genitourinary disease. Xylose was determined colorimetrically in urine excreted for 5 hr after ingestion; hourly blood levels were also determined in the 25 g tolerance test. Results from the 5 g tolerance test showed a sharp drop in xylose excretion in patients over 80 yr of age with a statistically significant difference between the mean values for all age groups from 29 to 79 yr and the mean value for the age group 80 yr and over. The same results were obtained for urinary xylose excretion in the 25 g tolerance test. Hourly blood xylose concentrations were much lower in the age group 80 yr and over, reached a maximum only after 3 hr (in contrast with 1 and 2 hr in other groups), and decreased much more slowly than in younger age groups. A definite decrease in relative renal clearance occurred with increasing age. These results suggest that in subjects over 80 the small intestine is less able to absorb xylose and the kidneys are less able to excrete it. Xylose absorption tests should be interpreted with caution in elderly subjects.

5723 DEVELOPMENT OF SUGAR AND AMINO ACID TRANSPORT BY INTESTINE AND YOLK SAC OF THE GUINEA PIG. *(E.)* Butt, J. H. II (Harvard Med. Sch., Boston, Mass.) and T. H. Wilson. *Amer J Physiol* 215(6): 1468-1477, 1968.

The development of active transport systems for L-proline, glycine, sarcosine, N,N-dimethylglycine, betaine, L-valine, L-lysine, and α-methylglucoside was studied by the tissue-accumulation method in the intestine and yolk sac of the guinea pig. At about 40 days of gestation, segments of the small intestine were capable of accumulating all of the amino acids, with the exception of betaine and N,N-dimethylglycine against a concentration gradient. The tissue concentration of the latter two amino acids remained considerably less than that in the incubation medium. The capacity to transport these N-substituted amino acids actively did not appear (by the criterion used) until about the 60th day of gestation, at which time it rapidly rose to adult levels. The developmental pattern of N,N-dimethylglycine transport in the yolk sac was also different from that of the other amino acids, and it was inferred that betaine and N,N-dimethylglycine are transported by a different membrane-carrier system. There was a marked increase in the capacity of the intestine to accumulate all of the substances studied at about the time of birth. This activity declined again to adult levels during the following few weeks. A somewhat similar pattern of development was seen in the β-galactosidase activity of the small intestine.

5724 **SECRETION OF ELECTROLYTES AND WATER BY THE GUINEA PIG SMALL INTESTINE** *IN VIVO.* *(E.)* Powell, D. W. (Walter Reed Army Med. Ctr., Washington, D. C.), S. J. Malawer and G. R. Plotkin. *Amer J Physiol* 215(5):1226-1233, 1968.

Net water, electrolyte and glucose transport by the jejunum, ileum, cecum and colon were determined in anesthetized, non-fasted guinea pigs (male, Walter Reed strain) by an *in vivo* perfusion technique. There was net lumen-to-blood transport (absorption) of water and total solute in the cecum and colon, but net blood-to-lumen transport (secretion) in the jejunum and ileum, as shown by direct measurement of volume and by changes in concentration of recovered polyethylene glycol (water marker technique). The small intestine showed significant absorption of glucose and Cl$^-$, but secretion of Na$^+$, K$^+$ and HCO$_3^-$. This secretion, against an osmotic gradient of 35 milliosmoles, suggests an active process. A reciprocal dependency between HCO$_3^-$ secretion and Cl$^-$ absorption was noted in the small intestine, which suggests a relation between the transport of these ions. Electrical potential differences, measured in the jejunum and ileum during secretion, and in the cecum and colon during absorption, were negative across all segments despite the movement of solute and water in opposite directions. Intestinal transport in this species is significantly different from transport in the rat intestine, previously studied by the same methods.

5725 **INTESTINAL ABSORPTION OF HEPARIN IN THE RAT AND GERBIL.** *(E.)* Engel, R. H. (Lederle Lab., Pearl River, N. Y.) and M. J. Fahrenbach. *Proc Soc Exp Biol Med* 129(3):772-777, 1968.

Studies showed that heparin can be rapidly absorbed by the intestinal mucosa of rats (CFE male) and gerbils (male, Mongolian) in the form of an oil-in-water emulsion. Preparations were administered intraduodenally at a dose of 100 mg heparin per kg body weight in volumes of 5-10 ml per kg. Absorption, as measured by turbidometric assay of clearing factor activity, began as early as 13 min after administration and increased linearly up to 60 min. The various surfactants used (5) were equally effective, but vegetable oils (corn, olive, peanut) were more effective than mineral oil, indicating that lipolysis is involved in the absorption process. Mactin B, although similar in structure to heparin, was not absorbed in the same way.

5726 **FUNCTIONAL DIFFERENCES BETWEEN THE SMALL AND LARGE INTESTINE, BASED ON THE DIFFERENT REACTIONS OF THE INTESTINAL MUCOSA TO ADMINISTRATION OF SOLUTIONS OF NONPENETRATING SOLUTES AND INCREASING OSMOLARITY.** *(Ger.)* Vogel, G. (Madaus Inst. Biol., Cologne-Merheim, Germany) and I. Stoeckert. *Pflueger Arch Ges Physiol* 303(3):262-273, 1968.

5727 **AN INFRARED SPECTROPHOTOMETRIC METHOD OF ESTIMATING POLYETHYLENE GYLCOL 4000.** *(E.)* Davies, M. W. (Royal Infirm., Cardiff, Wales) and R. Shields. *Gut* 9(5):617-619, 1968.

5728 **THE EFFECT OF CHYMOTRYPSIN ON THE GASTROINTESTINAL ABSORPTION, TISSUE PENETRATION, AND PHARMACOLOGICAL ACTIVITY OF DRUGS. I. PENETRATION OF PENICILLIN THROUGH THE BLOOD-BRAIN AND BLOOD-RETINAL BARRIERS.** *(E.)* Wohlman, A. (Denver Lab. Med. Res., Toronto, Canada), M. Syed and M. Ronchi. *Canad J Physiol Pharmacol* 46(6): 815-818, 1968.

5729 **STUDIES OF INTESTINAL IRON ABSORPTION USING A NEW PERFUSION APPARATUS.** *(Jap.)* Hara, Y. (Fac. Med., Kyushu U., Fukuoka, Japan), H. Wakasugi and T. Masuya. *Med Biol (Tokyo)* 77(3):85-89, 1968.

5730 **AN EXPERIMENTAL STUDY ON THE ABSORPTION OF VITAMIN B$_{12}$ BY THE SMALL INTESTINE.** *(Pol.)* Mastynska, M. (2nd Surg. Clin., Acad. Med. Poznan, Poland), J. Dzwonkowski and J. Woszczyk. *Pol Tyg Lek* 23(15):537-538, 1968.

5731 **EXCHANGE OF POTASSIUM IN THE HUMAN COLON.** *(It.)* Carratú, R., G. Arcangeli and R. Caprilli. *Arch Ital Mal Appar Dig* 34(6):593-601, 1967.

5732 **VENTROMEDIAL GLUCORECEPTOR SYSTEM.** *(E.)* Mayer, J. (Northeastern U., Boston, Mass.) and E. A. Arees. *Fed Proc* 27(6):1345-1348, 1968.

5733 **ENTERIC IRON ABSORPTION IN NORMAL AND SICK CHILDREN.** *(Ger.)* Doctor, S. (Pediat. Clin., U. Giessen, Germany), H. Rind and E. Gladtke. *Munchen Med Wschr* 110(49):2860-2865, 1968.

5734 **THE EFFECTS OF MUD APPLICATIONS ON THE ABSORPTIVE FUNCTION OF THE GASTROINTESTINAL TRACT.** *(Rus.)* Faitel'berg-Blank, V. R. (Inst. Balneol., Odessa, USSR) and A. L. Marshak. *Vop Kurort Fizioter* 33(5):444-447, 1968.

5735 **EFFECT OF COMBINED BALNEOLOGICAL THERAPY ON GASTRIC ABSORPTION.** *(Rus.)* Rashkovskaia, A. G. (S. M. Kirov Acad. Milit. Med. Leningrad, USSR). *Vop Kurort Fizioter* 33(5):439-444, 1968.

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5736 ACTION OF PANCREOZYMIN-CHOLECYSTOKININ ON THE ISOLATED GASTRIC MUCOSA OF THE BULLFROG.

(E.) Davidson, W. D. (Harbor Gen. Hosp., Torrance, Calif.), O. Urushibara and J. C. Thompson. *Proc Soc Exp Biol Med* 129(3):711-713, 1968.

The acid secretory response of bullfrog gastric mucosa to pancreozymin-cholecystokinin was compared to the response to gastrin and pentagastrin. Addition of pentagastrin (2.5 x 10^{-6} M) or pancreozymin-cholecystokinin (2.5 x 10^{-6} M) to the solution bathing the serosal side of the mucosa produced a maximal acid volume 30-45 min after incubation with the hormone (pancreozymin-cholecystokinin was 2-3 times as potent as pentagastrin). Porcine gastrin (2.5 x 10^{-8} M) produced a similar secretory response but was 25-35 times as potent as pancreozymin-cholecystokinin. Immunoassayable gastrin (0.026% by weight) was present in the pancreozymin-cholecystokinin but this could not account for the acid secretory response to the hormone. The similarity of the action of gastrin and pancreozymin-cholecystokinin is considered to be related to the identical amino acid sequence shared by both hormones.

5737 EFFECT OF DIET, ADRENALECTOMY, DIABETES, AND ACTINOMYCIN D ON EXOCRINE PANCREAS.

(E.) Snook, J. T. (Dept. Food Nutrit., Cornell U., Ithaca, N. Y.). *Amer J Physiol* 215(6):1329-1333, 1968.

Substitution of hexane-extracted whole-egg powder for casein in the diet of rats produced a statistically significant increase in the RNA content and the chymotrypsinogen, trypsinogen, amylase, and lipase activities of the pancreas. The chymotrypsinogen, trypsinogen, and lipase activities were the same in intact and adrenalectomized rats, but the pancreatic RNA content and amylase activity were reduced in adrenalectomized animals fed either diet. Induction of diabetes with alloxan and dehydroascorbic acid produced a marked decrease in pancreatic amylase activity but enhanced the response of the other enzymes to the substitution of whole-egg protein. The pancreatic RNA content was reduced in diabetic rats fed casein and elevated in diabetic rats fed whole-egg protein. The egg-induced increase in RNA was prevented by s.c. injection of 12 μg actinomycin D/day during adaptation to the egg diet; enzyme activities were not affected by this treatment. These results indicate that adaptation of pancreatic enzymes to whole-egg protein is not dependent on RNA synthesis mediated by adrenal hormones, insulin, or DNA.

5738 EXOCRINE FUNCTION OF THE CHICK PANCREAS AS AFFECTED BY DIETARY SOYBEAN MEAL AND CARBOHYDRATE.

(E.) Dal Borgo, G. (Dept. Anim. Sci., Washington State U., Pullman), A. J. Salman, M. H. Pubols and J. McGinnis. *Proc Soc Exp Biol Med* 129(3):877-881, 1968.

From age one day to 3 weeks, chicks were fed 4 different diets containing unheated or autoclaved soybean meal with either glucose or cornstarch as the source of carbohydrate. Amylase, trypsin, chymotrypsin, and lipase activities were determined on samples of pancreatic juice collected every 3 hr for 4 days through a cannula inserted into the main pancreatic duct. Chicks were then sacrificed and the pH of the duodenal contents measured. Daily secretion of pancreatic juice and the pH of the duodenal contents were higher for chicks fed unheated soybean meal, but the specific activities of all enzymes except trypsin were lower. The increased secretion of trypsin in the pancreatic juice of chicks fed unheated soybean meal is attributed to the intestinal trypsin inhibitor present in soybean meal. In contrast to the findings of Snook (*Fed Proc* 24, 941, 1965), a higher ratio of chymotrypsin to trypsin was found in the pancreatic juice of chicks fed unheated soybean meal. These higher values may be a result of using N-benzoyl-L-tyrosine ethyl ester rather than acetyl-L-tyrosine ethyl ester as the substrate in the chymotrypsin assay.

5739 EFFECT OF PANCREOZYMIN ON INSULIN AND GLUCAGON LEVELS IN BLOOD AND BILE.

(E.) Buchanan, K. D. (Dept. Med., U. Washington, Seattle), J. E. Vance, A. Morgan and R. H. Williams. *Amer J Physiol* 215(6):1293-1298, 1968.

A continuous infusion of 0.25 U/min secretin for 30 min produced a copious flow of pancreatic juice in dogs but did not cause any change in the levels of immunoreactive insulin or immunoreactive glucagon in the pancreatic venous blood. Rapid infusion of 75-100 Ivy units of pancreozymin caused a significant increase in both hormone levels, a rise in plasma glucose, and a decrease in free fatty acids. After administration of pancreozymin the concentration of immunoreactive glucagon was much higher in the pancreatic vein than in the jejunal vein. In pancreatectomized dogs pancreozymin had no effect on the secretion of immunoreactive glucagon, suggesting that pancreozymin has no effect on immunoreactive glucagon in the gut. The levels of immunoreactive insulin and glucagon in the bile corresponded with those in the blood.

5740 PANCREATIC SECRETION STIMULATED BY THE ACTION OF ALCOHOL ON THE GASTRIC ANTRUM.

(E.) Schapiro, H. (U. Tennessee Med. Units, Memphis), L. D. Wruble, J. W. Estes and L. G. Britt. *Amer J Dig Dis* 13(6):536-539, 1968.

In an attempt to elucidate the mechanisms by which alcohol produces acute pancreatitis, 4 dogs with total pancreatic fistulas, isolated innervated pouches of the pyloric gland area, gastrojejunostomies, and denervated Heidenhain pouches were subjected to perfusion of the antral pouch with a 10% alcohol solution. After a latent period of 10-30 min there was a 30% increase in the volume of pancreatic secretion and a 44% increase in pancreatic amylase. This is interpreted to be due to the release of a gastrin-like substance which acts directly on the pancreas. An increase in gastric secretion began 10-20 min after the pancreatic response. To prevent any stimulation of the acid-secretin mechanism via receptors in the proximal jejunum, 2 dogs were also provided with gastric fistulas. Results were about the same, indicating that the acid-secretin mechanism is not involved in producing the effects described above.

5741 HISTAMINE AND THE EFFECT OF GASTRIN ON GASTRIC ACID SECRETION IN MAN.

(E.) Levine, R. J. (Yale U. Sch. Med., New Haven, Conn.). *Fed Proc* 27(6):1341-1344, 1968.

Administration of Brocresine (4-bromo-3-hydroxybenzyloxyamine), an inhibitor of histidine decarboxylase, to 2 patients with Zollinger-Ellison syndrome resulted in a significant decrease in basal gastric acid secretion. The compound was administered at a maximum dose of 400 mg every 4 hr for up to 5 weeks. In the one case studied, there was no significant change in response to histamine administered during treatment, as measured by total acid output or mean acid concentration of 4 consecutive 15-min collections of gastric secretion. These results, in conjunction with similar studies in rats and the demonstration of histidine decarboxylase activity in human gastric tissue (a gastric carcinoid tumor and mucosa from the acid-secreting portion of a stomach removed shortly after death from a nonfasting patient), are taken as preliminary evidence that histamine may mediate the effects of gastrin on gastric acid secretion in man.

5742 **MODELS FOR THE “SECRETION” OF PEPSIN AND OTHER PROTEINS BY THE HUMAN STOMACH.** *(E.)* Makhlouf, G. M. (Lemuel Shattuck Hosp., Boston, Mass.), E. W. Moore and A. L. Blum. *Gastroenterology* 55(4):457-464, 1968.

Dose-response curves for the effect of infusions of gastrin II (2.08-20.83 μg/hr) and histamine acid phosphate (3.82-38.2 μg/kg/hr) on acid and pepsin secretion were constructed in a male subject weighing 66 kg. The calculated maximal responses for gastric acid and pepsin were not significantly different for the two stimulants. Unlike the ratio in animals, the pepsin acid ratio in man appeared to increase with increasing dose of either stimulant. Two models were then proposed to describe the elaboration of pepsin by the human stomach, both leading to nearly the same conclusions. In the first model, which was formally similar to that for acid secretion, pepsin secretion was viewed as a response to direct stimulation of peptic cells. In the alternative model, pepsin secretion was viewed as the dispersal of pepsin by the outflow of parietal fluid along the tubule. The assumption that the protein content of gastric secretion is composed of a peptic protein and a protein fraction associated with the passage of interstitial fluid was tested by analyzing the relation between the concentration of acid or sodium and the difference in the concentrations of total and peptic protein. The inverse proportionality of hydrogen ion concentration and the direct proportionality of sodium concentration to the concentration of the protein residue appeared to confirm this assumption.

5743 **CALCIUM IN NORMAL HUMAN GASTRIC JUICE: A FOUR-COMPONENT MODEL WITH SPECULATION ON THE RELATION OF CALCIUM TO PEPSIN SECRETION.** *(E.)* Moore, E. W. (Lemuel Shattuck Hosp., Boston, Mass.) and G. M. Makhlouf. *Gastroenterology* 55(4): 465-480, 1968.

The entry of calcium into the normal human stomach was studied in the same male subject described in the previous paper, still using both histamine and gastrin as stimulants. Preliminary data indicated that observed calcium was derived from both “secretory” and “nonparietal” sources, and that the concentration and output of both calcium and pepsin fell during stimulation. A 4-component model was then constructed to account for the observed calcium concentration at any given level of secretion. This model was based on the assumption that the observed calcium concentration represents the algebraic sum of 4 distinct fractions: ionized calcium from gastric interstitial fluid; soluble, diffusible calcium complexes from nonparietal fluid; calcium originally bound to albumin (also a nonparietal component); and calcium bound to or associated with pepsin, representing the secreted calcium component. A quantitative prediction was made for the total observed calcium concentration at any given level of secretory activity. The data appeared to conform to the predictions of the model in every respect. When estimated secretory calcium concentrations were subtracted from observed total calcium concentrations, the resulting nonparietal calcium concentrations were almost exactly those predicted by the model. Ionized calcium concentrations determined by an ion-exchange electrode also appeared to be compatible with the model. Having established a close correlation between prediction and observation, speculation as to the possible relation of calcium to zymogen structure and pepsinogen release became possible.

5744 **EFFECT OF PROLONGED ADMINISTRATION OF AN ANTICHOLINERGIC DRUG, PROPANTHELINE BROMIDE, ON GASTRIC HISTOLOGY AND ACID SECRETION.** *(E.)* Kaye, M. D. (Welsh Natl. Sch. Med., Cardiff), A. Jacobs, H. Ireson and D. Trevett. *Amer J Dig Dis* 13(11):994-1002, 1968.

The effect of prolonged administration of an anticholinergic drug, propantheline bromide, on acid secretion and gastric histology was studied in 160 adult male Wistar rats divided into 3 groups: control, oral administration and parenteral administration. Although anticholinergics are known to produce temporary inhibition of gastric acid secretion, it was found that after prolonged administration (17-30 weeks), gastric acid secretion, measured 48 hr after discontinuation of the drug, was unchanged in rats that had received propantheline orally (10 mg/kg/day) and was increased in those that had received the drug parenterally (5 mg/kg 12 times weekly s.c.). Although gastric mucosal atrophy has been reported following similar treatment, in this series the fundic mucosa showed an increase in thickness and surface area.

5745 **CORRELATION BETWEEN THE WEIGHT OF GASTRIC MUCOSA AND MAXIMAL ACID SECRETION.** *(E.)* Cooke, H. J. (VA Ctr., Los Angeles, Calif.) and A. R. Cooke. *Digestion* 1(4):209-212, 1968.

Eight dogs weighing between 13.2 and 21.3 kg were used to study the correlation between histamine-induced acid secretion from a Heidenhain pouch and mucosal weight. The size of the pouch was varied deliberately and the mucosal weights ranged from 7.0 to 20.5 g. A significant linear correlation ($r = 0.98$) was found between the maximal acid output in response to histamine (0.2-0.4 mg/kg/hr) and the weight of the pouch mucosa. Thus, the maximal acid output varied from 742 to 2638 μEq/15 min but the acid output/min/g varied only between 7.06 and 8.57 μEq and averaged 7.60 μEq for all 8 dogs. It is concluded that the weight of the gastric mucosa may be used as a simple and reliable index of maximal acid output.

5746 **STUDIES ON THE EFFECT OF PENTAGASTRIN ON GASTRIC SECRETION IN RATS.** *(Ger.)*

Klein, H. J. (Inst. Path., U. Cologne, Germany), T. Gheorghiu, H. Frotz and M. Eder. *Z Ges Exp Med* 148(2):124-130, 1968.

Studies in male Wistar rats with indwelling gastric catheters showed that dose-dependent maximal secretion of gastric juice in response to s.c. pentagastrin is obtained at doses of 40-50 μg in a volume of 1.0 ml saline (160-200 μg/kg compared to a maximal stimulatory dose of 6 μg/kg in man). Smaller doses of pentagastrin (0.25-10 μg) produced a moderate increase in acid secretion but no change in total volume, so that the HCl concentration rose, while larger amounts of pentagastrin (up to 500 μg) had an inhibitory effect on acid secretion, again with no effect on total volume. When the pentagastrin was diluted so that the same amount was given in 2-10 ml instead of 1.0 ml, the acid and water secretion were both decreased so that the HCl concentration remained essentially constant. When pentagastrin was given by repeated injection at 30 μg 4 times a day, the fasting secretory level increased somewhat after 8 injections followed by a slight decrease in HCl concentration. These results indicate that water and acid secretion in response to pentagastrin proceed via two distinct mechanisms.

5747 **RELATIVE POTENCY OF COMMERCIAL SECRETINS.** *(E.)* Stening, G. F. (VA Ctr., U. California Sch. Med., Los Angeles), M. Vagne and M. I. Grossman. *Gastroenterology* 55(6):687-689, 1968.

The relative potency of 4 batches of Gastrointestinal Hormone Laboratory (GIH) secretin and 4 batches of Boots secretin was studied by i.v. bioassay in 6 fasted dogs with pancreatic and gastric fistulas. A fifth batch of GIH secretin was randomly selected to serve as a reference standard. The 2 commercial secretins showed a marked difference in activity/U; one clinical unit of GIH secretin was 8-9 times more potent than one Crick, Harper and Raper unit of Boots secretin. However, the 5 batches of GIH secretin differed by less than 20% in potency and the 4 batches of Boots secretin, although much weaker/U, were also of similar potency. This indicates that the standardization of batches is satisfactory for each product and that the difference between them lies in the activity of the differing systems of unitage that they employ.

5748 **STUDIES ON THE ROLE OF SEROTONIN IN THE INHIBITION OF GASTRIC ACID SECRETION BY THE DUODENUM.** *(E.)* Wise, L. (Johns Hopkins U. Sch. Med., Baltimore, Md.), J. Burkholder, A. Zagaloff, M. Schuck and W. F. Ballinger II. *Ann Surg* 168(5): 824-830, 1968.

The effect of duodenal acidification on gastric secretion was studied in mongrel female dogs with innervated Pavlov pouches. These experiments confirmed that perfusion of the duodenum with 0.1 N HCl (120 ml/hr) causes gastric inhibition. In addition, i.v. injection of a duodenal mucosal extract caused a significant drop in gastric secretory volume and acid output, although not in acid concentration. Gastric inhibition after duodenal acidification was abolished in dogs by pretreatment with methyl-D-lysergic acid butanolamide (2.5 mg/kg i.m.) or reserpine (0.1 mg/kg i.m.), and considerably diminished by prior instillation of 2% cocaine. These results as well as other evidence suggest that gastric inhibition following duodenal acidification is at least partly effected through a neural mechanism by serotonin, although there was no change in blood serotonin levels.

5749 **BUFFERING CAPACITY OF FOOD IN RELATION TO STIMULATION OF GASTRIC SECRETION.** *(E.)* Williams, C. B. (Welsh Natl. Sch. Med., Cardiff, Wales), A. P. M. Forrest and H. Campbell. *Gastroenterology* 55(5):567-574, 1968.

Gastric acid and pepsin outputs following the ingestion of low- and high-protein meals were estimated in 4 dogs with innervated and 4 dogs with denervated gastric pouches. The neutralizing capacity of the meals was altered artificially by the addition of alkali, acid, and phosphate buffer. In all 8 dogs, an increase in neutralizing capacity resulted in an increased acid response; decreasing the neutralizing capacity decreased the acid response. The results indicate that the neutralizing capacity of food, regardless of its protein content, is an important factor in its ability to stimulate gastric acid secretion. In contrast, in dogs with denervated pouches, the pepsin output was the same after meals of meat or potato, and was not affected by addition of buffer, acid or alkali. In dogs with innervated pouches, meat evoked a significantly higher pepsin output than potato, but alterations in pH still had no effect.

5750 **CHONDROITIN SULFATE A AND SULFATED GLYCOPROTEINS IN DOG GASTRIC SECRETION FROM THE FUNDUS. I. ELECTROPHORETIC AND CHEMICAL CHARACTERIZATION.** *(E.)* DeGraef, J. (New York Med. Coll., New York City) and G. B. J. Glass. *Gastroenterology* 55(5):584-593, 1968.

The characterization of the sulfated aminopolysaccharides and sulfated glycoproteins in the juice and mucus from the dog gastric fundus was achieved by digestion of the gastric secretions with papain, precipitation of the papain digest with ethanol, fractionation of the papain digest with cetyl pyridinium chloride, and chemical and electrophoretic analysis of the fractions obtained before and after hyaluronidase digestion. A sulfated glycosamino-glycuronoglycan with the chemical composition and electrophoretic mobility of chondroitin sulfate A was present in larger amounts in dog gastric juice than in the mucus, where it was found only in traces. A sulfated glycoprotein containing hexosamine, hexoses, fucose, sialic acid, and sulfates was present in very small amounts in fundic juice, but in a large amount in gastric mucus. A low sulfated glycoprotein was found in gastric juice and mucus, while neutral glycoproteins of low negative charge were identified in small amounts in mucus and in still smaller amounts in fundic juice. No evidence was obtained for the secretion of a mucoitin sulfate.

5751 **CHONDROITIN SULFATE A AND SULFATED GLYCOPROTEINS IN DOG GASTRIC SECRETION FROM THE FUNDUS. II. TURBIDIMETRIC METHOD FOR THEIR QUANTITATION AND PRELIMINARY RESULTS OBTAINED IN DOG GASTRIC JUICE AND MUCUS.** *(E.)* DeGraef, J. (New York Med. Coll., New York City) and G. B. J. Glass. *Gastroenterology* 55(5):594-600, 1968.

A turbidimetric method is described for the quantitation of chondroitin sulfate A and sulfated glycoproteins in gastric juice and mucus. Preliminary studies in 4 dogs with Heidenhain pouches indicated that 90% of the sulfated mucosubstances in the gastric juice secreted during cholinergic stimulation could be accounted for by chondroitin sulfate A; in the mucus secreted under the same conditions, however, 84% of the sulfated mucosubstances were accounted for by sulfated glycoproteins and only 16% by chondroitin sulfate A. The concentration of chondroitin sulfate A in the gastric juice was increased from a fasting level of 7.1 μg/ml to 48.6 μg/ml by urecholine, but decreased to 4.2 μg/ml after histamine. The concentration of sulfated glycoproteins in the gastric juice was decreased by either histamine or urecholine (from a fasting value of 60.6 turbidity U/ml to 6.9-7.9). When the concentrations in the mucus were also considered, however, it was found that the total output of chondroitin sulfate A was not significantly affected by histamine but was markedly increased by urecholine, while that of sulfated glycoproteins was decreased by histamine and moderately increased by urecholine. The concentration and output of both sulfated mucosubstances in dog gastric juice are thus relatively high after urecholine stimulation and low after histamine.

5752 **A TECHNIQUE FOR PERFUSION OF THE ISOLATED CANINE PANCREAS: RESPONSES TO SECRETIN AND GASTRIN.** *(E.)* Hermon-Taylor, J. (London Hosp., England). *Gastroenterology* 55(4):488-501, 1968.

A technique for perfusion of the isolated canine pancreas is described and some of the functions of the preparation are discussed on the basis of an analysis of the physiological parameters in a series of some 160 perfusions; the findings obtained are compared with those described for the intact animal. The isolated pancreas responded to a sustained maximal secretin stimulus with a continuous flow of clear exocrine secretion (about 1 ml/min), concentrating bicarbonate 4-5 times compared with the perfusing blood. Supramaximal secretin stimulation did not suppress the maximal response, but a subsequent reduction in the rate of secretin infusion resulted in a significant increase in pancreatic secretion. Atropine had no effect on the response to secretin in this preparation. No significant difference was noted between the secretory or vascular effects of Boots and Vitrum secretin. Gastrin, either as an antral extract or as pure gastrin II, also had a direct effect on the pancreas, stimulating a flow of pancreatic juice rich in enzymes. The onset of the exocrine secretory response to both hormones was associated with a rise in metabolic activity and was preceded by vasodilation within the preparation.

5753 **THE EFFECT OF GASTRIN ANALOGS ON HCl SECRETION FROM VAGALLY INNERVATED AND DENERVATED PORTIONS OF THE STOMACH.** *(Pol.)* Konturek, S. (Med. Acad., Krakow, Poland) and K. Moczurad. *Acta Physiol Pol* 19(5):635-643, 1968.

The effects of two gastrin analogs, glycyltryptophanylmethionylaspartylphenylalanine (pentagastrin) and hydroxybenzoylalanyltryptophanylaspartylphenylalanine (tetrapeptide), on gastric acid secretion were compared with those of histamine in male cats with vagally innervated gastric fistulas or vagally denervated fundic (Heidenhain) pouches. With intact gastric innervation, the maximal secretory response was similar to all 3 stimuli (640-670 μEq HCl/15 min at 40 μg/kg of pentagastrin, 80 μg/kg of the tetrapeptide, or 400 μg/kg of histamine). However, additional HCl was produced by i.v. injection of either peptide (40-80 μg/kg) in cats previously given repeated submaximal doses of histamine (50 μg/kg/15 min s.c.). Following vagal denervation, the response to pentagastrin and the tetrapeptide was practically abolished (decrease from 250-300 to 10-12 μEq/15 min at doses of 10-20 μg/kg), while that to histamine (100 μg/kg) was decreased only from 200 to 45 μEq/15 min.

5754 **BRADYKININ AS AN INHIBITOR OF GASTRIC SECRETION.** *(Rum.)* Groza, P. (D. Danielopolu Inst. Norm. Path. Physiol., Bucharest, Romania), M. Corneanu, V. Buzoianu, S. Ionescu, C. Buşneac and L. Rusovici. *Fiziol Norm Pat* 14(4):311-316, 1968.

In 10 experiments on 2 dogs with Pavlov pouches, i.v. administration of 0.05 μg bradykinin produced significant inhibition of gastric secretion with corresponding decreases in free and total acidity and increases in pepsin and mucopolysaccharide concentrations. The degree to which gastric secretion was inhibited was proportional to the dose administered. This effect persisted 5 days after an 0.05 μg dose and 8 days after 0.075 μg. Studies of gastric secretion in 17 rats by Shay's method showed that in adrenalectomized rats histamine-induced gastric secretion was reduced with corresponding reductions in acid and pepsin concentrations; mucopolysaccharide concentrations remained essentially unchanged. Injection of 0.01 μg bradykinin i.p. into adrenalectomized rats produced only slight inhibition of gastric secretion but did increase total acidity and the pepsin concentration. Administration of hydrocortisone (1 mg/day for 3 days) to these adrenalectomized animals restored gastric secretion to normal levels while larger doses produced above-normal values. Free and total acidity were higher than in intact and adrenalectomized, untreated animals and pepsin concentrations increased to normal values. Administration of bradykinin to adrenalectomized, hydrocortisone-treated rats produced a 31% reduction in gastric secretion compared to 54% in intact animals. Corresponding decreases occurred in free and total acidity, the mucopolysaccharide concentration increased, and pepsin values remained essentially unchanged.

5755 **THE ACTIONS OF CAERULEIN ON GASTRIC SECRETION OF THE DOG AND THE RAT.** *(E.)* Bertaccini, G. (Inst. Pharmacol., U. Parma, Italy), R. Endean, V. Erspamer and M. Impicciatore. *Brit J Pharmacol* 34(2):311-329, 1968.

As expected from its chemical structure, cerulein is a potent stimulant of gastric secretion in the dog, rat and frog. In 13 dogs with denervated fundic pouches, cerulein was found to increase both the gastric secretory rate and the acid and pepsin output; the concentrations of acid and pepsin in the cerulein-stimulated gastric juice were greater than in control gastric juice. The threshold s.c. dose of cerulein was 0.15-0.5 μg/kg, while the threshold i.v. infusion rate was 0.25-0.5 μg/kg/hr. On a molar basis, cerulein is twice as active as human gastrin I on volume and acid output and four times as active on pepsin output. However, the sustained acid secretion stimulated by histamine infusion was inhibited by i.v. and s.c. cerulein. In 7 dogs, cerulein-stimulated acid secretion was inhibited by atropine (200 or 500 μg/kg) and unaltered by hexamethonium (5 mg/kg). The ratio of cerulein to gastrin activity is 7-30 in the rat when calculated on a molar basis, and is thus greater than in the dog; cerulein is three times as active as cholecystokinin-pancreozymin. In the perfused rat stomach preparation, the threshold dose of cerulein by rapid i.v. injection was 25 ng/kg, by i.v. infusion 0.25 μg/kg/hr, and by s.c. injection 0.25-0.5 μg/kg. Cerulein activity was sharply reduced in the rat by pretreatment with the histamine liberator 48/80 (0.2 ng/kg) and potentiated by pretreatment with the diamine oxidase inhibitor aminoguanidine (50 ng/kg). Rapid i.v. injection of cerulein during a primary histamine infusion enhanced and prolonged its effect. Cerulein (0.002-0.25 ng/ml) was found to increase the active transport of chloride in the isolated gastric mucosa of the frog. On a weight basis, human gastrin was at least 500 times less active than cerulein in its effect on active chloride transport.

5756 EFFECT OF VAGOTOMY ON HUMAN GASTRIC ACID SECRETION STIMULATED BY GASTRIN PENTAPEPTIDE AND BY HISTALOG.

(E.) Jepson, K. (Royal Infirm., Sheffield, England) and D. Johnston. *Gastroenterology* 55(6):665-669, 1968.

In a study on 10 male patients with duodenal ulcers (28-62 yr old) in which betazole hydrochloride (2 μg/kg s.c.) and pentagastrin (6-12 μg/kg/hr i.v.) were used to stimulate gastric secretion, maximal acid outputs in response to these agents were found to be almost identical, both before and 9 months after vagotomy and pyloroplasty. The vagotomy was complete in each of the 10 patients (insulin test). Preoperatively, the mean peak acid output in the 10 tests was 24.42 mEq/30 min with betazole hydrochloride and 24.11 mEq with pentagastrin. In the early postoperative period, betazole hydrochloride elicited a slightly higher mean peak acid output (11.79 mEq/30 min) than did pentagastrin (10.40 mEq/30 min) but the difference was not significant. Nine months after vagotomy, the mean peak 30-min response was 7.14 mEq to betazole hydrochloride and 7.39 mEq to pentagastrin, a reduction of 70.7% and 69.3%, resp., compared with the preoperative peak acid outputs. In 13 of the 14 tests, peak acid output was less than it had been in the early postoperative test; this decrease in secretion in the 9 months after vagotomy was statistically significant.

5757 EFFECT OF PROSTAGLANDIN E_1 ON GASTRIC SECRETION AND ULCER FORMATION IN THE RAT.

(E.) Robert, A. (Upjohn Co., Kalamazoo, Mich.), J. E. Nezamis and J. P. Phillips. *Gastroenterology* 55(4): 481-487, 1968.

In female Sprague-Dawley rats in which gastric secretions were collected for 4 hr after ligation of the pylorus, prostaglandin E_1, administered s.c. either by injection (50-400 μg/kg twice) or by constant infusion (0.5-2.0 μg/kg/min), inhibited the secretory volume, acid concentration and output, pepsin output, and fucose output, although pepsin concentration was not affected and fucose concentration increased. The degree of inhibition was dose dependent, with an ED_{50} of 200-300 μg/kg or 0.5-1.0 μg/kg/min, resp. Prostaglandin E_1 also strongly inhibited development of 2 types of ulcers in the rat: those produced by pyloric ligation (Shay ulcers) and those produced by administration of prednisolone (5 mg/day x 4). Mortality 21 hr after pyloric ligation was 0-14% in treated rats compared to 65% in controls, and perforation occurred in 21-29% of treated animals (90% of controls). Prostaglandin E_1 had no significant effect on body temperature. The mechanism of the antisecretory and antiulcer effect of prostaglandin E_1 is not known, but it may involve interference with formation of cyclic adenosine monophosphate in the stomach, or a change in gastric blood flow. Since prostaglandins are naturally occurring substances, they may play a physiological role in the regulation of gastric secretion and in gastrointestinal pathology.

5758 HUMORAL GASTRIC SECRETORY EFFECTS OF VAGOTOMY AND DISTAL ANTRECTOMY.

(E.) Dragstedt, L. R. II (VA Hosp., Des Moines, Iowa), R. E. Creagh-Larramendi, W. W. Jones, L. J. Lawson and L. T. Palumbo. *Arch Surg* 97(4):575-579, 1968.

In 9 mongrel dogs with Heidenhain pouches, antrojejunostomy of the Polya type without resection of antral mucosa increased the humoral phase of gastric secretion by 53% (range 16.89-124.48%). Since barium motor meal X-ray films failed to demonstrate gastric stasis, this result was probably due to increased reflux of intestinal content and elimination of the normal duodenal inhibition of humoral gastric secretion. Subsequent vagotomy further increased gastric secretion by 43%. In 9 of 11 other dogs with Heidenhain pouches, distal antrectomy, comprising only 11.4% of the total gastric mucosa, reduced hydrochloric acid secretion by 47% (range 1.65-77.60%), while in 2 dogs there was an increase. Vagotomy in 10 of these 11 dogs increased acid secretion by 40% (increase in 1 of 11). Subsequent removal of the remaining antrum (another 14% of the total gastric mucosa) reduced Heidenhain pouch secretion by 47% (range 10.9-100%), thus counteracting the effect of vagotomy. Vagotomy and distal antrectomy (25% gastrectomy) are recommended as an elective procedure for the average-risk patient with a duodenal ulcer.

5759 ASSAY OF GASTRIN BY MEANS OF ITS GASTRIC ACID STIMULATING ACTIVITY.

(E.) Blair, E. L. (Dept. Physiol., U. Newcastle upon Tyne, England), R. M. Keenlyside, D. J. Newell, J. D. Reed and D. D. Richardson. *J Physiol* 198(3):613-626, 1968.

A modified method, based on that of Blair *et al.* *(J Physiol* 163:47-48P, 1962), is described for the bioassay of gastrin activity in anesthetized cats. Under chloralose-ether anesthesia, a rubber tube is inserted into the stomach through an incision in the esophagus, a tape ligature is tied around the pylorus, the pancreatic and bile ducts are ligated, both vagi are cut in the neck, and the splanchnic nerves are cut extraperitoneally. Gastric secretions are washed out of the stomach at 15-min intervals with 100 ml of glycine solution (pH 6.2), and each dose of gastrin is injected i.v. in saline, adjusted so that the rate of acid secretion does not exceed 400 μEq/15 min. In this way, a gastrin of unknown potency can be assayed in terms of a stable Standard Gastrin Extract or of Synthetic Human Gastrin I; the latter was found to be 1594 times more active, weight for weight, than the Standard Gastrin Extract. The sensitivity of the method is such that it will detect as little as 100 ng of Synthetic Human Gastrin I, which is less than the minimum molar quantity so far detected by radioimmunological techniques. The index of precision of the assay was 0.14 when 300 μg of Standard Gastrin Extract was used as the background stimulus and 0.11 when 1000 μg of the extract was used. The gastrin activity of 12 "unknowns' could be determined with this precision in a 10-hr working day, making the method well suited to clinical studies.

5760 THE MECHANISM FOR GASTRIN RELEASE.

(E.) Dragstedt, L. R. (U. Florida Coll. Med.,

Gainesville), C. de la Rosa, E. R. Woodward, F. Herrera-Fernández and M. Tsukamoto. *Arch Surg* 97(5):816-825, 1968.

In dogs with both a Heidenhain pouch and an isolated, vagally innervated, antral pouch, separation of the antral mucosa from the muscle layer by surgical antroneurolysis or by infiltration of the antral submucosa with 5% phenol markedly inhibited gastric secretion in response to perfusion of the antral pouch with a liver suspension (average 30-43% decrease) but had little or no effect on the secretory response to perfusion of the pouch with acetylcholine (from no change to a 9% increase). Similar results were obtained in dogs with vagally denervated antral pouches. Gastric secretion from Heidenhain pouches increased markedly after bilateral transthoracic vagotomy, and increased still more in 4 of 5 dogs after subsequent antroneurolysis, both of which eliminate the vagal component of gastrin release but also cause stasis of food in the stomach. It is concluded that the release of gastrin in response to food is probably due to both vagal impulses and activation of a local antral mechanism. Vagal impulses probably act both directly, by stimulating the gastrin cells in the antral mucosa, and indirectly by increasing antral motility. The local mechanism is activated by contact of food with receptor cells which relay the stimulus to the gastrin cells by reflex arcs which extend into the submucosa. Contact of food with the antral mucosa seems to be a more potent stimulus than vagal impulses for the release of gastrin.

5761 **THE EFFECT OF SC 15396 ON GASTRIN STIMULATED PANCREATIC SECRETION.** *(E.)* Stening, G. F. (UCLA Sch. Med., Los Angeles, Calif.) and M. I. Grossman. *Proc Soc Exp Biol Med* 128(2):430-433, 1968.

The effect of SC 15396 (2-phenyl-2-[2-pyridyl]-thioacetamide) on gastrin-stimulated (400 mg/kg/hr) pancreatic and gastric secretion was investigated in 3 fasted conscious dogs with gastric and pancreatic fistulas. A dose of 2 mg/kg i.v. caused a marked inhibition of gastric secretion, but produced only a slight and statistically insignificant depression of pancreatic secretion. Pancreatic volume, bicarbonate output, and to a lesser extent bicarbonate concentration tended to be lower after SC 15396 than after saline, but most of the differences were insignificant. There was no difference in either protein concentration or protein output from the pancreas following a dose of SC 15396 or saline.

5762 **COMPARATIVE STUDY OF SOME STIMULANTS OF GASTRIC SECRETION.** *(Rus.)* Shevchenko, I. A. (S. M. Kirov Acad. Milit. Med., Leningrad, USSR). *Lab Delo* (9):559-562, 1968.

The effects of cabbage decoction, bread, bouillion, 5% ethanol, caffeine, histamine, insulin, and 10% NaCl on the composition of the stomach contents were compared in 120 normal males, aged 20-23 yr. Each of these 8 stimulants was given to a group of 15 fasting subjects. Of the stimulants administered orally, cabbage decoction produced the greatest increase in free HCl and total acidity and the largest decrease in the pH of the stomach contents. Cabbage decoction was followed by bread, bouillion, and caffeine in order of decreasing effectiveness. A 5% ethanol solution had the smallest effect. Of the parenterally administered stimulants, histamine caused the greatest increase in acidity, pepsin, gastric mucoproteins, and chlorides and a significant decrease in the pH of the stomach contents. It was followed by insulin and 10% NaCl in order of decreasing effectiveness.

5763 **A COMPARISON BETWEEN THE EFFECT OF PEPTAVLON AND HISTAMINE ON GASTRIC ACID SECRETION IN MAN AFTER SURGICAL VAGOTOMY.** *(E.)* Aagaard, P. (State Hosp., Copenhagen, Denmark). *Scand J Gastroent* 3(5):476-480, 1968.

Gastric acid secretion after i.v. infusion of peptavlon (pentagastrin; 0.1 µg/kg/min) and histamine (0.7 µg/kg/min) for 2 hr were compared in 20 patients who had been subjected to vagotomy and pyloroplasty for duodenal ulcer 6 months before. When the vagotomy was evaluated by the insulin test, 16 cases were negative and 4 were late positive (criteria of Hollander). The results revealed no statistically significant difference between histamine and pentagastrin for the maximal mean values of acid output, volume, or acidity. Maximal acid output generally occurred during the 2nd 15-min period after pentagastrin and the 4th 15-min period after histamine.

5764 **STATISTICAL STUDY OF THE CORRELATION BETWEEN ACID AND PEPSIN (K_{25}) IN HUMAN GASTRIC JUICE UNDER NORMAL AND PATHOLOGICAL CONDITIONS.** *(Fr.)* Lewin, M. (Bichat Hosp., Paris, France) and S. Bonfils. *Rev Franc Etud Clin Biol* 8(8):780-791, 1968.

The K_{25} ratio (pepsin secretion for 25 ml of primary acid secretion) was determined in 93 patients with duodenal ulcers, 17 with stomach ulcers, and 45 controls. These subjects were divided into 9 groups depending upon whether they were hypo-, hyper-, or normochlorhydric. In 21 randomly selected patients the K_{25} was determined before and after insulin stimulation. In addition, the K_{25} was determined for 10 patients with Zollinger-Ellison syndrome. The distribution of K_{25}'s among control and normochlorhydric groups approached a gaussian distribution while that of all other groups was skewed. Analysis of variance showed that among the total population there was no "principal effect of illness" on the K_{25} but that this effect was significant among patients with duodenal ulcers and hyper- or hypochlorhydria. The "effect of acidity" on the K_{25} differed among the groups studied and was very significant among patients with duodenal ulcers and hyper- or hypochlorhydria. The mean K_{25} was lowest among patients with Zollinger-Ellison syndrome. There was a direct relation between the dose of insulin and the increase it produced in the basal K_{25}. However, when the basal K_{25} exceeded 61, insulin either had no effect on the K_{25} or reduced it.

5765 **THE EXOCRINE PANCREAS AND BLOOD SUGAR MODIFIERS. I. EFFECT OF DIAZOXIDE, ALONE AND COMBINED WITH VICHY WATER, ON PANCREATIC AND BILIARY SECRETION IN THE RAT.** *(Fr.)* Debray, C. (Bichat Hosp., Paris, France), J. de La Tour, C. Vaille, C. Roze and M. Souchard. *Therapie* 23(3):649-662, 1968.

Ethylurethan-anesthetized male Wistar rats with biliary and pancreatic fistulas were given 15 mg diazepoxide in the femoral vein or two 5-ml doses of sodium bicarbonate (Grande-Grille Vichy water) 1 hr apart and, 15 min later, the same dose of diazepoxide. An increase in excorine pancreatic secretion occurred in the first hr after diazepoxide injection. This increase was not as large or long-lasting as that produced by Vichy water. The combination of diazepoxide and Vichy water increased pancreatic secretion to a level intermediate between that produced by either agent alone. Amylase activity remained unchanged. Vichy water had no effect on the progressive and long-lasting decrease in bile flow and increase in bilirubin secretion caused by diazepoxide. The amplitude of respiration decreased for 3 hr after diazepoxide injection; respiratory frequency decreased later. Vichy water tended to counter the changes in respiration induced by diazepoxide.

5766 EFFECTS OF A GASTRIN-CONTAINING ANTRAL EXTRACT ON ACID AND PEPTIC SECRETION IN THE RAT. *(Fr.)* Mignon, M. (U. Hosp., Seattle, Wash.), M. Lewin, L. S. Semb, S. Bonfils and L. M. Nyhus. *Rev Franc Etud Clin Biol* 13(6):575-583, 1968.

5767 CONCURRENT COLLECTION OF BILE AND PANCREATIC JUICE THROUGH THOMAS CANNULA. *(E.)* Wyman, J. B. (U. Minnesota, Rochester), K. G. Wakim, L. G. Bartholomew and J. C. Cain. *J Appl Physiol* 25(5):638-640, 1968.

5768 ELECTROPHORETIC STUDY OF PROTEASES IN GASTRIC JUICE AND THE GASTRIC MUCOSA OF DIFFERENT ANIMAL SPECIES. *(Fr.)* Cheret, A. M. (Bichat Hosp., Paris, France) and S. Bonfils. *Path Biol (Paris)* 16(23/24):1061-1070, 1968.

5769 ENZYMATIC DEGRADATION OF C-TERMINAL TETRAPEPTIDE AMIDE OF GASTRIN BY MAMMALIAN TISSUE EXTRACTS. *(E.)* Laster, L. (Natl. Inst. Hlth., Bethesda, Md.) and J. H. Walsh. *Fed Proc* 27(6): 1328-1330, 1968.

5770 ANTIBODIES TO GASTRIN. *(E.)* McGuigan, J. E. (Washington U. Sch. Med., St. Louis, Mo.). *Fed Proc* 27(6):1337-1340, 1968.

5771 STRUCTURE-ACTIVITY RELATIONSHIPS. *(E.,* Morley, J. S. (Imperial Chem. Indust. LTD., Cheshire, England). *Fed Proc* 27(6):1314-1317, 1968.

5772 PHYSIOLOGICAL ROLE OF GASTRIN. *(E.)* Grossman, M. I. (U. California Sch. Med., Los Angeles). *Fed Proc* 27(6):1312-1313, 1968.

5773 THE COMPARATIVE SECRETORY FUNCTION OF THE NORMAL GASTRIC MUCOSA FOLLOWING MAXIMAL STIMULATION WITH HISTAMINE OR TETRAPEPTIDE. *(It.)* Allegra, G. C. (Maggiore Hosp., Novara, Italy) and S. Maduli. *G Clin Med* 49(5):541-558, 1968.

5774 DIFFERENCES IN THE EFFECT OF SECRETIN BETWEEN OBESE SUBJECTS AND THOSE WITH NORMAL BODY WEIGHT. *(Ger.)* Raptis, S. (Ctr. Int. Med., U. Ulm, Germany), K. E. Schröder, M. Telib and E. P. Pfeiffer. *Wien Z Inn Med* 49(11):423-429, 1968.

5775 THE EFFECT OF BUFFER SOLUTIONS WITH VARYING pH VALUES ON THE ENTERIC PHASE OF GASTRIC SECRETION. *(Hun.)* Györffy, A. (Debrecen U. Med. Sci., Hungary), S. Dobi and T. Jávor. *Kiserl Orvostud* 20(4):357-359, 1968.

5776 THE MECHANISM OF THE INHIBITORY EFFECT OF EPINEPHRINE ON GASTRIC SECRETION IN RATS. *(Hun.)* Gáti, T. (Budapest U. Med. Sci., Hungary), P. Keszler and T. Zelles. *Kiserl Orvostud* 20(4):364-368, 1968.

5777 SOME ASPECTS OF THE DUODENAL EXOCRINE SECRETION IN RESPONSE TO PARTICULAR PHARMACOCHEMICAL STIMULI. *(It.)* Fontana, G. (Inst. Spec. Med. Path., U. Bologna, Italy), G. Gasbarrini and F. Faggioli. *Arch Pat Clin Med* 44(6):445-455, 1968.

5778 GASTROINTESTINAL HORMONES. *(E.)* Grossman, M. I. (VA Ctr., Los Angeles, Calif.). *Med Clin N Amer* 52(6):1297-1303, 1968.

See also: 5695, 5696, 5796, 5879, 6154, 6210, 6213, 6240, 6245, 6252, 6255, 6258, 6259, 6423, 6528, 6626

779 **THE IDENTITY OF VITAMIN A ESTERASE ACTIVITY OF RAT PANCREATIC JUICE.** *(E.)*
rlanson, C. (Div. Physiol. Chem., Chem. Ctr., U. Lund, weden) and B. Borgström. *Biochim Biophys Acta* 167(3):629-31, 1968.

n vitro studies with vitamin A palmitate and lyophilized rat ancreatic juice are reported which indicate that vitamin A sterase activity is possessed by two chromatographically disinct fractions of pancreatic juice. One fraction, which also ydrolyzes emulsified triolein and is probably identical with ipase, attacks emulsified vitamin A palmitate and is markedly nhibited by bile salts; the other fraction, which elutes slightly arlier with 0.15 M NaCl and hydrolyzes micellar monoolein, ttacks only bile salt-dispersed vitamin A palmitate and is probbly identical with a recently discovered carboxylic ester hydrolase. The emulsified substrate was produced by dissolving 10 μmoles of vitamin A palmitate in 2 mg hexadecane and dispersing this in 1.0 ml 0.05 M Tris-HCl buffer (pH 8.6) by sonication. The bile salt-dispersed substrate was obtained by sonicating 10 μmoles of vitamin A palmitate in 1.0 ml of 0.15 M phosphate buffer (pH 6.3) containing 6 mM sodium taurodeoxycholate. Lyophilized rat pancreatic juice was separated either by gel filtration or by columnchromatography on Sephadex.

5780 **FERMENTATION OF D-TAGATOSE BY LACTOBACILLI IN THE HUMAN INTESTINE.** *(Rus.)*
Lenzner, A. A. (Dept. Microbiol., Tartu State U., Estonian SSR), H. P. Lenzner and M. A. Toom. *Uch Zap Tartu Gos Univ, Tr Med* 18(215):250-254, 1968.

5781 **CORRELATIONS BETWEEN MECHANICAL AND ELECTRICAL ACTIVITY IN THE DUODENOJEJUNAL TRACT OF THE CAT.** *(It.)* Abbadessa, S. (Inst. Gen. Physiol., U. Palermo, Italy) and I. Di Gregorio. *Boll Soc Ital Biol Sper* 54(8):748-751, 1968.

In fasting cats of both sexes with exteriorized duodenojejunal loops (12-15 cm long) which were perfused with saline (18 ml/min) at a pressure of 10 cm H_2O) while preserving the innervation and blood supply, pressure changes within the loop were recorded by means of pressure transducers and correlated with changes in electrical activity recorded by means of silver needle electrodes imbedded in the small intestinal wall (3 cm and 11-13 cm from the pylorus, resp.). These recordings revealed 3 types of contractile (pressure) waves: small rhythmic oscillations amounting to 5-15 cm H_2O at a frequency of 13-16/min; broader variations in the basal pressure; and peaks reaching 30-50 cm H_2O or higher. The first type was found to correspond to a basal, rhythmic, electrical activity having the same frequency and an amplitude of 1-1.5 mV, while contractile peaks of the 3rd type were consistently preceded by electrical potential spikes having an amplitude of 0.3-2.0 mV. The speed of propagation of the slow electrical activity between the two implanted electrodes was calculated to be 2.2-2.8 cm/second.

5782 **CONTINUOUS ELECTRONIC RECORDING OF PRESSURE CHANGES IN THE BILE DUCTS.** *(Ger.)* Marth, W. (St. Markus Hosp., Frankfurt a.M., Germany). *Z Ges Exp Med* 148(2):108-114, 1968.

An electronic technique is described for the continuous measurement of pressure changes in the biliary tract following cholecystectomy. In this technique, the outer arm of the T-tube used for biliary drainage is connected to a Teleperm ND 100 pressure transducer, the entire system is filled with water, and variations in pressure are recorded on a Kompensograph L 144 x 144 at a paper speed of 120 mm/hr. Examples are given of recordings made an average of 14 days after cholecystectomy in patients with well-established drainage from the common bile duct. The technique was used to study the effect of anesthetic premedication, morphine and its derivatives, deep anesthesia and cholecystokinin on biliary tract motility, the action of the sphincter of Oddi, and the cholangiographic configuration of the ampulla of Vater. It is suggested that the technique would be particularly valuable in following the course of pathological biliary tract pressures, such as those caused by Type III waves, over relatively long periods.

5783 **THE MOTILITY OF THE COLONIC HAUSTRA IN THE GUINEA-PIG.** *(E.)* Pace, J. L. (Dept. Anat., Royal U. Malta). *Rass Fisiopat Clin Ter* 40(2):99-103, 1968.

In vivo cinematography was used to study the living proximal colon of a male guinea pig lightly anesthetized with N_2O/O_2/ ether, and the movements of the whole proximal colon were compared with those of the taeniae and haustra. Colonic movement was inhibited by deep anesthesia or intestinal constriction, and stimulated by light physical contact. There were no true peristaltic waves, the colonic movements consisting rather of small segmentation-like waves which moved the fecal contents backwards and forward. These waves were produced by the formation of haustral clefts between the taeniae, clefts which appeared and disappeared simultaneously in various parts of the colon in no orderly sequence. Each haustral cleft took 3.75 seconds to form and disappear, and the rate of formation was 14-16/minute. Some of the haustral clefts, however, were more marked and never became completely obliterated. In contrast to the haustra, the taeniae were stationary and showed no signs of contraction or relaxation. The distance between two fixed points on a taenia did not vary during colonic movement. It is concluded that, at least in the guinea pig, most of the haustra are functional in nature, appearing and disappearing but being constantly reformed at the same site.

5784 **EFFECT OF ATROPINE ON PARASYMPATHETIC RESPONSES OF THE GASTROINTESTINAL TRACT OF THE DOG.** *(E.)* Goldenberg, M. M. (Norwich Pharm. Co., N. Y.) and R. H. Burns. *Arch Int Pharmacodyn* 174(2):342-349, 1968.

Contractions of the stomach, duodenum, and colon in response to electrical stimulation of the vagus and pelvic nerves (voltage response curves) were studied in 32 anesthetized mongrel dogs (pentobarbital sodium) before and after atropine. Organs differed in the characteristic responses to extrinsic nerve stimulation: the stomach showed an immediate spike-like contraction which was followed by similar contractions, the duodenum displayed either an initial relaxation or delay followed by increased amplitude and frequency of contraction; and the colon showed 1 spike-like contraction which was either maintained or slowly declined during stimulation. Atropine, 0.1 mg/kg, abolished vagally-induced contractions of the stomach while similar doses depressed contractions of the duodenum but failed to inhibit them completely. The contractile response of these 2 organs to exogenously administered acetylcholine was blocked. Atropine, 0.01-5.0 mg/kg, diminished the contractile response of the colon to pelvic nerve stimulation. However, an atropine-resistant contractile component of the colon was uncovered which differed in shape and time course from the control response. Hexamethonium, 5 mg/kg, diminished but did not abolish these atropine-resistant responses. The possibility that a noncholinergic acetylcholine transmitter may mediate the atropine-resistant contractile responses in several viscera was discussed.

5785 **ATROPINE EXCITATION OF ESOPHAGEAL SMOOTH MUSCLE.** *(E.)* Christensen, J. (Dept. Int. Med., U. Iowa, Iowa City) and G. F. Lund. *J Pharmacol Exp Ther* 163(2):287-289, 1968.

Very large concentrations of atropine sulfate, but not atropine methyl bromide, elicited powerful rhythmic contractions of opossum esophageal circular smooth muscle *in vitro*. The response was dose-related, reversible by washing, and not subject to tachyphylaxis; it persisted during a long period of exposure. The mean threshold concentration of l-hyoscyamine, 4.59 x 10^{-5} g/ml, was significantly lower than that for racemic atropine sulfate, 3.22 x 10^{-4} g/ml. The response to atropine was not affected by nicotine, hexamethonium, tolazoline, dihydroergotamine, propranolol, d-tubocurarine, tripelennamine, methysergide or tetrodotoxin, nor by local anesthetics except at very high oncentrations. It persisted during histamine tachyphylaxis. he findings indicate that atropine acts directly on esophageal nuscle to produce the contractile response.

786 STRUCTURES ESSENTIAL FOR THE EFFECT OF CHOLECYSTOKININ ON THE GUINEA PIG SMALL INTESTINE *IN VITRO*. *(E.)* Hedner, P. (U. Hosp., Lund, Sweden) and G. Rorsman. *Acta Physiol Scand* 74(1/2): 58-68, 1968.

The stimulatory effect of cholecystokinin on peristalsis in trips of guinea pig ileum *in vitro* was blocked by atropine, norphine, and tetrodotoxin, but not by hexamethonium or pretreatment with reserpine. Desensitization to serotonin did not alter the cholecystokinin effect. Increasing the intraluminal pressure or infusing serotonin elicited a propagated contraction n the circular muscle layer, followed by a 20-60 sec refractory period; no such refractory period was registered after a cholecystokinin-induced response. Although the mechanism of action emains unknown, the stimulatory effect of cholecystokinin on he longitudinal and circular muscle layers of the guinea pig leum is apparently mediated via a cholinergic neural pathway without cholinergic synapses, and does not involve release of erotonin.

5787 THE MOVEMENT OF THE STOMACH AND JEJUNUM DURING NAUSEA AND VOMITING. *(Jap.)* Ohashi, K. (Okayama U. Med. Sch., Japan). *J Physiol Soc Japan* 30(9):736-750, 1968.

n experiments on anesthetized dogs (80 mg/kg chloralose) with a plastic window set into the abdominal wall, the onset of apomorphine-induced nausea and vomiting (0.1-0.2 mg/kg s.c.) was found to be accompanied by pronounced changes in gastrointestinal motility. The pyloric part of the stomach developed a persistent contraction, while the body and fundus showed spherical dilatation, on which were superimposed peristaltic and antiperistaltic waves which failed to evacuate the stomach contents. Nausea and vomiting were both accompanied by an initial increase in gastric motility, but in the case of nausea this was followed by an inhibitory phase. The jejunum, on the other hand, showed either a marked increase or a marked decrease in motility at the time of vomiting, preceded in either case by a phase of mixed stimulation and inhibition. Subsequent experiments in which the vagus and the splanchnic and lumbar sympathetic nerves were successively cut before administration of apomorphine indicated hyperexcitation of both the sympathetic and the parasympathetic nuclei of the central nervous system during nausea, resulting in a mixture of inhibitory and stimulatory influences on gastrointestinal motility. Thus, vagotomy resulted in a marked inhibition of both gastric and jejunal motility just before and during vomiting, preceded by a phase of increased motility. Section of only the splanchnic and lumbar sympathetic nerves had little effect on the gastrointestinal response to apomorphine, but all response was abolished when the vagus was also severed. In decerebrate dogs (transsection of the spinal cord at the 3rd thoracic segment), apomorphine produced marked stimulation of gastric motility associated with jejunal inhibition.

5788 THE MOVEMENT OF THE COLON AND GALLBLADDER DURING NAUSEA AND VOMITING. *(Jap.)* Ohashi, K. (Okayama U. Med. Sch., Japan). *J Physiol Soc Japan* 30(10):771-778, 1968.

Manometric studies on dogs anesthetized with chloralose (80 mg/kg) indicated that the onset of apomorphine-induced nausea and vomiting (0.1-0.2 mg/kg s.c.) was accompanied by moderate changes in the motility of the colon and gallbladder which did not reach the magnitude of those previously observed in the stomach and small intestine. The effects were also more pronounced in the proximal than in the distal colon. While gallbladder motility was increased just before vomiting and decreased during and after vomiting, colonic motility could be either increased or decreased at the time of vomiting. Subsequent experiments in which the vagus, pelvic, and splanchnic and lumbar sympathetic nerves were successively cut before administration of apomorphine showed that the vagus has both a stimulatory and an inhibitory effect on both the gallbladder and the large intestine, while the sympathetic nerves have an inhibitory effect on the gallbladder and the large intestine, while the sympathetic nerves have an inhibitory effect on the gallbladder and a mixed (but mostly inhibitory) effect on the colon. Although the effect of the vagus and pelvic nerves was occasionally mildly inhibitory, they had a generally stimulatory effect on large intestinal motility. Thus, after section of both the vagus and pelvic nerves, apomorphine produced marked inhibition of large intestinal motility, although after section of only the vagus nerve motility was increased by apomorphine.

5789 AN ELECTROMANOMETRIC STUDY OF THE DUODENUM: RESEARCH UNDER BASAL CONDITIONS AND AFTER SPECIAL STIMULATION, ALONG WITH A STATISTICAL ANALYSIS OF THE FINDINGS. *(It.)* Gasbarrini, G. (Inst. Spec. Med. Path., U. Bologna, Italy), N. Melchionda, F. Benfenati, B. A. Mantovani and M. G. Marlettini. *Arch Pat Clin Med* 44(2):93-173, 1968.

Duodenal motility measurements were made on 52 subjects, aged 19-65 yr (30 females and 22 males). These included 15 normal subjects, 10 with duodenal ulcers, 17 with gallbladder disease, 1 with biliary tract disease, 2 with hepatitis, 2 with anorexia nervosa, 1 with Simmond's disease, and 1 with cerebral atrophy and arteriosclerosis. Measurements were made with a balloon-tipped or open-tipped catheter connected to an electromanometer which transmitted pressure to a recorder. Motor activity was reduced in the duodenal bulb in ulcer patients and those with gallbladder hypotonia, both under basal conditions and after administration of substances which stimulate duodenal motility. Motor activity was increased, even under basal conditions, in patients with gallbladder hypertonia. Cholecystokinetic agents reduced motor activity in the bulb in all cases. In other segments of the duodenum simple and complex phasic waves (type I activity) accounted for 90% of the activity in controls and for 98% of that in patients with ulcers, gallbladder hypertonia and chronic duodenitis. Slow increases from the base line (type III activity) were not found in patients with hepatitis, chronic pancreatitis, and chronic cholecystitis with gallbladder hypotonia. 5-Hydroxytryptamine produced type I activity superimposed on type III in ulcer patients and those with gallbladder disease and gallbladder hypertonia. The same type of activity, but less intense, occurred in patients with gallbladder hypotonia, hepatitis, and chronic pancreatitis. No features of the response typical of normal subjects were found in patients with hiatus hernia. Abnormalities in type I and type III activity occurred in patients with ulcers and those with chronic gallbladder disease and gallbladder hypertonia after intraluminal administration of acetylcholine. The response to acetylcholine was weaker than normal in patients with gallbladder hypotonia, hepatitis, and pancreatitis. Analysis of the data by factorial analysis of variance showed that the findings in each segment differed from one group to another, the drugs employed had different effects on different groups, the response of the 4 duodenal segments to stimulation differed, and the effect of a single stimulation differed according to group and segment.

5790 **A STUDY OF HUMAN GALLBLADDER MUSCLE *IN VITRO*.** *(E.)* Mack, A. J. (Victoria Infirm., Glasgow, Scotland) and J. K. Todd. *Gut* 9(5):546-549, 1968.

In a physiological and pharmacological investigation of the contractility of the smooth muscle of human gallbladder, strips were suspended in an organ bath containing modified Krebs solution at 37C and gassed with 95% O_2:5% CO_2. Rhythmic spontaneous contraction and relaxation were observed along with sustained tone which sometimes showed slow but significant changes. Circular muscle was more active than longitudinal. Acetylcholine caused contraction at concentrations as low as 1 mμg/ml; the contraction reached a maximum in 3 min and gradually fell off over a 10-min period. This effect was potentiated by physostigmine and abolished by atropine. Histamine (1 μg/ml) caused a contractile response similar to that to acetylcholine and resembling the response in intestinal smooth muscle; it was not affected by atropine, but was abolished by mepyramine maleate. 5-Hydroxytryptamine (1-100 μg/ml) had no effect on the preparation, while barium chloride (100 μg/ml) caused a rise in tone with an increase in the amplitude of spontaneous contraction. Morphine (10-100 μg/ml), nicotine (100 μg/ml), glyceryl trinitrate (1-1000 μg/ml), and sodium nitrite (1-1000 μg/ml) all had no effect. Physiological doses of catecholamines also had no effect, and even very high doses could not reduce the response to acetylcholine. Gastrin (0.1-1.0 μg/ml) did not elicit a contractile response; at high doses, it reduced the amplitude of spontaneous contractions and reduced the contractile response to acetylcholine. Secretin (0.1 U/ml) caused a slight contraction, while pancreozymin (0.025-0.5 U/ml) gave a response which, although powerful, was slower than that of acetylcholine. The pancreozymin effect was not altered by atropine or hexamethonium. The lack of response to adrenergic agents and nitrites and the sensitivity to cholecystokinin are the chief features which distinguish gallbladder muscle from intestinal muscle.

5791 **STUDY OF THE MOTILITY OF THE NORMAL HUMAN DUODENUM UNDER BASAL CONDITIONS BY MEANS OF ELECTROMANOMETRY.** *(It.)* Gasbarrini, G. (Inst. Spec. Med. Path., U. Bologna, Italy), N. Melchionda, F. Benfenati, A. De Vecchis and A. Venerato. *Arch Pat Clin Med* 44(4):271-309, 1968.

Duodenal motility measurements were made with either a balloon-tipped catheter or an open-tip catheter connected to an electromanometer which transmitted the pressure to a recorder. Radiological studies (roentgenocinematography or roentgenotelevision) were made simultaneously with studies of electrical activity in the duodenal wall. Motor activity in the duodenum can be classified into: 1) simple or complex phasic waves (type I); 2) slow increases in the base line (type III); and 3) polymorphic waves; types II and IV are not found in the duodenum. In the duodenal bulb, all 3 types of activity were found but type III was rare. In other segments of the duodenum about 90% of the type I activity consisted of simple or complex phasic waves in brief repeated sequences and prolonged arrhythmic waves. Isolated waves accounted for 8% and prolonged rhythmic sequences for 2%. The basal rhythm had a frequency of 10-12/min. Type III activity occurred rarely. The basal pressure at sites at which contrast medium was stopped and at functional sphincters was the same as that in other parts of the duodenum.

5792 **THE ACTIONS OF CAERULEIN ON THE SMOOTH MUSCLE OF THE GASTROINTESTINAL TRACT AND THE GALLBLADDER.** *(E.)* Bertaccini, G. (Inst. Pharmacol., U. Parma, Italy), G. De Caro, R. Endean, V. Erspamer and M. Impicciatore. *Brit J Pharmacol* 34(2): 291-310, 1968.

Dogs, pigeons and rats were given s.c. and i.v. injections of cerulein in a study of the effects of the polypeptide on gastrointestinal motility, as indicated by vomiting and bowel evacuation. In the intact conscious dog, emesis and bowel evacuation were induced by 0.4-0.5 μg/kg i.v. and 3-4 μg/kg s.c.; 10 and 50 μg/kg cerulein in pigeons and 20-1000 μg/kg in rats failed to affect motility. In the guinea pig, 0.3 and 1.5 ng/kg injected i.v. and less than 1 ng/kg/min infused i.v. stimulated the gallbladder *in situ*; 0.03-2 ng/ml Krebs solution elicited a contractile response in the isolated gallbladder preparation. These findings demonstrate that the gallbladder is suitable for the bioassay of cerulein and related peptides. At doses as low as 1-5 ng/kg i.v., cerulein has a spasmogenic action on jejunal loops in the dog; at slightly higher doses, it is also effective in the cat. Stomach and large intestine are less sensitive. Cerulein has a significant spasmogenic effect on the rat pylorus, but it relaxes the sphincter of Oddi in the guinea pig. Isolated gastrointestinal preparations are relatively insensitive to the polypeptide and tachyphylaxis readily occurs. The spasmogenic effect of cerulein on the gallbladder is atropine-resistant. In all *in situ* and isolated organ preparations the effects of cerulein are similar to those of cholecystokinin-pancreozymin, although cerulein is more active on a molar basis. Human gastrin I has negligible activity when compared to cerulein. It is possible that the experimental results with cerulein may be valuable in investigating the mode of action of cholecystokinin-pancreozymin and gastrin.

5793 **THE MECHANICS OF BILE FLOW AND ITS RELATION TO THE MOTOR FUNCTION OF THE GUT: STUDIES ON CONSCIOUS DOGS.** *(Ger.)* Aziz, O. (Inst. Physiol., Free U., Berlin, Germany), D. Kaiser and F. J. Haberich. *Pflueger Arch Ges Physiol* 304(2):136-151, 1968.

Continuous pressure and flow measurements were made in the distal part of chronically implanted bile-duct loops in 3 adult dogs during constant-pressure infusions of Ringer's solution after fasts of 14-16 hr and at various intervals after meals. The critical closing pressure of Oddi's sphincter was about 140 mm H_2O in fasted dogs but only 60 mm H_2O 30 min after a meal. This decrease in muscle tone was associated with an increase in the amplitude of duodenal contractions. In experiments in which the hydrostatic pressure in the distal part of the loop was 10-20 m H_2O higher than the basal value, duodenal contractions oc- rred very regularly at 13-16 sec intervals 30 min after a meal. fter 3-4 hr these contractions occurred 3 sec apart or less. fter 14-16 hr fasts the contractions were irregular and backflow bile increased during this period. A heavy flow of bile with casional irregular decreases started when the dogs were offered lump of sugar before their meal of meat and rice. This was obably caused by a conditioned reflex which produced such laxation in the muscles of the bile duct that complete con- actions could not occur. A regular flow rhythm was only es- blished about 7 min after the meal.

794 **MOTOR FUNCTION OF THE LARGE INTESTINE DURING STIMULATION OF DUODENAL ECEPTORS.** *(Rus.)* Vorobieva, N. M. (Sci.-Res. Inst. Nutrit., iev, USSR). *Biull Eksp Biol Med* 66(9):11-13, 1968.

n 4 dogs with fistulas of the duodenum and large intestine, vhich had been fasted for 18-20 hr, duodenal receptors were timulated by blowing 30-50 ml air at a pressure of 16-30 mm Ig through the duodenal fistula or by introducing 10 ml or ess of a 10% peptone solution, 2% soap solution, or 5, 10, or 0% sucrose solution. After a latent period of 30-100 sec, nechanical stimulation of the duodenal receptors during rest roduced type I and II contractions in the large intestine. These contractions lasted for from 3-5 min to 8-9 min when ir was introduced for 1-2 min. Introduction of air during nild duodenal contractions increased the tonus of the large ntestine and the amplitude of its contractions. Similar but nore pronounced effects were seen after chemical stimulation. Reactions produced by peptone and soap lasted 8-15 min and -16 min, resp., and were dependent upon the functional state of the intestine. Sugar solutions stimulated contractions in the arge intestine in only 15 out of 24 experiments.

795 **STATISTICAL ANALYSIS OF ELECTROMANOMETRIC FINDINGS MADE ON THE UODENUM USING A PARTIALLY HIERARCHIC, MULTI- ACTORIAL DESIGN (SPLIT-PLOT) AND NONORTHOGONAL ACTORIAL ANALYSIS (SCHEFFE'S TEST).** *(It.)* Ielchionda, N. (Inst. Spec. Med. Path., U. Bologna, Italy). *G lin Med* 49(5):496-519, 1968.

lectromanometric studies were carried out at 4 levels of the uodenum (the duodenal bulb, the first and second segments f the descending part of the duodenum, and the end of the uodenum) in 28 subjects before and after stimulation with erotonin, acetylcholine, $MgSO_4$, and cholecystokinin. The ubjects were divided into 4 groups of 7 each: 1) normal ontrols, 2) patients with peptic ulcer, 3) those with gallbladder isease and gallbladder hypertonia, and 4) those with gallbladder isease and gallbladder hypotonia. Analysis of variance for the ercent motor activity and the mean amplitude showed that here were statistically significant differences between the 4 roups studied, the sites at which measurements were made, nd measurements made before and after stimulation. The sig- ificance of differences at the 4 different sites and differences roduced in pressures before and after stimulation differed from ne group to another. There were also significant differences n the responses of the 4 sites to stimulation, and the effect of ach drug differed from group to group and site to site.

5796 **STUDIES OF CHOLECYSTOKININ AND ANTICHOLECYSTOKININ ACTIVITY: CONCLUSIONS BASED ON PERSONAL RESEARCH.** *(It.)* Azzaroli, P. (Inst. Spec. Med. Path., U. Bologna, Italy). *Riv Gastroent* 19(6, Suppl.):893-912, 1967.

Cholecystokinin and anticholecystokinin were determined biologically and by electrophoresis in the serum and urine from normal subjects under basal conditions and after duodenal stimulation by direct instillation of sorbitol or corn oil. The results of biological determinations showed that both serum and urinary cholecystokinin activity increased more and for a longer time after corn oil stimulation. However, attempts to correlate the amount of the hormone with the rate of bile flow were unsuccessful. Electrophoretic separation of urinary cholecystokinin from urinary anticholecystokinin followed by biological determination of their respective activities showed that corn oil produced a slight increase in urinary cholecystokinin activity and a large decrease in that of urinary anticholecystokinin. Sorbitol, on the other hand, caused a significant increase in urinary cholecystokinin activity and a slight, but variable, decrease in that of urinary anticholecystokinin. Biological determination of these hormones gave precise reproducible results, while electrophoresis and evaluation of the area under the curve did not.

5797 **MOTILITY OF THE ANTRUM, PYLORUS AND DUODENUM IN THE RABBIT.** *(Ger.)* Fleischer, K. (Inst. Pharmacol., U. Tubingen, Germany) and K. F. Sewing. *Naunyn Schmiedeberg Arch Pharm Exp Path* 260(4):275-283, 1968.

5798 **EFFECT OF PARAPENZOLATE BROMIDE (N-METHYL-4-PIPERIDYL BENZYLATE METHYL BROMIDE) ON BILIARY TRACT MOTILITY.** *(Fr.)* Paris, J. (U. Hosp. Ctr., Lille, France), A. Robelet, Y. Salembier and M. Wantiez. *Therapie* 23(3):609-618, 1968.

5799 **CONTRIBUTION TO THE STUDY OF THE ELECTRICAL ACTIVITY OF THE DUODENUM.** *(It.)* Labo, G. (Inst. Spec. Med. Path., U. Bologna, Italy), L. Barbara, G. A. Lanfranchi and P. Azzaroli. *Arch Ital Mal Appar Dig* 34(6):570-576, 1967.

5800 **THE ELECTRICAL ACTIVITY OF THE DUODENUM IN THE DOG.** *(It.)* Barbara, L. (Inst. Spec. Med. Path., U. Bologna, Italy), F. Faggioli, G. Fontana, G. Galletti and G. A. Lanfranchi. *Arch Ital Mal Appar Dig* 34(4):396-405, 1967.

5801 **A NEW TECHNIQUE FOR THE STUDY OF DUODENAL MOTILITY.** *(It.)* Gasbarrini, G. (Inst. Spec. Med. Path., U. Bologna, Italy), N. Melchionda, P. Sassi and F. Benfenati. *Arch Ital Mal Appar Dig* 34(6): 538-556, 1967.

5802 **EFFECT OF 5-HYDROXYTRYPTAMINE ON GALLBLADDER MOTILITY.** *(It.)* Lanfranchi, G. A. (Inst. Spec. Med. Path., U. Bologna, Italy), L. Barbara, G. P. Gavelli, P. Azzaroli and F. Rossi. *Arch Ital Mal Appar Dig* 34(6):515-529, 1967.

MOTILITY

5803 BEHAVIOR OF GASTROINTESTINAL MOTILITY FOLLOWING ADMINISTRATION OF SEROTONIN OR OF A NEW SYNTHETIC SEROTONIN ANTAGONIST. *(It.)* Bayeli, P. F. (Inst. Gen. Clin. Med., U. Siena, Italy), G. Nieri, M. Montagnani. *Arch Ital Mal Appar Dig* 34(6):587-593, 1967.

See also: 5782, 5847, 5883, 5956, 6154, 6244, 6245

5804 **BILE ACIDS AND LIPID METABOLISM. III. INFLUENCE OF BILE ACIDS ON PHOSPHOLIPIDS IN LIVER AND BILE OF THE ISOLATED PERFUSED DOG LIVER.** *(E.)* Swell, L. (VA Hosp., Richmond, Va.), C. C. Bell Jr. and C. Entenmann. *Biochim Biophys Acta* 164(2):278-284, 1968.

The isolated perfused dog liver was used to study the effects of bile acids on the excretion of biliary phospholipids and the ^{32}P-labeling of the individual plasma, liver and biliary phospholipids. Although high amounts ^{32}P-phospholipids were found in the liver, none was excreted in the bile unless taurocholate was infused. Biliary phospholipid excretion continued to increase throughout the taurocholate infusion period. Although the liver was the source of the biliary phospholipids, the specific activity of the biliary phospholipids greatly exceeded that of the liver phospholipids during taurocholate infusion, suggesting either that a specific phospholipid is secreted into the bile by the liver or that the biliary phospholipids are derived from a specific pool in the liver. There was a marked heterogeneity in the labeling pattern of the plasma, liver and biliary phospholipids. The bile ^{32}P-phosphatidyl-choline fraction accounted for over 95% of the total ^{32}P-phospholipids. Marked differences between the composition of the liver and the plasma ^{32}P-phospholipids were noted. The data support the view that the stimulatory effect of bile acids on biliary phospholipid excretion is related to the formation of a biliary micellar complex, and that highly selective mechanisms are involved in the formation of plasma and biliary phospholipids.

5805 **BILE ACIDS AND LIPID METABOLISM. IV. INFLUENCE OF BILE ACIDS ON BILIARY AND LIVER ORGANELLE PHOSPHOLIPIDS AND CHOLESTEROL.** *(E.)* Swell, L. (VA Hosp., Richmond, Va.), C. Entenman, G. F. Leong and R. J. Holloway. *Amer J Physiol* 215(6):1390-1396, 1968.

The excretion of bile acids-^{14}C, phospholipids-^{32}P, and cholesterol-^{14}C (mass and radioactivity) in the bile from the isolated perfused rat liver was determined with and without infusion of sodium taurocholate. Although liver cholesterol and phospholipids were labeled before the infusion of taurocholate, there was very little excretion of bile acids and lipids in the bile without taurocholate. Taurocholate infusion for 1-2 hr markedly increased the biliary excretion of total and labeled phospholipids and cholesterol. The phosphatidyl choline fraction constituted 94-97% of the ^{32}P activity in the bile, and the specific activity of this fraction was higher in the bile than in the liver mitochondria and microsomes. There were also marked differences in the ^{32}P labeling pattern of the liver organelles and plasma phospholipids. The results suggest that bile acids in the enterohepatic circulation regulate the excretion of biliary lipids. This may be related to the formation of a specific macromolecular complex in the liver and its subsequent excretion in the bile.

5806 **PROTEIN AND RNA SYNTHESIS IN RABBIT LIVER FOLLOWING HEMORRHAGE.** *(Rus.)* Barmina, Ie. F. (Sverdlovsk Med. Inst., USSR). *Biull Eksp Biol Med* 66(10):23-25, 1968.

As estimated by the incorporation of ^{35}S-methionine and ^{32}P, resp., the synthesis of plasma and liver proteins and liver cytoplasmic and nuclear RNA was uniformly increased by either acute or chronic hemorrhage in rabbits. After chronic blood loss (10 ml/kg every other day x 5), the rates of plasma and liver protein synthesis were 224% and 174% of normal, resp. After acute hemorrhage (20 ml/kg), increased protein synthesis was noted as early as 6 hr later and reached a maximum in 12 hr (to 302% of the original rate for the plasma proteins and 182% for the liver proteins); the increase persisted for at least 48 hr. Incorporation of ^{32}P into RNA 12-24 hr after acute hemorrhage was increased by 40-41% for the S-RNA and 133-190% for the r-RNA of the liver cytoplasm, and by 141-169% for the R-RNA and 21-39% for the m-RNA of the liver cell nuclei. As a result, the ratio between these 4 RNA fractions, which is normally 4:1:8:22, shifted to 2:1:6:9 after 12 hr and to 2:1:8:13 after 24 hr.

5807 **CHANGES IN THE OPTICAL DENSITY OF THE MITOCHONDRIA OF THE LIVER AND HEART DURING NEUROGENIC DYSTROPHY IN RATS.** *(Rus.)* Zabrodin, O. N. (Inst. Exp. Med., Acad. Med. Sci. USSR, Leningrad) and L. S. Semenova. *Biull Eksp Biol Med* 66(10):26-28, 1968.

In experiments on male albino rats, electrical stimulation for 15-60 min via electrodes on the forepaws (10-millisecond pulses at 5 V and 50 cycles/sec) resulted in swelling of the liver mitochondria, an increased rate of swelling in KCl, and a disruption of their contractile ability in response to ATP and $MgCl_2$. A similar increase in mitochondrial swelling, but accompanied by increased contractility in response to ATP and $MgCl_2$, was observed in the heart. In the case of the cardiac mitochondria, the same effects could also be produced by administration of epinephrine (0.5 ml of 1:1000 solution i.p.), indicating that the sympathetic nervous system plays a role in these biochemical changes. The alterations in mitochondrial morphology and behavior are considered to be an early sign of the dystrophic processes which can be produced in these organs by electrical stimulation.

5808 **EFFECT OF HYDROCORTISONE ON LIPID METABOLISM IN RAT LIVER.** *(Pol.)* Smietanska, Z. (2nd Clin. Int. Dis., Acad. Med. Warsaw, Poland) and Z. Skowronska. *Endokr Pol* 19(4):437-441, 1968.

When male Wistar rats were given 5 mg of hydrocortisone i.p., followed in 2 hr by octane-1-^{14}C and 4 hr later by decapitation, the liver cholesterol was only 1.92 mg/g compared to 2.26 mg/g in controls, the fatty acids were increased to 0.1189 mEq/g compared to 0.0797 mEq/g in controls, and the hepatic fatty acids had only 1030 counts/g compared to 1810 counts/g in controls. While hydrocortisone thus apparently inhibited fatty acid synthesis in the liver while promoting their accumulation, it had no significant effect on the serum cholesterol levels (71.5 mg% in treated rats and 81.0 mg% in controls) or the rate of hepatic cholesterol synthesis (266 counts/g in treated rats and 262 counts/g in controls).

5809 STUDIES ON PLASMA PROTEIN SYNTHESIS WITH A NEW LIVER PERFUSION APPARATUS. *(E.)* Ryoo, H. (U. California Sch. Med., San Francisco) and H. Tarver. *Proc Soc Exp Biol Med* 128(3):760-772, 1968.

A perfusion apparatus is described in which the liver floats in the perfusate, thus simulating conditions existing in the body. The apparatus consists of 2 identical perfusion sets so that the metabolism of 2 livers can be compared under identical conditions. In studies on rat livers, the perfused liver continued to produce plasma proteins for 10 hr and more. At the end of this time there was no vacuolation or necrotic change in any part of the liver, only slight atrophy in the hepatic cells. Replacement of the blood plasma of the perfusate by Ringer-Locke solution resulted in increased plasma protein production and a decrease in the synthesis of liver tissue protein. When polyvinylpyrrolidine was added to the Ringer-Locke solution, the production of plasma protein decreased. *In vitro* and *in vivo* studies showed globulin to be synthesized at a higher rate than albumin. Gamma-globulin showed a faster turnover rate than albumin and alpha-globulin showed a still faster rate. Some of the implications of these findings for the maintenance of a normal albumin/globulin ratio and the mechanism of the decrease in this ratio in diseases such as cirrhosis are discussed.

5810 EFFECTS OF A CORTICOID ON LIPID METABOLISM AND ON UREA FORMATION IN ISOLATED RAT LIVER. *(E.)* Wu, C. H. (Res. Found., Washington Hosp. Ctr., Washington, D. C.), A. Lemberg, J. A. Daunas, B. Brodoff, E. C. Sodero, R. Levine and J. C. Penhos. *Metabolism* 17(9):751-763, 1968.

The effects of triamcinolone acetonide 21-phosphate (666 μg added at 0, 30 and 60 min of perfusion) on lipid metabolism and urea formation in isolated rat livers perfused with 50 ml blood from fasted rats were studied with and without the addition of hyperlipemic serum in livers from fasted and fed rats. Perfusate levels of glucose, nonesterified fatty acids, triglycerides, cholesterol, ketone bodies, and urea and the liver content of glycogen, triglycerides, and cholesterol were determined after perfusion for 0, 30, 60, and 90 min and compared with those of control livers perfused with 50 ml blood only. Although there was no demonstrable increase in liver glycogen, triamcinolone produced an increase in plasma nonesterified fatty acids and a significant reduction in glucose output with or without an added lipid substrate (hyperlipemic serum). In experiments without hyperlipemic serum, triamcinolone produced lower levels of triglycerides and cholesterol in both the perfusate and the liver, suggestive of reduced net hepatic output. When hyperlipemic serum was employed, triamcinolone retarded the decline of the perfusate levels and the liver content, suggestive of reduced net uptake. Triamcinolone alone had no effect on the formation of ketone bodies, but when hyperlipemic serum was added to the perfusate, the formation of ketone bodies increased. Triamcinolone also did not modify the production of urea, with or without the addition of a lipid substrate. These results indicate that glucocorticoids have no effect on liver protein catabolism *in vitro*, in contrast to *in vivo* studies. It seems, however, that they are directly involved in the transport of lipids in and out of liver, and that the ketogenic and gluconeogenic effects depend mainly on their extrahepatic mobilizing and catabolic effects.

5811 THE EFFECT OF FASTING ON GLUCOSE UTILIZATION IN THE ISOLATED PERFUSED RAT LIVER. *(E.)* McCraw, E. F. (Indiana U. Sch. Med., Indianapolis). *Metabolism* 17(9):833-837, 1968.

The effect of fasting on glucose utilization was studied in the isolated livers of fed and fasting Holtzman rats (250-340 g) perfused with varying concentrations of glucose-U-^{14}C (75, 300, and 600 mg glucose/100 ml medium). Fasted animals were given only water *ad lib.* for 1 or 4 days. At an initial glucose concentration of 75 mg%, glucose utilization fell from an initial rate of 60 ± 10 to 24 ± 10 μmoles/100 g/90 min after 1 day of fasting and 23 ± 10 μmoles/100 g/90 min after 4 days. At an initial glucose concentration of 300 mg%, the glucose utilization fell from 170 ± 10 μmoles/100 g/90 min in fed rats to 84 ± 14 μmoles/100 g/90 min after 1 day of fasting and 64 ± 12 μmoles/100 g/90 min after 4 days. At an initial glucose concentration of 600 mg%, glucose utilization fell from 250 ± 24 μmoles/100 g/90 min to 142 ± 32 μmoles/90 min after 1 day and 160 ± 34 μmoles/100 g/90 min after 4 days of fasting. Glucose utilization was thus proportional to the level of glucose in the medium under all conditions, and was not depressed by fasting beyond the first day. The results suggest that hepatic glucose phosphorylation during starvation in the presence of elevated blood sugar levels may be mediated by the phosphotransferase reaction rather than by hexokinase or glucokinase, which are known to be depressed by fasting.

5812 COMPARATIVE STUDY OF METABOLIC DISTURBANCES PRODUCED IN THE LIVER BY ADMINISTRATION OF ETHANOL AND OTHER COMPOUNDS. III. SIMULTANEOUS ADMINISTRATION OF FRUCTOSE AND ETHANOL. *(Fr.)* Baron, P. (Bichat Hosp., Paris, France), G. Griffaton and R. Lowy. *Enzymol Biol Clin (Basel)* 9(5):377-384, 1968.

Fasted male Wistar rats were given 3 g/kg ethanol and 5.2 g/kg fructose i.p. and killed at different times after injection. Significant increases in lactate, α-glycerophosphate, and malate concentrations and significant decreases in β-hydroxybutyrate, acetoacetate, and NADPH were seen in the liver. Oxaloacetate, NAD, and NADH concentrations did not change appreciably. The increase in dihydroxyacetone phosphate was transitory and that of pyruvate was delayed. A comparison of these results with those obtained previously with ethanol alone and fructose alone suggests that ethanol enhances production of α-glycerophosphate from fructose with decreases in lactate and pyruvate production.

5813 THE EFFECT OF ETHIONINE ON LIPID METABOLISM IN MOUSE LIVER. *(Jap.)* Ochiai, N. (Sapporo Med. Coll., Japan). *Sapporo Med J* 33(3):163-174, 1968.

The effects of i.p. ethionine (1 mg/g) on the incorporation of ^{32}P-orthophosphate and [methyl-^{14}C]-methionine into hepatic phospholipids and proteins were studied in fasted female mice weighing 15-35 g. Although the neutral lipid content of the liver was approximately doubled 6 and 24 hr after administration of ethionine (to 38.9 and 116.4 mg/g, resp., compared to 17.5 and 53.9 mg/g in controls merely fasted for 22 and 40 hr), there was no significant change in phospholipid content. There was also no effect on the incorporation of ^{32}P into total phospholipids and lecithin, but its incorporation into cephalin was decreased by about 70%. The incorporation of radioactive methionine into hepatic proteins and phospholipids was decreased by 70% and 40%, resp., 6 hr after administration of ethionine labeled methionine given 1 hr before sacrifice). Chemical degradation studies showed that the ^{14}C content of the lipids was limited to the choline moiety of lecithin.

5814 **BILIARY EXCRETION OF ANTIBIOTICS IN MAN.** *(E.)* Acocella, G. (Maggiore Hosp., Milan, taly), R. Mattiussi, F. B. Nicolis, R. Pallanza and L. T. Tenconi. *Gut* 9(5):536-545, 1968.

The biliary excretion of 9 antibiotics and the effect of antibiotic therapy on bile volume and the biliary excretion of bili- ubin and cholesterol were investigated in 39 adult female patients with T-tubes in the biliary tract following surgery; in ddition, the concentration of antibiotics in the gallbladder wall was determined in 18 patients subjected to cholecystectomy. Cephaloridine, novobiocin, penicillin G, rifamide and tetracycline were given i.m.; rifamycin SV and rolitetracycline were given i.v.; ampicillin, erythromycin, novobiocin and tetracycline were administered orally, all at the usual clinical dosage. The esults of agar plate assays demonstrated that i.m. administra- ion is generally followed by more constant concentrations in he bile. Oral administration is likely to produce very irregular evels; furthermore, the appearance of antibacterial activity in he bile can be delayed and the probability of the antibiotic not being excreted in the bile seems to be higher. None of the ntibiotics had any effect on the volume of bile secreted (14.0-16.4 ml/hr). The concentration of bilirubin in the bile ranged between 115.8 and 127.0 mg%, and was decreased only follow- ng the administration of rifamycin SV and rifamide. Decreases n cholesterol (normally 61.8-70.5 mg%) were observed follow- ng administration of rifamycin SV, ampicillin and rifamide. Of he antibiotics tested, rifamycin and rifamide showed the highest ffinity for biliary excretion, 25-34% of the administered dose (i.v. and i.m., resp.) being recovered in the bile compared to 0.03-0.8% for the other drugs; only these 2 antibiotics and tet- racycline produced bile levels higher than the minimal inhibitory concentrations for common pathogens.

5815 **THYROID FUNCTION AND THE EFFECT OF CHOLESTEROL ON THE VITAMIN A AND B_1 CONTENT OF THE LIVER.** *(Pol.)* Haluszka, J. (Acad. Med., Krakow, Poland). *Endokr Pol* 19(5):545-548, 1968.

As previously reported, the feeding of a high-cholesterol diet (250 mg cholesterol/day) to male rats for periods of 90 days produced an increase in hepatic vitamin A and a decrease in hepatic vitamin B_1 (to 416 U/g and 47.6 µg%, resp., compared to 239.2 U/g and 74.6 µg% in controls). Administration of thyroxine (0.5 mg/day x 7) or thyrotropic hormone (5-Heyl-Laqueur U/day x 6) increased the vitamin A content of the iver still more (to 796.8 and 764.2 U/g, resp., in rats on a high-cholesterol diet and to 1172 and 571 U/g, resp., in rats on a normal diet), but abolished the significant difference between the two dietary groups. Thyroxine and thyrotropic hormone had little effect on hepatic vitamin B_1 levels in cholesterol-fed animals, but decreased these levels in controls (to 39.2 and 56.0 µg%, resp.). Thyroidectomy, on the other hand, increased the hepatic vitamin B_1 levels in cholesterol-fed animals and decreased them in controls (from 47.6 to 64.0 µg% and from 74.6 to 64.7 µg%, resp.). However, when thyroxine was administered to thyroidectomized rats, the hepatic vitamin B_1 levels decreased sharply regardless of the diet (to 16.8-19.8 µg%). These results indicate that the effect of dietary cholesterol on hepatic vitamin A and B_1 levels depends on the functional state of the thyroid.

5816 **CORRELATION OF RNA AND DNA SYNTHESIS DURING LIVER CELL PROLIFERATION AFTER PARTIAL HEPATECTOMY.** *(Ger.)* Rabes, H. (Inst. Path., U. Munich, Germany) and H. Brändle. *Virchow Arch Abt B Zellpath* 1(4):317-322, 1968.

Hypophysectomies were performed on male Sprague-Dawley rats and, after 28-30 days, 2/3 of their liver was removed. At various intervals after partial hepatectomy, animals were given cytidine-5-^3H and their livers were removed and examined by autoradiography. By 2 hr after partial hepatectomy an increase in labeling, particularly in the cell nucleoli, had occurred evenly overall parenchymal cells. This period of RNA synthesis is interpreted to be a specific response of liver cells to a specific proliferation stimulus. At 27 hr the labeled parenchymal cells were limited to the acinoperipheral parts of the liver. This occurred immediately before DNA synthesis began in these areas of the liver.

5817 **LIVER GROWTH ASSOCIATED WITH THE INDUCTION OF AMINOPYRINE DEMETHYLASE ACTIVITY AFTER PHENOBARBITAL TREATMENT IN ADULT MALE RATS.** *(E.)* Argyris, T. S. (Dept. Zool., Syracuse U., N. Y.). *J Pharmacol Exp Ther* 164(2):405-411, 1968.

Drug-induced changes in liver growth, total protein, aminopyrine methylase activity, RNA, DNA, nuclear count, and hepatocyte mitotic activity were investigated in young-adult male Holtzman rats given 100 mg/kg phenobarbital i.p. daily for 5 days. The rats were sacrificed 24 hr after the last drug injection. An increase in aminopyrine methylase activity was associated with a marked increase in liver weight and total protein. Total DNA and RNA content increased, but not the nuclear count. Hepatocyte mitotic activity was greatly increased, during the first 4 days of treatment. There was also a marked increase in the protein/nuclei, DNA/nuclei, and RNA/nuclei ratios in the phenobarbital-treated rats when compared to saline-treated controls, suggesting that cell enlargement had occurred.

5818 **THE EFFECT OF LOW PROTEIN DIETS ON THE TURNOVER RATES OF SERUM, LIVER AND MUSCLE PROTEINS IN THE RAT, MEASURED BY CONTINUOUS INFUSION OF L-[^{14}C]LYSINE.** *(E.)* Waterlow, J. C. (Trop. Metab. Res. Unit, U. West Indies, Jamaica) and J. M. L. Stephen. *Clin Sci* 35(2):287-305, 1968.

The amount and specific activity of free lysine and the specific activity of protein nitrogen in samples of blood, liver and muscle were measured in 75 white rats 6 hr after i.v. infusion of L-^{14}C-lysine. The specific activity of free lysine in the serum and liver reached a plateau within 3 hr; in muscle the plateau was not quite reached in 6 hr. Calculated serum and liver protein synthesis rates were higher than those obtained by other methods. These higher rates may be due to technical error or to liberation of significant amounts of unlabeled amino acid from protein breakdown. Male rats showed greater muscle protein synthesis that female rats, and young rats showed greater synthesis than old rats. Large reductions in muscle protein synthesis were observed during starvation or protein deprivation; changes in serum protein synthesis rates were smaller, while liver protein remained unchanged. Changes were greater in males. This lability of muscle protein synthesis may play an important part in the process of adaptation to low protein diets, and the mobilization of so-called reserves. The mechanism may involve a reduction in amino acid uptake by muscle protein. Other elements of this mechanism are unknown, but a possible endocrine control is mentioned.

5819 **THE ORIGIN OF PLASMA ARACHIDONIC ACID IN DOGS.** *(E.)* Caren, R. (Cedars-Sinai Med. Res. Inst., Los Angeles, Calif.) and L. Corbo. *Metabolism* 17(11):1043-1050, 1968.

Abdominal evisceration was performed in 6 fasted dogs, hepatectomy in 5, and sham operations in 3 to determine whether the liver is the sole source of plasma arachidonic acid. Venous blood collected preoperatively and hourly for 5-6 hr postoperatively was analyzed for blood glucose and plasma lipids. Unesterified fatty acids, phospholipids, triglycerides, and cholesterol esters were fractionated by column chromatography and the percentage of each determined by gas-liquid chromatography. Only the proportion of arachidonic acid in the plasma unesterified fatty acid fraction fell after evisceration and hepatectomy; no other fatty acids were depressed in this fraction, and no other lipid fraction showed any change, although palmitoleic acid was significantly increased in the unesterified fatty acid fraction. Sham operated dogs showed no change in any fatty acid fraction. Lipolysis of adipose tissue triglycerides apparently replenishes all fatty acids in the unesterified fatty acid fraction except arachidonic acid, which it lacks. It is concluded that since the liver was absent in the eviscerated and hepatectomized dogs, it must be the sole source of plasma arachidonic acid in fasting animals.

5820 **MECHANISM OF p-NITROBENZOATE REDUCTION IN LIVER: THE POSSIBLE ROLE OF CYTOCHROME P-450 IN LIVER MICROSOMES.** *(E.)* Gillette, J. R. (Natl. Heart Inst., NIH, Bethesda, Md.), J. J. Kamm and H. A. Sasame. *Molec Pharmacol* 4(6):541-548, 1968.

The nitroreductase, cytochrome P-450, and cytochrome c reductase activities of fractionated liver homogenates obtained from control, carbon tetrachloride-treated (2.5 ml/kg p.o.), and phenobarbital-treated (80 mg/kg i.p.) mice, rats, and rabbits were assayed in an incubation system containing NADP (1 μmole), p-nitrobenzoic acid (6 μmoles), $MgCl_2$ (15 μmoles), nicotinamide (40 μmoles), glucose-6-phosphate (30 μmoles), glucose-6-phosphatase (1 U), potassium phosphate buffer (1 ml of 0.2 M at pH 7.4), enzyme equivalent to 300 mg of liver, and sufficient water to bring the volume up to 3 ml; 2 μmoles of nitrobenzene, nitrosobenzene, or phenylhydroxylamine were used as substrate. Homogenates were subjected to ultracentrifugation to separate the nuclear (600 X g), mitochondrial (9000 X g), and microsomal (105,000 X g) fractions. The nitroreductase activity of the microsomal fraction was lower than that in the 9000 X g supernatant fraction, which was lower than that in the whole homogenate. The addition of boiled mitochondrial and nuclear preparations to the 9000 X g supernatant restored the activity to that of the control, whereas the addition of boiled soluble fraction restored the activity to that of the reconstituted 9000 X g supernatant. These findings suggest that nuclei, mitochondria, and soluble fractions of liver all contain unidentified nitroreductase activators. Boiled microsomal fractions enhanced nitroreductase activity in assay systems of unboiled liver microsomes. Carbon monoxide blocked the reduction of p-nitrobenzoate by liver microsomes from mouse, rat, and rabbit; it also blocked the increase in activity on addition of boiled preparations. Carbon monoxide blocked the formation of aniline from nitrosobenzene and phenylhydroxylamine about as well as it inhibited the reduction of nitrobenzene. Nitroreductase activity was enhanced by pretreatment with phenobarbital; this increase closely paralleled the increase in microsomal cytochrome P-450. Phenobarbital treatment did not markedly alter the effectiveness of carbon monoxide in blocking the reduction of p-nitrobenzoate. The administration of carbon tetrachloride decreased nitroreductase, cytochrome P-450, and NADP-cytochrome c reductase activity. The data suggest that the reduction of p-nitrobenzoate by liver microsomes is mediated by cytochrome P-450. Carbon monoxide blocks nitroreduction and the degree of inhibition is proportional to the amount of cytochrome P-450 bound to carbon monoxide as a complex.

5821 **EFFECTS OF GLUCAGON ON CARBOHYDRATE SYNTHESIS AND ENZYME ACTIVITY IN RAT LIVER.** *(E.)* Eisenstein, A. B. (Washington U. Med. Svc., St. Louis, Mo.) and I. Strack. *Endocrinology* 83(6):1337-1348, 1968.

In vivo and liver perfusion studies were used to investigate the effects of glucagon on hepatic glucose production and the activities of enzymes involved in gluconeogenesis in fasted and fed normal and adrenalectomized male Holtzman rats (190-225 g). Phosphoenolpyruvate carboxykinase activity rose and liver glycogen fell within 3 hr after a single s.c. dose of 40 μg glucagon; 5 μg or 40 μg glucagon twice daily for 2 days reduced liver phosphoenolpyruvate carboxykinase levels and increased liver glycogen. Dexamethasone (0.5 mg/day for 2 days) increased the activities of glutamic pyruvic transaminase, glutamic oxaloacetic transaminase and phosphoenolpyruvate carboxykinase and induced a rise in liver glycogen; pyruvate carboxylase activity did not increase. Glutamic pyruvic transaminase activity decreased markedly and glucose-6-phosphatase decreased slightly in fed adrenalectomized rats given dexamethasone. Glutamic oxaloacetic transaminase, phosphoenolpyruvate carboxykinase, glutamic pyruvic transaminase and glucose-6-phosphatase activities remained well below normal in fasted adrenalectomized rats; liver glycogen was almost completely depleted. Glucagon stimulated gluconeogenesis from pyruvate in the normal perfused liver, but was ineffective in altering the activities of enzymes which catalyze regulatory reactions in carbohydrate synthesis. Gluconeogenesis was not enhanced in glucagon-perfused livers of adrenalectomized rats. The stimulatory effect of glucagon was restored by the addition of dexamethasone to the medium. Although gluconeogenesis increased in adrenalectomized rat livers perfused with dexamethasone and glucagon, enzyme activity remained unchanged. These findings demonstrate that the effect of glucagon on glucose production is controlled by a mechanism not involving enzyme activation or synthesis.

5822 **COMPETITION BETWEEN BILIRUBIN AND SODIUM OLEATE, BROMSULFONPHTHALEIN**

D BILE SALTS FOR BONDING WITH ALBUMIN AS
ASURED BY GEL FILTRATION ON SEPHADEX G25.
.) Kučerová, L. (Fac. Gen. Med., Charles U., Prague,
choslovakia) and V. Hoenig. *Sborn Lek* 70(10):307-313,
58.

e effect of BSP, sodium oleate, glycocholate and dehydro-
late on the binding of bilirubin to serum albumin was stud-
by gel filtration techniques in solutions of crystalline bili-
in in lyophilized human serum albumin and in sera from 4
lts with obstructive or intrahepatic jaundice and 5 children
h erythroblastosis. All of the substances studied displaced
rubin from its bond with albumin when they were added
excess, but the amount displaced depended not only on the
led agent but also on the type of hyperbilirubinemia. Thus,
proportion of bilirubin bound to serum albumin was de-
ased to 13.6% and 60.9% by addition of sodium oleate
56 μEq/ml) to sera from patients with hemolytic and ob-
uctive jaundice, resp., and to 58.1% and 79.5% by addition
BSP (1.06 mg/ml) to these same sera (100% of the bilirubin
s bound to albumin in control sera). Sodium glycocholate
l dehydrocholate (1.33 mg/ml) produced only a small de-
ase in the proportion of bilirubin bound to albumin (to 83-
%) and there was no difference in behavior between conjugated
l nonconjugated bilirubin (i.e., between sera from different
es of jaundice). Intermediate results were obtained with
ificial solutions of bilirubin. The stability of the bond be-
een bilirubin and serum albumin, which affects the transport
bilirubin in the bloodstream, seems to be affected by the
emical state of the bilirubin as well as by the presence of
ids and bile salts.

23 COMPARATIVE STUDY OF METABOLIC
DISTURBANCES PRODUCED IN THE RAT
VER BY ADMINISTRATION OF ETHANOL AND OTHER
MPOUNDS. II. INTRAPERITONEAL INJECTION OF
UCTOSE. *(Fr.)* Baron, P. (Bichat Hosp., Paris, France),
Griffaton and R. Lowy. *Enzym Biol Clin (Basel)* 9(5):369-
6, 1968.

le Wistar rats, weighing 120-130 g, were fasted for 18 hr,
en 5.2 g/kg fructose i.p., and killed 15, 60, and 90 min after
ection. Liver tissue was immersed immediately in liquid
rogen and metabolites, with the exception of acetoacetate,
re determined enzymatically. Studies were run at two dif-
ent times of the year to rule out the possibility of seasonal
riations. Injection of fructose produced significant increases
the concentrations of lactate, pyruvate, α-glycerophosphate,
ydroxyacetone phosphate, and NAD and significant de-
eases in β-hydroxybutyrate, acetoacetate, and NADH. The
dings indicate that the Embden-Meyerhof pathway and the
ebs cycle are speeded up by fructose. Acetyl coenzyme A
utilized in such a way that the energy liberated goes into
nthesis of fatty acids rather than cholesterol and ketone
dies, and α-glycerophosphate then esterifies the fatty acids.
ese effects could be explained by a mechanism involving
rate as an intermediate.

24 INFLUENCE OF INDUCTION OF HEPATIC
MICROSOMAL ENZYMES BY PHENOBARBITAL
N TOXICITY OF ORGANIC PHOSPHATE INSECTICIDES.
(E.) DuBois, K. P. (Toxicity Lab., U. Chicago, Ill.) and F. K. Kinoshita. *Proc Soc Exp Biol Med* 129(3):699-702, 1968.

5825 A STUDY OF THE COMPARATIVE BIOCHEMISTRY OF THE GLUTAMIC-OXALOACETIC TRANSAMINASE ISOZYMES. *(Jap.)* Yana, K. (Jap. U. Med. Sci. Tokyo, Japan). *Nippon Ika Daig Z* 35(3):210-220, 1968.

5826 STUDIES ON PERFUSION OF THE LIVER AND HEART. PART 10. THE EFFECTS OF EPINEPHRINE, NOREPINEPHRINE AND ATP ON CARBOHYDRATE METABOLISM IN THE LIVER. *(Jap.)* Aida, H. (Japan U. Sch. Med., Tokyo). *Nichidai Ig Z* 27(9):1035-1059, 1968.

5827 METABOLIC BREAKDOWN PRODUCTS OF EXOGENOUS CHEMICAL AGENTS IN THE HEPATIC MICROSOMES. *(Fr.)* Truhaut, R. (Fac. Pharm., Paris, France), C. Bohuon and A. Amar-Costesec. *Ann Biol Clin (Paris)* 26(10/12):1261-1265, 1968.

5828 A PHOSPHATE ACTIVATED GLUTAMINASE IN RAT LIVER DIFFERENT FROM THAT IN KIDNEY AND OTHER TISSUES. *(E.)* Horowitz, M. L. (Harvard Med. Sch., Boston, Mass.) and W. E. Knox. *Enzym Biol Clin (Basel)* 9(4):241-255, 1968.

5829 CHANGES IN EXCRETORY HEPATIC FUNCTION IN DOGS FOLLOWING TRANSFUSION OF HETERO- AND ISOINCOMPATIBLE BLOOD. *(Rus.)* Garfunkel', M. L. (Cent Inst. Hematol. Blood Transf., Moscow, USSR) and R. V. Nedoshivina. *Pat Fiziol Eksp Ter* 12(4): 22-26, 1968.

5830 THE EFFECT OF FAST ALLERGIC REACTIONS ON THE RATE OF BIOLOGICAL INACTIVATION OF CORTISOL IN THE DOG LIVER. *(Rus.)* Pytskii, V. I. (Sci. Res. Lab. Allergol., Acad. Med. Sci. USSR,) and Iu. L. Gulyi. *Pat Fiziol Eksp Ter* 12(4):12-17, 1968.

5831 CHANGES IN SERUM GLYCOPROTEIN LEVELS IN HEPATECTOMIZED RATS. *(Hun.)* Piukovich, I. (Szeged U. Med. Sci., Hungary), M. Tenyi, G. Thury and J. Morvay. *Kiserl Orvostud* 20(4):432-435, 1968.

5832 CHANGES IN SERUM GLYCOPROTEIN LEVELS IN HEPATECTOMIZED RATS TREATED WITH PAPAIN. *(Hun.)* Piukovich, I. (Szeged U. Med. Sci., Hungary), M. Tényi, G. Thury and J. Morvay. *Kiserl Orvostud* 20(4): 436-441, 1968.

5833 THE SYNTHESIS OF TISSUE PROTEINS IN HEPATECTOMIZED RABBITS. *(Rus.)* Korotkina, R. N. (A. V. Vishnevskii Inst. Surg., Acad. Med. Sci. USSR, Moscow), V. I. Nikulin and A. S. Konikova. *Vop Med Khim* 14(5):527-533, 1968.

5834 RESPONSES OF LIVERS OF MICE TO TYPE OF DIETARY FAT. *(E.)* Wicks, M. S. (U. Mississippi, Sch. Med., Jackson) and C. R. Ball. *Anat Rec* 162(2):233-239, 1968.

See also: 5717, 5765, 6449, 6519, 6535, 6536, 6537, 6539, 6540, 6543, 6549, 6550, 6557, 6623

5835 **EVALUATION OF GASTRIC LAVAGE CYTOLOGY UNDER DIRECT VISION BY THE FIBERGASTROSCOPE EMPLOYING HANKS' SOLUTION AS A WASHING SOLUTION.** *(E.)* Kasugai, T. (Aichi Cancer Ctr. Hosp., Nagoya, Japan). *Acta Cytol* 12(5):345-351, 1968.

The diagnostic accuracy of gastric cytological examination has been increased markedly by the use of the lavage method under direct vision with a fibergastroscope. This method yielded a positive diagnosis in 363 of 375 proven gastric cancers (96.8%), including 62 of 64 cases of known early cancer (96.9%), with only 5 false-positives among 281 controls. This represents a diagnostic accuracy of 97.3%, compared to 80.9% for routine gastric lavage (110 of 136 known cases). When the 2 methods were compared in the same series of 84 cases (69 of advanced cancer and 15 of early cancer), gastric lavage under direct vision yielded 66 and 13 positive diagnoses, resp., compared to 61 and 6 for routine gastric lavage; the diagnostic accuracy of the 2 techniques was therefore 94% and 79.8%, resp. There was no significant relation between the diagnostic accuracy and the macroscopic classification, location, or extent of the lesion. When the lesion was definitely recognized and the washing solution was aimed precisely at the lesion, then positive results were obtained in all cases, both advanced and early. Clinical and experimental studies demonstrated that Hank's solution is the best washing fluid, producing less degeneration and better staining of the cells. Further studies should be undertaken, however, to improve the instrumentation, washing solutions, and techniques for the staining and microscopic examination of cells.

5836 **LIVER FUNCTION TESTS AFTER ADMINISTRATION OF NICOTINIC ACID. I. A CLINICAL STUDY.** *(Ger.)* Finck, C. (Munic. Hosp., Jena, Germany) and D. Jorke. *Acta Hepatosplen* 15(5):315-321, 1968.

Serial determinations of unconjugated (indirect) serum bilirubin were carried out following the slow i.v. injection of nicotinic acid (3-4 ml of a 1% solution) in 178 patients with various hepatic diseases (including 100 diagnosed by biopsy) and 84 healthy controls. The half-life of the transitory increase in endogenous serum bilirubin following a nicotinic acid load was found to be pathological (longer than 2 hr) in 10 of 84 apparently healthy controls, 28 of 40 patients with sequellae of hepatitis, 31 of 39 with chronic hepatitis, 9 of 12 with cirrhosis, 3 of 32 with fatty liver, 13 of 21 with ascending hepatitis, 16 of 17 with secondary hepatic diseases, and 16 of 17 with hyperbilirubinemia. The response in pure fatty liver was therefore essentially the same as in normal controls (half-life ranging from less than 0.5 to about 5.0 hr). There was no correlation between the results of this test and changes in plasma proteins, serum lability or the galactose test; even the BSP test was normal in 44 of 67 cases with a pathological half-life (due mostly to diseases not diagnosable by the BSP) and abnormal in 9 of 60 cases with a normal half-life. The nicotinic acid loading test is concluded to be of particular value in the diagnosis of hyperbilirubinemia of varying etiology.

5837 **NEW POSSIBILITIES IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE INTRA- AND EXTRAHEPATIC CHOLESTASIS.** *(Ger.)* Lévai, F. (Comm. Hosp., Szentes, Hungary). *Zbl Chir* 93(42):1465-1473, 1968.

The ^{131}I-labeled Rose Bengal test, before and after prednisolone (30 mg/day x 4), was given to 20 patients with acute intra- (10) or extrahepatic (10) cholestasis in an attempt to improve upon the accuracy of conventional liver function tests in the differential diagnosis of viral hepatitis and biliary tract neoplasms. The serum bilirubin (9.8-19.4 mg%), thymol turbidity (0.48-5.64 U), serum cholesterol (316-680 mg%), alkaline phosphatase (42.2-81.0 K-A U) and plasma protein picture were essentially the same in the two groups. The SGOT, SGPT and antistreptolysin titer were much higher in the patients with intrahepatic obstruction (124-283, 270-467 and 1600-3200 U, resp.) than in those with extrahepatic obstruction (16-75, 37-84 and 200-560 U, resp.), but it is pointed out that these parameters are often elevated in such patients later in the course of the disease due to increasing reactive cholangitis. The slow uptake of radioactive Rose Bengal by the liver cells and the persistently high blood level after i.v. injection were characteristic of patients with intrahepatic jaundice. Particularly diagnostic, however, was the response to prednisolone, which produced marked clinical improvement accompanied by a striking increase in the rate of Rose Bengal uptake by the liver in patients with intrahepatic obstruction. No such lasting improvement was noted in patients with extrahepatic obstruction. These differences confirm the validity of ^{131}I-Rose Bengal scintillation scanning in the differential diagnosis of jaundice.

5838 **RESULTS IN THE DIAGNOSIS OF ENTEROPATHOGENIC *E. COLI* BY MEANS OF IMMUNOFLUORESCENCE MICROSCOPY AND CULTURAL PROCEDURES.** *(Ger.)* Schneider, H. (Munic. Hosp., Mannheim, Germany). *Kinderaerztl Prax* 36(10):471-474, 1968.

The 1500 stool specimens obtained from infants admitted for dyspepsia, infants developing enteritis while in the hospital, and children coming into contact with infants who developed diarrhea during the course of 1 yr at the Mannheim Pediatric Clinic were subjected to immunofluorescence microscopy using fluorescent antibodies against 11 serotypes of pathogenic *Escherichia coli;* the presence of 4 other serotypes was investigated by the indirect method using fluorescent antibody prepared against immunoglobulin. Positive results were obtained in 24% of the samples; pathogenic *E. coli* detected included 101 strains of 0 128:B 12, 79 strains of 0 124:B 17, 54 strains of 0 119:B 14, 47 strains of 0 126:B 16, 41 strains of 0 26:B 30 strains of 0 125:B 15, and 1-11 strains of 6 other serotypes. Two types were present simultaneously in 30 children, and 3 types were found in each of two children. Positive results were more common in summer than in winter. In 844 cases, serological and microbiological studies were also performed. Although the immunofluorescent and cultural techniques agreed in 80% of these cases, fluorescence microscopy failed to reveal the organism in 28 cases (including 5 in which there was marke nonspecific fluorescence) and cultural techniques failed in 138 cases. Immunofluorescence microscopy is a rapid and extremel useful technique for the detection of pathogenic *E. coli,* but it should be supplemented by cultural techniques since only the latter permit the determination of antibiotic sensitivity.

5839 **THE VALUE OF GASTRIC SCINTILLATION SCANNING WITH TECHNETIUM 99m AS A**

)UTINE DIAGNOSTIC PROCEDURE. *(Ger.)* Fridrich, R. ıst. Radiol., U. Basel, Switzerland) and G. Engelhart. *Schweiz ed Wschr* 98(45):1789-1793, 1968.

ıe techniques and advantages of gastric scintillation scanning th ^{99m}Tc are outlined on the basis of experience with over 0 cases. Illustrative scans are presented for patients with stroenterostomy, duodenal ulcer, stomach cancer and atrophic stritis. The technique involves scanning over the lower thorax d upper abdomen after the i.v. injection of 2 mC of ^{99m}Tc the form of the pertechnetate (TcO_4^-), thyroid uptake being ocked by Lugol's solution. It is particularly valuable in dis- guishing neoplastic from inflammatory conditions in cases which the results of gastroscopy and X-ray are unclear. This because the isotope is taken up normally by inflammatory sue, while the scan of a neoplastic area is "cold". Control dies in 16 patients showed that the excretion of ^{99m}Tc is oportional to the volume of gastric secretion and begins within min after i.v. injection. The isotope is taken up selectively the gastric mucosa (fundus, corpus and antrum), distinguish- these areas clearly from the cardia and small intestine.

40 AN ANALYSIS OF THE INSULIN TEST AFTER VAGOTOMY USING SINGLE AND MULTIPLE ITERIA.

(E.) Gillespie, G. (Western Infirm., Glasgow, otland), I. E. Gillespie and A. W. Kay. *Gut* 9(4):470-474, 58.

results of post-vagotomy insulin tests were compared with se of pre- and postoperative augmented histamine tests in duodenal ulcer patients. The 5 criteria of a positive insulin oonse were an increase in gastric secretory volume, a basal l output greater than 2 mEq/hr, an increase in acid concen- ion by more than 20 mEq/L within 2 hr after insulin in- ion or an increase of more than 10 mEq/L if basal secretion anacid (i.e., the criteria of Hollander), the same changes urring within 1 hr after insulin, and total acid output greater n 2 mEq (or free acid output greater than 1.5 mEq) in any r period. There was no significant difference in the mean centage reduction in augmented histamine response between ılin-negative and insulin-positive patients when considering y increases in gastric secretory volume or basal acid output. mean percentage reductions after vagotomy in those judged ılin-positive by the other 3 criteria were significantly smaller n the mean percentage reductions in the insulin-negative ups. In 35 of 135 patients evaluated, the insulin tests were ative by all criteria; 41 patients had 2 positive criteria, 24 3 positive, 9 had 4 positive, and 6 had all 5 positive. The ın percentage reduction in augmented histamine response de- ısed from about 70% in insulin-negative cases (or those with positive criteria) to 57% in those with 3 positive criteria, in those with 4 positive criteria, and 45% in those who e positive to all 5 criteria. Two patients developed recurrent ers, one having 4 criteria of a positive insulin response and other satisfying all 5 criteria. These results support the gestion that consideration of multiple criteria of a positive ılin response would be more likely to detect those patients h incomplete vagal nerve section who are most at risk of eloping recurrent ulceration.

41 CORRELATION BETWEEN WEIGHT, NITROGEN, AND FAT CONTENT OF STOOL.

(E.) Pinter, K. (Tulane U. Sch. Med., New Orleans, La.) and A. McLean. *Amer J Clin Nutr* 21(11):1310-1313, 1968.

A total of 363 fecal samples, collected from a large group of patients with steatorrhea over periods of 3-4 days, were analyzed in order to study the relation between fat and nitrogen content and wet stool weight. The weight of the specimens varied from 41 to 998 g/day; the fat, from 5 to 95 g/day; and the nitrogen, from 1 to 7 g/day. Samples which exceeded 1000 g/day, or came from patients with watery diarrhea, regional enteritis or ulcerative colitis, were excluded from the statistical calculations because of the excessive water content. It was found that a correlation existed between the weight of the wet stool sample and its nitrogen and fat content. A nomogram was constructed which may be used to estimate the nitrogen and fat content from the gross weight of a 3-day stool sample.

5842 UNALTERED BROMSULPHALEIN RETENTION FOLLOWING CHOLECYSTOGRAPHY.

(E.) Johnson, R. A. (VA Hosp., Jackson, Miss.) and L. O. Mora. *Postgrad Med* 44(4):108-113, 1968.

The results of BSP tests performed before and after ingestion of gallbladder dyes (3-6 g of iopanic acid, iodoalphionic acid or sodium ipodate) in a series of 54 male patients (36-74 yr old), including 35 with apparently normal livers and 19 with confirmed liver disease but no jaundice, indicate that BSP retention is not significantly altered following cholecystography. The observations showed minor variations in BSP retention but, in view of other clinical and laboratory results, the data do not support the inference that currently used cholecystographic media significantly alter BSP retention. Thus, the BSP retention test can be done on the same day as cholecystography provided that the test is performed properly. However, the results must be interpreted subjectively in conjunction with other liver function tests and clinical findings.

5843 AN IMPROVED PROCEDURE FOR THE PREPARATION OF INDIUM LABELED LUNG AND LIVER SCANNING COMPOUNDS.

(E.) Bruno, F. P. (VA Hosp., Gainesville, Fla.), O. A. Sorsdahl and C. M. Williams. *Amer J Roentgen* 104(4):762-765, 1968.

Modifications to the method of Stern *et al.* *(Nucleonics* 24:65, 1966) for the preparation of ^{113m}In-colloid for use in scintillation scanning of the liver are described which have substantially decreased the preparation time of this reagent. ^{113}In of high specific activity, but without radionuclide impurities, is obtained by daily elution of a ^{113}Sn/^{113}In generator. Indium hydroxide bound to colloidal gelatin can then be prepared simply and quickly. The procedure differs from others previously described in the following respects: the use of presterilized pyrogen-free reagents to prepare the compounds in a closed reaction vessel; the substitution of ammonium hydroxide for sodium hydroxide for neutralization; and the use of a colorimetric pH indicator in the injectable material. Another advantage is the decreased handling time, resulting in lowered radiation exposure of the technical staff. This method produces macroparticles of extremely uniform size and the entire preparation procedures including elution and sterilization, takes only 30 min.

5844 NORMAL CELIAC AND HEPATIC ARTERIOGRAM.

(E.) Ruzicka, F. F. Jr. (St. Vincent's

Hosp., New York City) and P. Rossi. *New York J Med* 68(23): 3032-3034, 1968.

A scheme is presented for the classification of variations in the celiac axis and hepatic artery, based on the findings in 185 patients with adequate arteriographic visualization of the celiac, hepatic, and superior mesenteric arteries. For the sake of discussion, it was found convenient to set up 6 types of variations in the celiac trunk and 4 types of variations in the hepatic artery, with the classic type accounting for 93% and 72% of the cases, resp. Each type is illustrated and its frequency indicated in percentage terms (0.5-4% for the less common types of celiac trunk and 4-11% for the less common types of hepatic artery structure). Analysis of these types shows that superior mesenteric arteriography must be added to celiac arteriography to obtain complete visualization of the liver in about 15% of all patients. It is hoped that this classification scheme will provide a practical means of recognition of the anatomical variations that may be encountered during celiac and hepatic arteriography.

5845 **PERFORATION OF THE LARGE BOWEL DURING ROUTINE BARIUM ENEMA STUDIES.** *(E.)* Goldston, A. B. (1501 W. 10th, Amarillo, Tex.), L. Bast and S. Hands. *Texas Med* 64(11):52-56, 1968.

Detailed case reports are presented for 5 patients (4 men and 1 woman, 51-76 yr old) with diverticulitis of the sigmoid colon (2), advanced carcinoma of the colon (1), or colostomies resulting from surgery for intussusception or rectal cancer, in whom perforation of the large bowel occurred during barium enema. Immediate diagnosis was made in only 2 cases, and 2 resulted in death from peritonitis and septicemia (a 3rd patient recovered from the perforation but died shortly of cancer). The difficulty of removing the barium by irrigation and gauze sponges is stressed. Successful treatment is related to the time between rupture and surgery and the amount of barium in the abdominal cavity. Barium enemas should be done with particular care in patients with colostomies, cancer or diverticulitis, since the mortality rate is about 50%.

5846 **USE OF BARIUM SULFATE AS AN UNABSORBABLE FECAL MARKER.** *(E.)* Figueroa, W. G. (Dept. Med., U. California, Los Angeles), T. Jordan and S. H. Bassett. *Amer J Clin Nutr* 21(11):1239-1245, 1968.

The accurate measurement of fecal flow and correction for its variability is very important in metabolic balance studies. A method is described for the measurement of fecal flow using barium sulfate as an unabsorbable fecal marker. The technique requires only exact weighing of the material before administration. In tests on 5 patients, the total barium sulfate administered, 97.7-103% was recovered in the stools, indicating the completeness of the collections. Further, no untoward effects were noted in any of the 5 patients. Based on the results of calcium balance studies corrected by this technique, it is concluded that barium sulfate permits a more accurate assessment of the fecal flow than other marker substances.

5847 **THE EFFECT OF EMOTIONAL FACTORS ON GASTROINTESTINAL MOTILITY IN CHILDREN.** *(Fr.)* Kowalski, R. (Gabriel-Touvé Hosp., Bamako, Mali). *Ann Radiol* 11(7/8):545-550, 1968.

Based on the radiological findings in 10 children (6 months- 12 yr old) examined by barium swallow because of chronic constipation, abdominal pain or suspected appendicitis, it is concluded that emotional factors can interfere with gastrointestinal motor function, even in normal subjects. Simple positioning of the table, possibly aggravated by noise and darkness, produced abnormalities such as complete temporary cessation of small intestinal peristalsis, hypermotility, or hypertonia of the large intestine in children who showed normal gastrointestinal motility when they were calm. Illustrative findings are presented for 4 children in whom X-rays taken shortly after positioning of the apparatus revealed jejunal and ileal hypotonia, gastrointestinal hypertonia and spasm, regional hyper- and hypotonia of the small intestine, and colonic spasm, resp., although earlier and later X-rays were normal.

5848 **INTRAHEPATIC PRESSURE AND SPLEEN PULP PRESSURE: A SIMPLE METHOD FOR THE QUANTITATIVE DETERMINATION OF PORTAL PRESSURE DURING LAPAROSCOPY.** *(Ger.)* Sickinger, K. (Med. Clin. Polyclin., U. Göttingen, Germany), L. Kallmann and J. Emmrich. *Med Klin* 63(1):247-251, 1968.

Measurements of the intrahepatic and spleen pulp pressures during laparoscopy in 140 patients revealed that the intrahepatic pressure is often increased in cases of early cirrhosis, severe steatosis, or systemic diseases affecting the liver, before the development of obvious signs of portal hypertension. The average intrahepatic pressure varied from 11 ± 2 cm H_2O in normal subjects to 16-18 cm H_2O in moderate fatty liver, chronic hepatitis, periportal fibrosis or hepatic congestion, 24-25 cm H_2O in mild cirrhosis or severe fatty liver, and 31-45 cm H_2O in moderate-severe cirrhosis with obvious development of collaterals. Thrombosis of the splenic vein was indicated by a marked pressure drop between the splenic pulp and the liver, but this pressure difference was eliminated by the simultaneous increase in intrahepatic pressure in cases of coexisting congestive heart failure, periportal fibrosis, Budd-Chiari syndrome or cirrhosis. Measurement of the intrahepatic pressure is suggested as a simpler substitute for the measurement of the obstructed hepatic vein pressure in the diagnosis of hepatic diseases.

5849 **THE DIAGNOSIS OF PRIMARY LIVER CANCER BY THE DEMONSTRATION OF EMBRYOSPECIFIC ALPHA$_1$-GLOBULIN IN THE SERUM.** *(Fr.)* Sankalé, M. (Med. Clin., Sch. Med. Pharm., Dakar, Senegal), R. Masseyeff, R. Camain, C. Quenum, J. P. Ancelle and L. Leblanc. *Presse Med* 76(43):2047-2049, 1968.

Double immunodiffusion studies with rabbit antibodies on the sera from 118 patients with primary carcinoma of the liver in an advanced stage (confirmed histologically in 56 cases) revealed the presence of embryospecific α_1-globulins in 90 cases (76.2%), including 42 of the 56 histologically diagnosed cases and 48 of the 62 others. The 42 positive cases included one patient with cirrhosis in whom the tumor was discovered only at autopsy. There was no relation between the presence of embryospecific α_1-globulin and sex, age, clinical symptoms, liver function tests, prognosis or histological features of the cancer. Whereas the test has been positive in 80.7% of a total of 224 cases of primary hepatoma seen in recent years, it has been uniformly negative in all other adult patients examined (3230 healthy subjects, 222 with cirrhosis, 375 with hepatitis, amebiasis or hepatic congestion, 2836 with various illnesses, and 119 with various other malignancies), the single exception being 1 of 10 patients with testicular teratoblastoma. The test is even negative in patients with hepatic metastases from other cancers.

5850 **SPECIFICITY OF CERTAIN METHODS FOR THE DETERMINATIONS OF PANCREATIC LIPASE.** *(E.)* Barrowman, J. A. (Div. Physiol. Chem., U. Lund, Sweden) and B. Borgström. *Gastroenterology* 55(5): 601-607, 1968.

Gel filtration and ion exchange chromatography were used to separate the lipase (glycerol ester hydrolase) and the esterase (carboxylic ester hydrolase) found in rat pancreatic juice so that the specificity of these enzymes for a series of substrates proposed for lipase determination could be determined. Methods employing emulsified long-chain triglycerides as substrates were found to be the best and most specific for assay of pancreatic lipase, although different methods based on chromogenic and fluorogenic systems have been proposed and may offer prospects of more convenient and sensitive assays. In some assay systems employing the diesters of fluorescein, the substrates were not split by lipase under any conditions, while in other systems using β-naphthol and 4-methyl-umbelliferone esters some substrates were split by lipase, some by esterase, and some by both, depending on factors such as the chain length of the fatty acids and the presence or absence of bile salts in the system. Dispersions of the chromogenic β-naphthol laurate and oleate and p-nitrophenyl laurate were split principally by lipase; however, the addition of bile salt to the incubation systems, even at low concentrations, altered their specificity in favor of the esterase.

5851 **A NEW TWIN HOLE CAPSULE FOR PERORAL INTESTINAL BIOPSY IN CHILDREN.** *(E.)* Sebus, J. (Wilhelmina Children's Hosp., Utrecht, Netherlands), J. Fernandes and J. A. van der Bult. *Digestion* 1(4):193-199, 1968.

The 3 main problems which arise in performing peroral intestinal biopsy in children are failure to introduce the relatively large instrument, bleeding and perforation after biopsy has been taken, and failure to recover the instrument. In an attempt to solve some of these problems, a tiny instrument with two 2-mm holes was designed which operates by a suction mechanism, thus differing from the spring-operated Crosby capsule. The same negative pressure which draws in part of the jejunal mucosa through the small holes also drives the sharp-edged cylinder past the same holes. Consequently, the instrument can be reset while in position in the intestine. When this instrument was used in 47 children ranging in age from less than 1 yr to more than 6 yr, there were no complications and only 2 failures out of 52 attempts. Both failures were due to inability to pass the capsule beyond the pylorus. After changing the sedation procedure the last 35 biopsies were all successful. The tissue obained was always adequate for examination, although in the case of children with celiac disease the specimens were someimes rather small. The average time required for the jejunal biopsy was 1 hr, and sometimes the whole procedure took less han 45 min. The total fluoroscopy time seldom exceeded 30 ec.

5852 **STUDY OF GASTRIC ACID SECRETION BY MEANS OF "GASTROTEST."** *(Rum.)* Ciplea, A. (D. Danielopolu Inst. Norm. Path. Physiol., Bucharest, Romania) and A. Oană. *Fiziol Norm Pat* 14(4):317-324, 1968.

"Gastrotest", a dye-test for studying gastric acidity, involves the ingestion of 2 white tablets containing 0.2 g of caffeine sodium benzoate and 3 yellow tablets containing 0.05 g of 3-phenylazo-2,6-diaminopyridine. After fasting for 8 hr, patients take the white tablets with 50 ml water, urinate and 1 hr later, take the yellow tablets. Urine is collected 1.5 hr later and diluted with 200 ml water. A small portion is then diluted 1:1 with 25% HCl to give a pink color which is compared with the colors on a standard scale. Studies are run as quickly as possible so that bacteria which might be present in the patient's urine cannot produce changes in the pH. This test was run on: 1) 22 patients in whom gastric secretion had previously been studied after alcohol stimulation and who secreted free HCl, and 2) 14 patients in whom gastric secretion had previously been studied after alcohol (6 cases) or histamine (8 cases) stimulation and who secreted no HCl. The Gastrotest was positive in all 22 patients in the first group and negative in 11 of the 14 in the second group. The weak positive results obtained on 3 patients are attributed to medication in 1 case, erroneous results from intubation in another, and the stronger stimulation of gastric secretion by caffeine than by histamine in the third. None of the patients showed any signs of intolerance and there were no complaints of nausea or headaches.

5853 **A NEW METHOD OF STUDYING INTESTINAL MUCOSAL DYNAMICS.** *(E.)* McFarland, J. B. (Dept. Surg., U. Liverpool, England), A. Asplin and H. M. Hallaway. *Brit J Surg* 55(10):786-789, 1968.

A technique is proposed for DNA estimation as the basis for measurements of cell-loss from the intestine; this method for studying cell-turnover could be acceptable in human investigations and is applicable in some pathological situations. Results in preliminary trials with a 20-kg beagle dog demonstrated reasonable reproducibility in the amount of DNA washed out of a jejunal loop in a stated time period. Studies on 13 surgical patients also showed a good correlation in DNA recovery between different studies; in 3 cases in which surgery lasted 60 min, the second post-sample washes contained only about 10% of the DNA recovered for the sample period. It is concluded that the total DNA accumulation can be calculated from the sample plus 3 post-sample washes. When the total DNA accumulation in 30 cm of jejunum was plotted, 11 of the 13 points fell approximately within 2 standard deviations. Preliminary human data indicate accumulation of 105 μg DNA-P in 30 cm of jejunum in 1 hr, from which it can be calculated that the turnover time for the epithelium of the villi is approximately 55.3 hr, and that for the epithelium of the villi and crypts together is about 4.5 days.

5854 **LYMPHANGIOGRAPHY IN THE STUDY OF CHRONIC RECURRENT PANCREATITIS.** *(It.)* Prior, C. (Inst. Surg. Path., U. Genoa, Italy) and E. Cariati. *Rass Arch Chir* 5(3):77-86, 1967.

Lymphangiograms of 10 patients with chronic recurrent pancreatitis showed that the flow of contrast medium stopped at the L_1-L_2 level where it remained for several hr before moving further. In all 10 cases the thoracic duct was compressed by the hypertrophied pancreas; above the pancreatic region the duct had a normal diameter and was almost normal morphologically. Evidence was found for lymphovenous fistulas in 2 patients. In 1 patient with mild ascites the cisterna chyli was dilated due to compression of the thoracic duct which resulted in stasis and considerable hypertrophy of the lymph vessels and nodes below this region. In contrast to Sarles *et al.* *(Presse Med* 73(50):2885, 1965), who found hypertrophied lymph nodes in patients with pancreatitis, 3 of the patients in the present study had hypoplastic lymph nodes and spasms in the collectors of the ileolumbar region and the dorsum of the foot. Therefore, it is unlikely that the flow of contrast medium is blocked by enlarged lymph nodes. Instead it seems to be caused by compression of the lymphatics by the hypertrophied pancreas, fibrosis, adhesions of the peripancreatic capsule and, possibly, sympathicoganglionitis.

5855 PANCREATIC SCINTIGRAPHY: PRELIMINARY RESULTS WITH A NEW TECHNIQUE. *(Fr.)*

Blanquet, P. (St.-André Hosp., Bordeaux, France), C. Beck and J. Pigneux. *Bordeaux Med* 1(6):1111-1124, 1968.

Pancreatic scintigraphy was successfully performed on 56 of 58 fasting patients who had been given a 30-g protein meal 1 hr before i.v. injection of 200 μC of ^{75}Se-selenomethionine and 150 μC of ^{198}Au, followed by ingestion of 900 mg of glutamic acid. By connecting the scintillation counter to a computer with 4096 channels, the counts from ^{75}Se-selenomethionine, which is fixed in both the pancreas and liver, could be subtracted from those of ^{198}Au, which is fixed only in the liver. Normal images were obtained in 5 patients with normal pancreases. No false negative results were obtained in 4 cases of confirmed pancreatic cancer, but the scintigrams had different patterns in each case. Absence of fixation gives no clue to the etiology of the pancreatic disorder except in chronic pancreatitis in which a slow rate of fixation and low activities were characteristic. These were only seen in one very extensive cancer of the pancreas.

5856 THE EARLY DIAGNOSIS OF ACUTE PANCREATITIS WITH STRONTIUM 85: EXPERIMENTAL STUDIES. *(It.)*

Rossi, R. (Gen. Surg. Clin. U. Milan, Italy), V. De Marzo and O. Ferri. *Arch Ital Chir* 94(2):274-290, 1968.

No significant differences were found between the curves for ^{85}Sr activity in the blood of 2 normal dogs and 2 with acute pancreatitis induced by injection of olive oil into the excretory ducts of the pancreas. This may be due to mobilization of Ca and Sr from other organs into the blood to compensate for their deposition in the pancreas. A correlation was found between the pathological signs of pancreatitis and the pancreatic ^{85}Sr concentration in dogs in which pancreatitis had been induced by different methods. Those with olive-oil induced pancreatitis had pancreatic ^{85}Sr activities much higher than lung, skin, liver, spleen, heart, muscle, kidney, or bone activities. This difference was obvious as early as 40 min after injection of ^{85}Sr. Diffuse uniform pancreatic necrosis was found in these animals, all of which died within 12 hr. Dogs with pancreatitis induced by mineral oil had pancreatic ^{85}Sr concentrations higher than those in any other organs, but lower than those in the bones. These animals survived more than 36 hr, and pancreatic damage was less severe in these cases. Dogs with pancreatitis induced by bile and trypsin survived 33 hr. Pathological findings were similar to those described in man and ^{85}Sr concentrations were analogous to those in dogs with mineral-oil induced pancreatitis. It is suggested that this method might prove useful in the clinical diagnosis of acute pancreatitis if ^{47}Ca, which has a much shorter half-life, were substituted for ^{85}Sr.

5857 PANCREATIC GAMMAGRAPHY. *(Sp.)*

Escobar Prieto, A. (Natl. Inst. Nutrit., Mexico City, Mexico), I. Mariscal Ibarra and E. Tovar Zamora. *Rev Invest Clin* 20(2): 141-149, 1968.

Pancreatic gammagraphy was performed on 52 patients with various diseases and on 5 normal controls 5-10 min after i.v. injection of 200 μC of ^{75}Se-selenomethionine. Images were obtained in 48 of the 57 cases studied. Normal images were obtained in 16 of 20 subjects without clinical evidence of pancreatic disease, including the 5 controls. The other 4 subjects with false positive images included 1 with a liver abscess, 1 with an old mesenteric thrombosis, 1 with stomach cancer, and 1 with gallbladder carcinoma and hepatomegaly due to metastases. Abnormal images were obtained in 15 out of 16 cases of chronic recurrent pancreatitis. Exocrine and endocrine pancreatic insufficiency were present in 4 of 5 other patients with chronic recurrent pancreatitis for whom images could not be obtained. False normal images were found in 2 patients with pseudocysts of the pancreas and in 2 with diffuse hyperplasia of the islets of Langerhans. All 13 patients with pancreatic carcinoma and 3 who had undergone resection of the pancreas had abnormal images.

5858 INDUCED URINARY EXCRETION OF BSP: ITS DIAGNOSTIC SIGNIFICANCE, PRELIMINARY RESULTS, AND SOURCES OF ERROR. *(Fr.)*

Toulet, J. (International Hosp., U. Paris, France). *Arch Franc Mal Appar Dig* 57(10):821-836, 1968.

In this test, 5 mg/kg BSP were injected i.v. and 4 urine fractions were collected: from 0-2 hr, 2-4 hr, 4-10 hr, and 10-24 hr. When the quantity of BSP was determined in each fraction and graphs were plotted to show the total BSP excreted since the beginning of the test, 4 types of curves were obtained: 1) a regular increase in BSP excretion (with 3 different subtypes), found in obstructive jaundice, primary biliary cirrhosis, pancreatic cancer, cancers of the hepatic hila, and the initial stage of viral hepatitis; 2) an increase to a lower level than that found in type 1 curves, seen in patients with later stages of viral hepatitis; 3) very high initial values followed by increased excretion with a break around 10 hr, seen in patients with ascitic cirrhosis; and 4) a continuous low plateau, seen in moderately progressive cirrhosis, during the 2nd half of viral hepatitis and in convalescence cirrhotogenic hepatitis, subacute hepatitis, compensated cirrhosis and minor liver disorders. A graph is presented for correlating urinary BSP values with 7 diagnostic zones. Brief reports are presented for patients whose urinary BSP excretion aided in the diagnosis of cirrhosis, pancreatic cancer, viral hepatitis, and cholestatic hepatitis induced by an antibiotic containing hexacycline and an erythromycin salt. Results obtained in this test sometimes disagreed with blood values. Errors may result from associated disorders in kidney function.

5859 CHANGES IN PRE- AND POSTOPERATIVE TRANSAMINASE VALUES IN SOME HEPATOBILIARY DISEASES COMPARED WITH THE VALUES OF CONVENTIONAL LIVER FUNCTION TESTS.

(It.) Marrone, N. (Inst. Gen. Clin. Surg., U. Bologna, Italy), F. Farruggia and L. Zanella. *Riv Gastroent* 19(6, Suppl.):739-750, 1967.

SGOT and SGPT activities were determined at 24-hr intervals on 50 fasting patients (31 females and 19 males, aged 18-77 yr) for 7 days in a row. The patients included 3 with simple cholecystitis; 17 with cholelithiasis; 2 with cholelithiasis and choledocholithiasis; 1 with cholelithiasis, choledocholithiasis, and hepatic lithiasis; 1 with gallbladder hydrops; 1 with gallbladder torsion; 1 with glissonitis; 4 with gallbladder empyema; 7 with echinococcal cysts of the liver; 4 with carcinoma of the head of the pancreas; 2 with stomach carcinoma; 4 with biliary neoplasms; 2 with hepatic carcinoma; and 1 with metastatic carcinoma of the liver. Surgery was performed under closed-circuit anesthesia with NO$_2$, O$_2$, ether, and succinylcurarin after premedication with atropine and promethazine and induction with pentothal. Results for each case are presented in a table. SGOT and SGPT values were within normal limits in most patients with cholecystitis and echinococcosis but were elevated in those with obstructive jaundice. After surgery, transaminase values were at least slightly elevated in all patients and were highest in those with jaundice and severe toxic conditions. Values had returned to normal after 7 days in patients with cholecystitis and echinococcosis and when the obstruction was removed in obstructive jaundice. In patients with obstructive jaundice caused by neoplasms the transaminase values decreased significantly after surgery, but not to normal levels. In general, transaminase values agreed with the results of conventional liver function tests.

5860 SMALL-CALIBER FIBERSCOPE FOR VISUALIZATION OF THE URINARY TRACT, BILIARY TRACT, AND SPINAL CANAL.

(E.) Takagi, T. (Fac. Med., U. Tokyo, Japan) T. Go, H. Takayasu, Y. Aso and R. Hioki. *Surgery* 64(6):1033-1038, 1968.

The flexible fiberoptic endoscope designed by the authors is 70 cm long and 2.7 mm in diameter and has both the optical and illuminating systems housed in a single plastic sheath. The optical system is an image-transmitting fiber bundle 1 mm^2 in cross-section with about 7000 glass fibers, each 12 microns in diameter. An annular layer of the light-transmitting fiber bundle surrounds this image-transmitting bundle. The objective lens has an angle of view of 35° and a fixed focus of 1.8 mm with a depth of field of 5-50 mm. A schematic drawing shows the entire assembly. A technique for visualizing the common bile duct and intrahepatic bile duct was first established in cadavers and then in patients with choleithiasis and choledocholithiasis during laparotomy. The common bile duct, the bifurcation of the hepatic and cystic duct, and the intrahepatic bile duct were clearly visualized and are illustrated in figures.

5861 ELIMINATION OF COLOR QUENCH IN LIQUID SCINTILLATION COUNTING OF FECAL EXTRACTS BY OZONIZATION.

(E.) Krabisch, L. (Div. Physiol. Chem., Chem. Ctr., U. Lund, Sweden) and B. Börgstrom. *Scand J Gastroent* 3(5):458-460, 1968.

The deep color of fecal lipid extracts tends to interfere with the analysis of radioactivity by liquid scintillation counting. The color quench is often such that valid counts cannot be obtained using internal standardization. O$_3$ has been found to be an effective decolorizing agent which can be used on the nonsaponifiable fractions of fecal lipid extracts without loss of radioactivity. The neutral steroid fractions from subjects on a normal diet and subjects fed a sitosterol-^{3}H labeled meal were extracted with 1:1 chloroform: methanol, saponified and extracted with light petroleum ether. Ozonization was performed in solution with 10 ml chloroform in 100 ml flasks. Decolorization was usually complete in about 5 min. No loss of radioactivity occurred when a solution of pure cholesterol-4-^{14}C was so treated. The presence of other lipids (e.g., triolein) led to low counts if counting was done immediately after ozonization or after only a short period of desiccation. Prolonged storage *in vacuo*, however, resulted in complete recovery of activity.

5862 TRANSDUODENAL EXPLORATION OF THE COMMON DUCT WITHOUT CHOLEDOCHOTOMY.

(E.) Michels, T. A. (Highland Gen. Hosp., Oakland, Calif.) and A. J. Hunnicutt. *Amer J Surg* 116(2):152-157, 1968.

Transduodenal exploration of the common duct without choledochotomy was used in 82 patients with suspected common duct stones (31), stenosis of the duodenal papilla (36) or pancreatic disease (16). Common duct stones were subsequently found in 10 patients; these were successfully removed through the ampullary end of the common duct. In addition, 10 cases with papillary stenosis, 3 with adenomatous polyps, 3 with inflammatory polyps and 1 with carcinoma of the ampulla were found. Malfunction of the duodenal papilla (stenosis) was diagnosed clinically by the following triad: 1) pain immediately upon eating, 2) tenderness in the pancreatic area, 3) a feeling of epigastric fullness and a sensitivity to tight clothing. Of 36 suspected cases, stenosis, as defined by an ampullary diameter of 2 mm or less, was present in 31. Also found were 4 granular polyps and 1 adenomatous polyp in the ampulla. Patients (16) explored for a preoperative diagnosis of pancreatic carcinoma or reflux biliary pancreatitis showed pancreatitis in 8 cases and stenosis of the duodenal papilla in 10. It is noted that the transduodenal approach to the common duct eliminates T-tube drainage and the risks of choledochotomy and provides direct visualization of the papillary area.

5863 EVALUATION OF THE INDUCED TETRACYCLINE FLUORESCENCE TEST IN THE GASTRIC SEDIMENT.

(Sp.) Ramírez Ramos, A. (2nd of May Hosp., Lima, Perú), B. Herrera Mandelli and M. E. Morán. *Acta Cancerol* 7(1):17-25, 1968.

Fluorescence measurements were made on the gastric sediment, basal secretion, posthistamine secretion, and gastric washings from 50 controls who had not been given tetracyclines and on gastric sediment from 1) 25 patients with duodenal ulcers, chronic atrophic gastritis, or cholelithiasis; 2) 28 patients with stomach ulcers; and 3) 45 patients with malignant stomach neoplasms including 34 with confirmed adenocarcinomas and 1 with primary Hodgkin's disease of the stomach. No patients with pyloric obstructions were included. Patients in groups 1-3 were given a short course of oxytetracycline parenterally or demethylchlortetracycline p.o. or a long course of oxytetracycline p.o. Gastric washings were taken 44 hr after the last dose of tetracycline, the solution was adjusted to pH 8-9 with 5% $NaHCO_3$, and fluorescence measurements were made on the sediment obtained by centrifuging at 30,000 rpm for 10 min. Spontaneous fluorescence was seen in 30 of the 200 samples from controls; it occurred most frequently in sediment and basal secretion and less often in gastric washings. No fluorescence was found in any of the samples from patients in group (1). Three false positive tests were obtained from patients with stomach ulcers. These included 2 patients who had been given oxytetracycline and 1 who had been given demethylchlortetracycline. Sediment from patients with stomach cancer was fluorescent in 17 (54.9%) of the 31 patients who were given oxytetracycline and in 12 (85.7%) of the 14 who were given demethylchlortetracycline. These results indicate that the best diagnostic results are obtained with demethylchlortetracycline.

5864 A POSSIBLE TEST FOR INTESTINAL ABSORPTION OF FATS: PRELIMINARY REPORT. *(Sp.)* Olmedo Centeno, C. (Mexican Inst. Soc. Sec., Puebla Mexico). *Medicina (Mex)* 48(1047):584-593, 1968.

Lipid absorption studies were run on 15 controls (7 males and 8 females, aged 3-78 yr) and 40 patients with gastrointestinal diseases and possible malabsorption (aged 3-69 yr). After urinating, subjects were given chloramphenicol palmitate (1 g of chloramphenicol base to subjects weighing more than 50 kg, 20 mg base/kg to those weighing less). The method of Bessman and Stevens was used to measure the amount of chloramphenicol excreted in 24-hr urine specimens and that present in blood samples 2 hr after administration of its palmitate. Controls excreted a mean of 75.8 ± 5.3% chloramphenicol in the urine and their blood samples contained a mean of 10.55 ± 5.3% chloramphenicol in the urine and their blood samples contained a mean of 10.55 ± 1.9 μg/ml. Of the patients, 8 had blood and urine values lower than normal, 9 had normal blood levels and low urine values, and 23 had normal blood and urine values. Those with low blood and urine values included 3 patients with giardiasis, 2 with chronic pancreatitis, 2 with postnecrotic cirrhosis, and 1 with viral hepatitis. Those with low urine and normal blood values included 3 with nonspecific diarrhea, 2 with giardiasis, 2 with "diabetic diarrhea", 1 with diabetes mellitus, and 1 with amebiasis.

5865 ELECTROPHORETIC BEHAVIOR OF LDH, GOT, AND G6PD ISOENZYMES IN SOME CARCINOMAS OF THE GASTROINTESTINAL TRACT (ESOPHAGUS, STOMACH, CECUM, COLON, AND RECTUM). *(It.)* Notario, A. (Inst. Gen. Clin. Med., U. Pavia, Italy), E. Bobbio Pallavicini and G. Malvicini. *Minerva Med* 59(54):3049-3060, 1968.

Electrophoresis on strips of Cellogel was used to separate the isoenzymes in homogenates of gastric mucosa from 5 patients who did not have stomach cancer and homogenates of tumor tissue and adjacent normal mucosa from 3 patients with esophageal carcinoma, 5 with gastric neoplasms, 3 with neoplasms of the colon, 2 with neoplasms of the cecum, and 5 with rectal neoplasms. Except for the greater frequency of thin bands migrating toward the cathode, the separation of lactic dehydrogenase (LDH) isoenzymes was very similar in gastric mucosa from patients without cancer and in normal mucosa taken close to the site of tumors. In tumor tissue, however, there was a definite increase in fractions migrating toward the cathode and a proportional reduction in those migrating toward the anode, particularly the LDH_5 fraction. In tumor tissue from patients with gastrointestinal neoplasms an inversion occurred in the ratios between slow and fast fractions of glutamic-oxaloacetic transaminase (GOT) and glucose-6-phosphate dehydrogenase (G6PD) migrating toward the anode. Increases occurred in the slow fractions and decreases in the fast fractions. These differences in the electrophoretic behavior of isoenzymes in normal and tumor tissue are attributed to changes in cell metabolism related to the degree of cell maturation, the rhythm of cell proliferation, and biochemical and morphological features of the cells.

5866 A CLINICAL STUDY OF SERUM GLUTAMIC OXALACETIC TRANSAMINASE ISOENZYMES IN LIVER DISEASES. *(E.)* Gabrieli, E. R. (E. J. Meyer Mem. Hosp., Buffalo, N. Y.) and A. Orfanos. *Proc Soc Exp Biol Med* 128(3):803-807, 1968.

A column chromatographic technique was used to examine SGOT isoenzyme patterns in the sera of 10 healthy subjects and 21 patients with various forms of liver disease [chronic alcoholism (10), cirrhosis (6), acute viral hepatitis (5)]. In healthy subjects, 3 distinct isoenzymes were found, while in patients with liver diseases, the SGOT isoenzyme pattern was distorted. Two isoenzymes (A_1 and B) were substantially increased, whereas the third (A_2) remained the same. The most striking changes were noted in the acute viral hepatitis group; isoenzyme A_2 was not detectable, and the isoenzyme pattern was similar to that in liver homogenates. The changes in isoenzyme patterns are a more precise reflection of the extent of hepatocellular damage than the total SGOT activity.

5867 THE VALUE OF DETERMINING THE ABSORPTION OF ^{58}Co-LABELLED VITAMIN B_{12} IN SOME DISEASES OF THE GASTROINTESTINAL TRACT. *(Cz.)* Vulterinová, M. (Inst. Res. Human Nutrit., Prague, Czechoslovakia), I. Skála, J. Brousil, F. Hrubá, A. Krondl, R. Stastna and M. Kotková. *Cesk Gastroent Vyz* 22(5):349-356, 1968.

The Schilling test was run on 116 subjects, including 39 who had undergone gastrectomies (19 Billroth I, 20 Billroth II) in the preceding 5 yr for peptic ulcers; 27 who had undergone resections of the small intestine; 11 recovering from acute pancreatitis; 19 with chronic recurrent pancreatitis; 10 with diffuse lesions of the intestinal mucosa due to celiac disease, diffuse enteritis, scleroderma, and regional enteritis; and 10 normal controls. Results of the Schilling test were significantly lower among patients who had undergone both types of gastrectomies than among the controls. Significantly lower values were obtained among patients with gastrectomies and symptoms of malabsorption than among asymptomatic patients or controls. Histamine-resistant achlorhydria was present in a significantly larger number of patients with symptoms of malabsorption. Administration of 50 mg of intrinsic factor did not improve vitamin B_{12} absorption in 5 of the 15 patients tested. Vitamin B_{12} absorption was also significantly lower than normal among patients who had undergone extensive resections of the small intestine and those operated on for terminal ileitis, but was normal among those in whom the terminal ileum was not involved. There were no significant differences between patients with acute pancreatitis and controls, but patients with chronic pancreatitis had values significantly higher than normal. Patients with diffuse mucosal lesions of the small intestine had normal values for vitamin B_{12} absorption unless the terminal ileum was affected. There were no correlations between the results of the Schilling test and fecal fat or nitrogen excretion and results of the xylose test. There was a positive correlation between the Schilling test and serum vitamin B_{12} levels. It is concluded that the Schilling test is suitable for early diagnosis of vitamin B_{12} deficiency and the diagnosis and prevention of macrocytic anemia in gastrointestinal diseases.

5868 **SEVERE GASTROINTESTINAL HAEMORRHAGE: HAEMANGIOMATA DEMONSTRATED BY SELECTIVE VISCERAL ARTERIOGRAPHY.** *(E.)* Ashby, E. C. (Westminster Hosp., London, England), T. J. Mott and F. Starer. *Brit Med J* 4(5633):737-739, 1968.

Case histories are presented for 2 patients in whom selective visceral arteriography was found to be invaluable for the diagnosis of severe gastrointestinal hemorrhage associated with cavernous hemangiomas: a 28-yr-old male with a large gastric hemangioma and a 41-yr-old male with a small jejunal hemangioma. The technique, which has been highly successful with the celiac and superior mesenteric arteries and with the inferior mesenteric artery in other than elderly subjects, is safe, need not unduly delay surgery, and may reduce intraabdominal manipulations and operation time by locating the bleeding source. Arteriography is suggested when laparotomy does not disclose the cause of bleeding, when the patient's general condition makes a prolonged operation impossible, and when superficial hemangiomas have been noted.

5869 **DIRECT PORTAL HEPATOGRAPHY AND METABOLIC STUDIES VIA THE REOPENED UMBILICAL VEIN: EFFECT OF VASOACTIVE DRUGS ON PORTAL PRESSURE, BLOOD GASES AND LACTATES.** *(E.)* White, J. J. (Johns Hopkins U. Sch. Med., Baltimore, Md.). *Amer Surg* 34(12):852-859, 1968.

Portal hepatography and metabolic studies of the splanchnic circulation were successfully carried out in 27 of 36 adult and pediatric patients by passing a cannula to the portal hilum via the reopened umbilical vein. Only 2 of the 9 failures were due to obliteration of the vein. Concomitant portal pressure measurements, liver biopsies and hepatoportograms demonstrated normal livers in several patients previously considered to be suffering from hepatic disease, and indicated the extent of fibrosis, degree of portal venous outflow block and collateral circulation in patients with cirrhosis. Portal hepatography permitted delineation of hepatic tumors as small as 1 cm in diameter, and the umbilical vein route was successfully utilized for the diagnosis and chemotherapy of liver abscesses. Serial resting portal pressure measurements reflected the underlying hepatic pathology and degree of portal venous obstruction, with little variation due to posture or meals. Portal lactate levels were generally elevated in portal hypertension. While vasopressin administration reduced portal pressure, metabolic acidosis and excess lactate production resulted which required correction by isoproterenol. It is suggested that the use of vasopressin to reduce portal hypertension in a debilitated cirrhotic with variceal hemorrhage be undertaken with great care because of the danger of producing a concomitant metabolic acidosis.

5870 **SERUM IMMUNOGLOBULINS IN THE DIFFERENTIAL DIAGNOSIS BETWEEN INTRAHEPATIC VIRAL JAUNDICE AND EXTRAHEPATIC OBSTRUCTIVE JAUNDICE.** *(E.)* LoGrippo, G. A. (Henry Ford Hosp., Detroit, Mich.), T. A. Fox and M. A. Block. *Henry Ford Hosp Med J* 16(4):289-300, 1968.

Serum immunoglobulin levels were determined in 75 patients with jaundice (32 with viral hepatitis and 43 with extrahepatic obstruction due to chole- or choledocholithiasis, pancreatic carcinoma, biliary tract cancer, pancreatitis or other conditions). Serum IgM and IgG levels were elevated at least 2 standard deviations above the mean values in 32 and 23 of the patients with viral hepatitis, resp., and all 3 immunoglobulins were significantly elevated in 13 of the 32. However, there was no relation between immunoglobulin elevation and severity as reflected by serum transaminase levels. Neither IgM nor IgG levels were significantly elevated in 33 of 43 patients (76.7%) with obstructive jaundice, although 21 showed elevations in IgA. Of the remaining 10 patients with obstruction, 7 showed elevated IgM levels, 1 had an elevated IgG, and 2 had elevation of both IgM and IgG. These results emphasize the value of specific serum immunoglobulin determinations in the differential diagnosis of the jaundiced patient.

5871 **AN EVALUATION OF DIAGNOSTIC PARACENTESIS IN THE ACUTE ABDOMEN.** *(E.)* Prout, W. G. (St. Peter's Hosp., Chertsey, England). *Brit J Surg* 55(11):853-857, 1968.

Diagnostic abdominal tap was performed 71 times in 50 patients with acute abdominal pain (26 males and 24 females, 12-86 yr old). The group included 25 patients (group 1) in whom the diagnosis and management were uncertain and 25 patients (group 2) in whom tap was performed immediately before laparotomy. In group 1, there were 19 true positive, 1 false positive, 3 true negative, and 2 false negative taps. In group 2, there were 17 true positive, 1 false positive, 4 true negative, and 3 false negative taps. Diagnostic paracentesis may be of value in the differential diagnosis of acute pancreatitis and abdominal trauma, in the postoperative period, and in the patient in whom extra-abdominal factors may make the diagnosis of intraperitoneal disease difficult.

5872 **DRIP INFUSION CHOLANGIOGRAPHY.** *(E.)* Payne, R. F. (Bristol Royal Infirm., England). *Clin Radiol* 19(3):291-295, 1968.

The results of drip infusion cholangiography, using 250 ml of 30% dextrose and 60 ml of Biligrafin Forte administered i.v. over the course of 1 hr, in 15 patients (26-76 yr old) were compared with those obtained in another 25 patients by conventional i.v. cholangiography with 20 ml of the same contrast medium. In the study series, the common bile duct did not fill in 2 (8%), filled poorly in 4 (16%), showed average filling in 10 (40%), and showed good filling in 9 (36%) cases. In the patients studied by conventional i.v. cholangiography, the common bile duct did not fill in 6 (24%), filled poorly in 7 (28%), showed average filling in 6 (24%), and showed good filling in 6 (24%) cases. Among the 20 patients in the study group who still had gallbladders, there was no filling in 4 (20%), poor filling in 8 (40%), and good filling in 8 (40%) cases. Comparable figures for the 18 patients with gallbladders studied by conventional i.v. cholangiography were 15 (84%), 1 (5%), and

2 (11%). Pyelograms were visible in 5 (20%) of the patients subjected to drip infusion and 13 (52%) of the patients given conventional i.v. cholangiography. The drip infusion technique provides superior opacification of the biliary tract, and often delineates the gallbladder when other methods have failed.

5873 **ANGIOGRAPHY IN ACTIVE GASTROINTESTINAL BLEEDING.** *(E.)* Salmon, R. B. (Charlotte Mem. Hosp., N. C.). *Coll Works Cardiopulm Dis* 14:77-86, 1968.

The angiographic diagnosis of a bleeding site relies on the demonstration of contrast material in the bowel lumen and is relatively independent of the presence of other foreign materials such as blood, food, or feces in the same area. Five cases of gastrointestinal bleeding are presented in which angiography established the source of bleeding when conventional methods, including laparotomy, had either failed or would have been useless in localizing the bleeding point. Patients selected for visceral angiography should carefully fulfill the following criteria: (a) active bleeding at the time of angiography, (b) failure of diagnosis by conventional means, or inability to utilize conventional techniques due to the severity of hemorrhage, and (c) the decision to operate on the patient has already been made, so that the study is actually a preoperative one. Angiography is not meant to replace conventional studies where they are applicable. However, in a patient who continues to bleed and in whom surgery is indicated, angiography should precede barium studies. Barium studies can immediately follow arteriography if the bleeding source still cannot be identified.

5874 **THE PLAIN CHEST RADIOGRAPH IN SEVENTY-FIVE CASES OF PRIMARY CARCINOMA OF THE LIVER.** *(E.)* Sanders, C. F. (Mpilo African Hosp., Bulawayo, Rhodesia). *Clin Radiol* 19(3):341-346, 1968.

Significant abnormalities in the chest X-ray were observed in 60 of 75 African patients (68 males and 7 females, 9-77 yr old), with primary carcinoma of the liver. Observed lung changes were divided into: (a) elevation of the diaphragm, 49 cases (but a definite localized bulge was seen in only 7 cases); (b) 'parallel lines at the right base', 35 cases; (c) lack of clarity of the cardiophrenic angle, 35 cases; and (d) secondary deposits, 24 cases. There was a close association between the first 2 signs, and 31 of the 35 cases in which the cardiophrenic angle was obscured also had elevation of the diaphragm. The secondary deposits were of 2 main forms: 1) multiple, small, nonspecific rounded deposits, and 2) large, clear-cut rounded deposits. Pleural effusions were seen in only 3 cases. Of the 60 patients with lung changes, 37 were dead within a few months, and only 1 patient lived for more than 6 months. However, no appreciable difference in life span was seen in the 15 patients without lung changes. It appears that hepatoma patients with lung changes do not have any different prognosis than those without lung changes.

5875 **DIAGNOSTIC VALUE OF MARROW HEMOSIDERIN PATTERNS.** *(E.)* Nixon, R. K. (Henry Ford Hosp., Detroit, Mich.) and J. P. Olson. *Ann Intern Med* 69(6):1249-1254, 1968.

Marrow hemosiderin patterns in 500 patients were analyzed to determine their diagnostic value. The quantity and predominant particle size of iron was evaluated, and an excellent correlation of the hemosiderin pattern with known ferrokinetics was obtained in each type of anemia studied (iron-deficiency, primary or secondary refractory, hemolytic, post-hemorrhagic, and vitamin B_{12} deficiency). The previously reported absence of marrow iron in polycythemia vera was confirmed in all cases studied. A hemosiderin pattern differing from that of cirrhosis or due to other causes was observed in primary hemochromatosis. It is felt that direct visualization of marrow hemosiderin patterns provides an accurate, rapid, and simple means of evaluating iron dynamics. The procedure is of value in the initial diagnostic approach to the polycythemias. Hemosiderin patterns may be of singular aid in the differentiation of primary hemochromatosis and cirrhosis. In contrast to ferrokinetic studies, the simplicity of marrow hemosiderin evaluation lends itself to routine application.

5876 **DRAINAGE OF RADIOGRAPHIC CONTRAST MEDIA FROM THE ABDOMINAL CAVITY: EXPERIMENTAL STUDIES IN RATS.** *(E.)* Lindgren, I. (U. Cent. Hosp., Turku, Finland), E. J. Nagy and P. Virtama. *Acta Radiol* 7(6):481-488, 1968.

The absorption and possible toxic effects of water-soluble, oily and particulate contrast media were studied in adult Wistar rats of both sexes after i.p. injection of 0.1-0.25 ml of Lipiodol, Thorotrast 25%, Micropaque 10% suspension, zirconium oxide 10% suspension, Dionosil Aqueous, Isopaque, Solu-Biloptin, Urografin, or Perjodal H. Animals were examined radiographically immediately after injection, at 2 and 10 hr, and at 2, 5, 7, 21, and 32 days. $^{140}BaSO_4$ or $Ba^{35}SO_4$ (300 μC) was injected i.p. into other animals which were examined scintigraphically every other day. Dionosil, Isopaque, Solu-Biloptin, Perjodal and Urografin disappeared from the peritoneal cavity within 1-4 hr and could not be found in any other organs. Thorotrast was seen in the lymphatic channels of the diaphragm and the mediastinum at 2-3 days and in the lymph glands of the neck within 1 week. Lipoidol remained in the peritoneal cavity for 8-10 days and persisted for 30 days in mediastinal and diaphragmatic lymphatic channels. Barium sulfate and zirconium oxide remained in the peritoneal cavity for up to 60 days; neither appeared outside the peritoneal cavity and adhesions always occurred after 20 days. Total-body scintigrams revealed a small part of the radioactive barium suspension in the upper mediastinum 4-8 days after injection.

5877 **THE INTRADERMAL TEST IN THE DIAGNOSIS OF HUMAN INTESTINAL CAPILLARIASIS.** *(E.)* Cabrera, B. D. (Inst. Hyg., U. Philippines, Manila) and R. B. Silan. *Acta Med Philipp* 4(4):175-180, 1968.

Intradermal tests using suprascapular injections of 0.05 ml *Capillaria* antigen (supernatant of dried adult worms in sterile merthiolated saline) and 0.05 ml sterile merthiolated saline as a control were performed in 71 capillariasis patients (47 males and 24 females) at the San Lazaro and Philippine General Hospitals and in 51 controls (30 American Peace Corps Volunteers (PCV) and 21 Filipino student nurses and technicians). Wheal areas of 1.0 cm^2 or greater were considered positive and those of 0.9 cm^2 or lower were considered negative. Antigen wheal areas (in cm^2) averaged 1.6782 in capillariasis patients (1.8436 in males and 1.3542 in females), 0.6100 in male Filipino controls, 0.5545 in female Filipino controls, 0.5824 in male PCV, and 0.5545 in female PCV. Control wheal areas (in cm^2) averaged 0.4972 in the capillariasis patients (0.5340 in males and 0.4250 in females), 0.5200 in male Filipino controls, 0.3545 in female Filipino controls, 0.4412 in male PCV, and 0.4615 in female PCV. The greater reactivity found in males suggests that separate limits are needed for the two sexes. When stool examinations were performed to determine the sensitivity and specificity of the intradermal test for capillariasis, 57 out of 60 egg passers (95%) had been diagnosed as positive with the intradermal test and 2 American controls were false positive. The fact that 7 out of 11 "watchers" (persons staying at the bedside of capillariasis patients during confinement) were positive for the skin test and negative for *Capillaria* eggs, were members of the patients' families, and came from the same endemic area suggests the possibility of either past or current infections. No cross-reactions were observed with trichuriasis, ancylostomiasis, or schistosomiasis.

5878 **THE NORMAL AND ABNORMAL PANCREATIC SCAN.** *(E.)* Melmed, R. N. (Royal Free Hosp., London, England), J. E. Agnew and I. A. D. Bouchier. *Quart J Med* 37(148):607-624, 1968.

The limits of normality and patterns of abnormality of pancreatic scans using ^{75}Se-selenomethionine are defined on the basis of a study of 200 patients with pancreatic and extrapancreatic diseases. The pancreatic diseases included carcinoma, pancreatitis, pancreatic pseudocyst, diabetes mellitus, previous partial pancreatectomy, fibrocystic disease and amyloid disease. The pancreatic scans of these 73 patients were normal in 13, abnormal in 52 and unreadable in 8, the poorest diagnostic reliability being noted in diabetes and acute pancreatitis. The extrapancreatic diseases included intrahepatic cholestasis, bile duct carcinoma, cholelithiasis, peptic ulcer, previous gastric surgery or vagotomy, aortic aneurysm, sarcoidosis, Gardner's syndrome and others. The pancreatic scans of these 60 patients were normal in 43, abnormal in 16 and unreadable in 1, the most false-positive being obtained in aortic aneurysm, cholelithiasis, peptic ulcer and following gastric surgery. Pancreatic scans of 67 normal controls were normal in 57, abnormal in 4 and unreadable in 6. The results of a series of 40 consecutive unselected pancreatic scans which were evaluated without any knowledge of the clinical details were used to assess the diagnostic accuracy of the procedure. These scans were assessed as being either normal or abnormal, and if abnormal, the diagnostic possibilities were listed in order of probability. There were 3 errors in diagnosis in the 40 patients, including 2 false-positive scans among the 23 with a normal pancreas and one missed case of pancreatic carcinoma. It is concluded that an unequivocally normal pancreatic scan is strong evidence that the gland is normal.

5879 **STUDY OF GASTRIC SECRETION BY THE TELEMETRIC METHOD: PERSONAL EXPERIENCE WITH 120 EXAMINATIONS.** *(Fr.)* Hancy, A. (Northern Hosp., Marseille, France) and H. Monges. *Gaz Med (France)* 75(30):6001-6017, 1968.

Measurements of the pH of the gastric juice in 90 fasting subjects, using a Heidelberg capsule,revealed no significant difference between the mean for 45 normal controls (1.9) and that for 25 patients with bulbar ulcers (1.7). However, none of the ulcer patients had pH values higher than 3 and 15 of the 25 had pH values below 1. The mean pH values were significantly elevated in 11 patients with stomach ulcers and in 9 with confirmed gastritis (4.1 and 5, resp.). Fasting pH values measured on 29 subjects by the Heidelberg capsule were lower in 22 cases than those measured by pH determinations on aspirated fluid. Alkali neutralization times, determined telemetrically by Noller's method, were significantly shorter than in controls in 9 patients with bulbar ulcers and hyperchlorhydria and significantly longer in 12 patients with stomach ulcers or gastritis and hypochlorhydria. A modification of the method of Sircus *et al.* for the successive neutralization test showed that the mean total acid flow/hr after histamine stimulation was significantly greater in 17 patients with bulbar ulcers than in 30 normal controls, while that in 8 patients with stomach ulcers and 11 with gastritis was significantly smaller. On the average, patients with bulbar ulcers required twice the number of titrations as controls and 3 times as many as those with stomach ulcers or gastritis. The shape of the curve distinguished between subjects with normochlorhydria, hyperchlorhydria, and hypochlorhydria, and threshold values for 6 parameters are tabulated to distinguish between these states. A comparison of histamine and betazole stimulation in the successive neutralization test on normal subjects and those with bulbar ulcers showed that betazole gave clearer results and was tolerated better than histamine. There was good agreement between values for total acid flow/hr determined telemetrically and those obtained by the classical intubation method. However, in contrast to the findings of Sircus *et al.*, acid flow was higher in 17 of 24 subjects when determined telemetrically. This is attributed to gastrin-induced acid secretion caused by alkaline conditions at the antrum. The telemetric method was also used to demonstrate the superiority of buffering agents over sodium bicarbonate.

5880 **TRANSPARIETAL HEPATIC CHOLANGIOGRAPHY: ITS TECHNIQUE, EFFECTIVENESS, COMPLICATIONS, AND RESULTS (A STUDY OF 101 CASES).** *(Por.)* Reibscheid, S. (São Paulo Med. Sch., Brazil), F. Secaf, M. P. Vilela, S. J. Miszputen and A. J. Ganc. *Rev Ass Med Brasil* 13(12):416-422, 1967.

The method of Edholm, Fernstrom, Lindblom, and Seldinger was used to obtain transparietal hepatic cholangiograms from 101 patients in order to make a differential diagnosis between biliary and obstructive jaundice and to diagnose biliary tract lesions in anicteric patients. Cholangiography gave results which agreed with the final diagnosis in 76 of these 101 cases. This procedure gave the poorest results in patients without biliary tract obstruction, in hepatitis, cirrhosis, postcholecystectomy syndrome, chronic calcareous cholecystitis, and schistosomiasis (16 correct diagnoses out of 28), and in those with chronic pancreatitis (8 of 14). In 22 cases the results of transparietal hepatic cholangiography agreed with those of surgical cholangiography performed by injecting contrast medium above the obstacle. Complications occurred in 9 patients with mechanical obstructions, including hemobilia in 4 patients, choleperitoneum in 2, fever in 2 with choledocholithiasis, and hemoperitoneum in 1. All recovered without difficulty.

5881 **THE ORAL AMMONIUM CHLORIDE LOADING TEST IN NORMAL SUBJECTS, CIRRHOTICS,**

AND PATIENTS WITH OBSTRUCTIVE JAUNDICE: A COMPARATIVE STUDY. *(Por.)* Reibscheid, S. (São Paulo Med. Sch., Brazil), S. J. Miszputen, A. J. Ganc, R. D. Santos, J. M. Marlet and M. P. Vilela. *Rev Ass Med Brasil* 13(11):375-378 , 1967.

Syrup containing 3 g of NH_4Cl was given p.o. to 41 normal subjects, 27 with hepatic cirrhosis, and 20 with obstructive jaundice after they had fasted. A modification of Seligson and Hirahara's method was used to determine ammonia in whole blood collected from the femoral artery before and 45 min after ingestion of NH_4Cl. No changes occurred in the arterial ammonia levels of normal subjects. Fasting arterial ammonia levels were above normal limits in 44.4% of the patients with cirrhosis and 50% of those with obstructive jaundice. After ingestion of NH_4Cl these values increased to 96.3% and 75%, resp. Measurements of ammonia were made on blood from the femoral artery, suprahepatic veins, and superficial veins of the abdominal wall originating from the portal vein on 1 patient with schistosomiasis and Curveilhier-Baumgarten syndrome. No increases in ammonia levels occurred in blood from the suprahepatic veins while large increases occurred in blood from the femoral artery and superficial veins of the abdominal wall. This suggests that the liver removes ammonia adequately and that elevated ammonia levels in arterial blood are caused by a portosystemic shunt through the umbilical vein.

5882 THE APPLICATION OF I^{131}-LABELED HEAT-DENATURED HUMAN SERUM ALBUMEN TO RADIOACTIVE LIVER SCANNING.

(E.) Watson, A. (Royal Infirm., Manchester, England) and H. B. Torrance. *J Roy Coll Surg Edinb* 13(6):315-323, 1968.

Liver scanning using ^{131}I-labeled heat-denatured human serum albumin (HSA) was performed in 170 patients with suspected hepatomegaly, jaundice, upper abdominal masses, and malignant disease. In the 104 patients in whom laparotomy or autopsy data were available for confirmation, overall accuracy was 93%: 58 out of 61 patients were actually normal and 35 out of 39 were actually positive for hepatic metastases. Of the 35 with metastases, 15 (43%) had clinically impalpable livers and 18 (52%) had normal liver function tests. Hepatic cirrhosis (18), Riedel's lobe (3), hepatoma (2), and 1 each of the following: subphrenic abscess, gastric carcinoma with lymph node involvement, retroperitoneal sarcoma, and ovarian cyst were also revealed; 4 scans were technically unsatisfactory. The smallest detectable lesion was 2.0 cm. The fact that liver scanning is relatively inexpensive to perform, causes little patient inconvenience, and gives a low liver radiation dose renders the procedure a valuable aid in the differential diagnosis of hepatic disease.

5883 CLINICAL ELECTROGASTROGRAPHY AND ITS RELATIONSHIP TO GASTRIC SURGERY.

(E.) Nelsen, T. S. (Stanford U. Sch. Med., Palo Alto, Calif.) and S. Kohatsu. *Amer J Surg* 116(2):215-222, 1968.

Comparative gastric electromyographic studies showed that the gastric electrical frequency and its statistical variations are useful diagnostic indicators of vagal disturbance, impaired response to feeding, and the presence of multiple pacemakers or conduction paths. Electrodes implanted directly into the muscle were used in 13 patients undergoing gastrostomy for obstructing esophageal carcinoma (10), vagotomy for duodenal ulcer (2), or pyloroplasty for gastric retention (1). Cutaneous or swallowed electrodes, used in 15 additional patients and controls, gave tracings suitable for analysis by electronic data processing in 90% of the cases tested. The implication of such data with regard to surgery is that certain operations (transection of the stomach resection along the lesser curvature) may result in desynchronization by interrupting the propagation of gastric electrical and mechanical waves. In exploration of the stomach, longitudinal incisions should be used.

5884 THE INCIDENCE OF ACHLORHYDRIA AND HYPOCHLORHYDRIA IN HEALTHY SUBJECTS AND PATIENTS WITH GASTROINTESTINAL DISEASES.

(E.) Christiansen, P. M. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand J Gastroent* 3(5):497-508, 1968.

The incidence and significance of achlorhydria were evaluated by means of the augmented histamine test in 1881 patients (1010 males and 871 females, 10-89 yr old) with gastrointestinal diseases or symptoms and 170 healthy subjects (115 males and 55 females, 10-99 yr old). Among the 115 healthy males, 8 had achlorhydria, 4 had hypochlorhydria, and the mean acid output of the 103 others was 20.45 mEq HCl/hr. Among the 55 healthy females, no case of achlorhydria was found but 3 had hypochlorhydria; the remaining 52 had a mean acid output of 16.9 mEq HCl/hr. Among the 1881 patients with gastrointestinal disease, 120 had achlorhydria and 71 had hypochlorhydria. The incidences of achlorhydria and hypochlorhydria were higher in females than in males, but the differences were not significant. In patients younger than 60 yr the incidences of achlorhydria and hypochlorhydria were 2.3% and 2.0%, resp., but in the older patients, the incidence was 5 and 3 times as high, and the differences were significant for both males and females. The incidence of achlorhydria doubled in every decade from the 5th to the 8th in both sexes. The data demonstrated that 75% of achlorhydric and 63% of hypochlorhydric patients had gastric cancer, pernicious anemia, gastritis or a postoperative stomach. Patients with duodenal or prepyloric ulcer all had titratable acid secretion, but 2 patients with gastric ulcer had achlorhydria. In patients with gallbladder diseases the incidence of achlorhydria did not differ significantly from the incidence in healthy subjects. When adjusted for age, the incidence of achlorhydria and hypochlorhydria in healthy subjects did not differ significantly from that in the total number of patients with gastrointestinal diseases, and it is concluded that the secretory defects are related primarily to old age.

5885 THE APOMORPHINE TEST. I. CHOICE OF DOSE EVALUATED IN HEALTHY VOLUNTEERS

(E.) Fenger, H. J. (State Hosp., Copenhagen, Denmark) and E. Gudmand-Höyer. *Scand J Gastroent* 3(5):471-475, 1968.

The establishment of a proper dose level for the apomorphine test, a simple preoperative prognostic test for the dumping syndrome, was undertaken in 16 healthy volunteers who had previously been tested with intra-intestinal hypertonic glucose; 0.25, 0.5, 0.75, or 1.0 mg apomorphine was administered s.c. in an attempt to provoke symptoms similar to those provoked by 280 ml 18% glucose. Subjects in whom the glucose test failed to provoke symptoms showed a tendency to stronger reactions after the injection of 0.25 or 0.5 mg apomorphine; after adaptation, however, the results were the same as those produced by glucose. Too strong a reaction was generally elicited by 0.75 and 1.0 mg apomorphine, although the reactions of 1 subject to 0.75 mg apomorphine and glucose were identical. In these subjects showing severe persistent dumping symptoms following glucose provocation, the results with 0.5 mg apomorphine were similar to and as severe as those with glucose in all cases, while the results with 0.25 mg were weaker in all cases. An overall evaluation showed that provocation with 0.75 mg apomorphine produced stronger reactions than that with 0.5 mg, while there was no significant difference between 0.25 and 0.5 mg. Since 0.5 mg apomorphine provoked reactions identical to those following intra-intestinal glucose, this dosage is the most suitable for evaluating the predisposition to a post-gastrectomy dumping syndrome.

5886 **JEJUNAL BIOPSY IN THE MALABSORPTION SYNDROMES AND CHRONIC DIARRHEAS OF THE ADULT.** *(Fr.)* Vachon, A., P. Paliard, M. Abry and J. Barthe. *J Med Lyon* 49(1146):1211-1224, 1968.

5887 **"PROTECTED" JEJUNAL INTUBATION.** *(Fr.* Vachon, A., J. Barthe, J. Sedallian, M. Abry, Brunet and T. Khouzan. *J Med Lyon* 49(1146):1225-1235, 1968.

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5894 **THE GASTROCAMERA.** *(E.)* Hadley, G. D. (Middlesex Hosp., London, England). *Postgrad Med J* 44(515):763, 1968.

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5960 LAPAROSCOPY IN THE DIAGNOSIS OF OBSTRUCTIVE JAUNDICE. *(Rus.)* Bragin, F. A. (Patrice Lumumba U. Int. Friendship, Moscow, USSR), L. I. Gorokhov and V. I. Kochiashvili. *Sovet Med* 31(9): 75-78, 1968.

5961 CHROMOCHOLEGRAPHIC EXPLORATION OF THE GALIBLADDER. *(Rus.)* Korziuk, L. S. (Clin. Hosp., KGB, USSR Council Ministers, Moscow) and I. K. Novikova. *Sovet Med* 31(9):78-82, 1968.

5962 THE VALUE OF FIBROSCOPES IN THE ENDOSCOPIC DIAGNOSIS OF ESOPHAGOGASTRIC DISEASE. *(Fr.)* Debray, C. (Bichat Hosp., Paris, France), P. Housset, G. Perreau and J. Richard. *Entret Bichat [Med]* pp. 567-576, 1968.

5963 LAPAROSCOPY AS A TECHNIQUE FOR STUDYING THE SMALL INTESTINE. *(Fr.)* Debray, C. (Bichat Hosp., Paris, France), J. A. Paolaggi and B. Mugnier. *Entret Bichat [Med]* pp. 59-64, 1968.

5964 VASCULAR STUDIES IN THE RETROPERITONEAL TUMORS IN CHILDREN. *(Fr.)* Ingelrans, P. (U. Hosp. Ctr., Lille, France), P. Debeugny, J. Remy and M. P. Maillard. *Ann Chir Infant* 9(2):93-102, 1968.

5965 THE QUANTITATIVE DETERMINATION OF THE WEIGHT OF EXPERIMENTALLY INDUCED HEPATOMAS USING COLLOIDAL ^{198}Au. *(Ger.)* Matthes, T. (Inst. Cancer Res., Berlin, Germany), H. J. Altenbrunn and B. Kuhn. *Arch Geschwulstforsch* 32(4): 353-357, 1968.

5966 MEASUREMENT OF PORTAL PRESSURE AND TRANSUMBILICAL PORTOGRAPHY. *(Fr.)* Schmidt, A. (State Hosp., Copenhagen, Denmark) and M. Andreassen. *Actualites Hepatogastroent Hotel Dieu* 4(3):B95-B102, 1968.

5967 THE SIGNIFICANCE OF METOCLOPRAMIDE IN GASTROINTESTINAL RADIOLOGICAL DIAGNOSIS: A STATISTICAL STUDY. *(Fr.)* Collard, M. (Munic. Hosp., Charleroi, Belgium), P. Fraiture, J. M. Colette and J. Bacq. *Actualites Hepatogastroent Hotel Dieu* 4(3): B113-B119, 1968.

5968 CLASSIFICATION OF TECHNIQUES FOR PANCREAS BIOPSY. *(Sp.)* Figueroa, E. *An Paul Med Cir* 95(5):308-310, 1968.

5969 HEPATOGRAPHY BY MEANS OF LYMPHOGRAPHY (PRESENTATION OF TWO CASES). *(Sp.)* Nogueras, F. M. (Santa Cruz y San Pablo Hosp., Barcelona, Spain) and J. Vives Cunillera. *An Med [Cir]* 54(3):181-188, 1968.

5970 A NEW TEST FOR DYSPROTEINEMIA: THE CHLOROGENIC ACID TEST. II. APPLICATION TO SERA OF PATIENTS SUFFERING FROM HEPATIC DYSFUNCTION. *(Fr.)* Tayeau, F. (Fac. Med. Pharm., Bordeaux, France), F. J. Traissac, J. Marquevielle, J. Sechet, E. Joizier, A. Duhamel-Reiss and J. Gratadour. *Ann Biol Clin (Paris)* 26(10/12):1193-1201, 1968.

5971 DETERMINATION OF AMYLASE IN DILUTE BLOOD SERUM AS A TEST FOR CHRONIC PANCREATITIS. *(Rus.)* Tuzhilin, S. A. (Inst. Nutrit., Acad. Med. Sci. USSR, Moscow) and A. A. Petrova. *Lab Delo* (7):424-426, 1968.

5972 SIMPLE METHOD FOR OBTAINING A RELIABLE VACUUM IN ASPIRATION NEEDLE BIOPSY OF THE LIVER. *(Rus.)* Vinogradova, M. R. (Leningrad Med. Inst. Sanit. Hyg., USSR). *Lab Delo* (7):442, 1968.

5973 DIAGNOSIS AND FINDINGS IN EXUDATIVE ENTEROPATHIES. *(Ger.)* Gramlich, F. (1st Med. Clin., Johannes Gutenberg U., Mainz, Germany). *Therapiewoch* 18(48):2163-2165, 1968.

5974 FUNCTIONAL SCINTIGRAPHIC STUDIES IN INTERNAL MEDICINE. *(Ger.)* Adam, W. E.

(Ctr. Int. Med., U. Ulm, Germany), W. Böhm and W. J. Lorenz. *Therapiewoche* 18(48):2159-2160, 1968.

5975 HAZARDS AND ACCIDENTS IN THE USE OF CLOSED SYSTEMS FOR THE ADMINISTRATION OF CONTRAST MEDIA INTO THE COLON. *(Ger.)* Schmerwitz, W. (Citizen's Hosp., Stuttgart, Germany) and M. Staiger. *Therapiewoche* 18(48):2155-2156, 1968.

5976 THE CURRENT STATUS OF PANCREATIC GAMMAGRAPHY. *(Sp.)* Del Olmo, J. and E. Meroño. *Acta Iber Radiol Cancer* 23(83):15-22, 1968.

5977 CONTRIBUTION TO THE DIAGNOSIS OF CHRONIC PANCREATITIS: RADIOLOGICAL AND SCINTIGRAPHIC STUDY. *(It.)* Farruggia, F. (Inst. Gen. Clin. Surg. U. Bologna, Italy), L. Zanella and P. Berardi. *Riv Gastroent* 19(6, suppl.):819-854, 1967.

5978 THE SEMIAXIAL PROJECTION OF CLAESSENS AND DEJONG IN THE RADIOLOGICAL STUDY OF THE RECTOSIGMOID REGION. *(It.)* Mattii, M. (Inst. Radiol., U. Bologna, Italy) and C. Pecunia. *Riv Gastroent* 19(6, suppl.):789-909, 1967.

5979 LIVER BIOPSY IN THE DIFFERENTIAL DIAGNOSIS OF EXTRAHEPATIC AND INTRAHEPATIC CHOLESTASIS. *(Por.)* Scheuer, P. J. (Royal Free Hosp., London, England). *An Paul Med Cir* 95(5):317-319, 1968.

See also: 5722, 5744, 5747, 5764, 5795, 6140, 6141, 6203, 6212, 6235, 6378, 6625, 6643, 6651

GASTROINTESTINAL DISEASES

GENERAL

5980 **ACTIVITY OF THE REACTION FOR ACID MUCOPOLYSACCHARIDES IN THE SMALL AND LARGE INTESTINE DURING EXPERIMENTAL CHRONIC PERITONITIS.** *(Pol.)* Szulc, H. (3rd Surg. Clin., Acad. Med., Poznan, Poland) and C. Majewski. *Pat Pol* 19(1): 201-208, 1968.

In experiments on 70 adult rats of both sexes, specimens of mucosa were taken from the small and large intestines 7-365 days after the i.p. injection of 2.5 g of sterile talc, which resulted in the development of chronic peritonitis. Histochemical studies on the mucosal sections revealed a moderate increase in acid mucopolysaccharide content 7-10 days after injection of talc, but a marked decrease 14-21 days after injection. All specimens showed a distinctly positive reaction for acid mucopolysaccharides, which was slightly greater than normal, 30-42 days after injection of talc, while 365 days afterwards the reaction for acid mucopolysaccharides was only weakly positive. It is felt that the acid mucopolysaccharides may play an important role in the nonspecific defense mechanisms of the intestinal wall.

5981 **ASPIRIN–ROSETTA STONE OF GASTROINTESTINAL HEMORRHAGE: A POINT OF VIEW.** *(E.)* Smith, V. M. (Mercy Hosp., Baltimore, Md.) and R. R. Babb. *Milit Med* 133(12):965-970, 1968.

Intensive questioning of 100 patients treated for acute upper gastrointestinal bleeding during a 1-yr period revealed that 94 had taken a salicylate during the 24 hr preceding hemorrhage, compared to only 7 of a control group of 50 consecutive patients admitted for other reasons. Of the 94, 2 had also been taking prednisone, and 4 of the remaining 6 had taken either indomethacin (2 cases), phenylbutazone or an anticoagulant. Almost all of the 94 patients used salicylates intermittently rather than chronically, and in many cases no salicylates had been taken for months or even years before the most recent dose. The underlying pathology included hemorrhagic patchy gastritis (29 cases), duodenal or gastric ulcers (45 cases), esophageal varices (4 cases), and other or undetermined diseases, which is the same distribution seen in other series. Based on these findings, it is concluded that salicylates are the principal immediate cause of acute gastrointestinal bleeding, regardless of the underlying disease, and that the treatment of gastrointestinal bleeding should be modified to allow for a period of observation and salicylate withdrawal before surgery. The precise mechanism by which salicylates cause bleeding from preexisting lesions remains unclear.

5982 **TREATMENT OF HUMAN *TAENIA SOLIUM* AND *T. SAGINATA* INFESTATION WITH AN ETHER EXTRACT OF MALE FERN ADMINISTERED BY DUODENAL INTUBATION.** *(Por.)* Alterio, D. L. (Carlos Chagas Inst., Sao Paulo, Brazil). *Rev Hosp Clin Fac Med S Paulo* 23(3):150-152, 1968.

Male fern extract was used in the treatment of 100 patients, aged 5-68 yr, with taeniasis; most had previously been treated unsuccessfully with other preparations. The diagnosis of taeniasis was based on the finding of ova or proglottids in the stools. Preparation included a light meal the evening before treatment followed by a saline purgative and, in the morning, another purgative. A solution containing 6-7 g of an ether extract of male fern, 15-30 g Na_2SO_4, and 120-200 g of potion gommeuse was introduced into the duodenum through a fine-tipped Eithorn catheter. For children the dose of male fern extract was reduced to 0.25-0.50 g for each yr of age. Treatment was successful in 97 cases. Mild symptoms of intolerance, such as nausea and vomiting, developed in 3 patients.

5983 **MECHANICAL SUTURING BY MEANS OF A STAPLER IN SURGERY ON THE DIGESTIVE TRACT.** *(Pol.)* Androsov, P. I. (Surg. Clin., Sklifosovskii Inst., Moscow, USSR). *Pol Przegl Chir* 40(10):1101-1113, 1968.

Detailed descriptions are provided for the construction and operation of 5 types of staplers developed in the Soviet Union for surgery on the gastrointestinal tract: the UKZ-8 for suture of the gastric stump following partial gastrectomy, the PKS-25 for esophagoenterostomy following total gastrectomy, the KTs-28 for anastomoses involving the large intestine, the NZKA-60 for side-to-side gastroenterostomy, and the UTL-60 which is a universal stapler for both abdominal surgery and surgery on the soft tissues. Generally excellent results have been obtained with these staplers in 601 patients with various diseases of the digestive tract, including 208 procedures with the UKZ-8 following Hofmeister-Finsterer (157 cases) or Billroth I (51 cases) gastrectomy for peptic ulcer (185 cases) or cancer (23 cases), 35 procedures with the PKS-25 (2 of which were unsuccessful), 43 procedures with the KTs-28, and 315 gastric stump sutures with the UTL-60. It is concluded that the use of mechanical suturing devices significantly shortens the time of the operation, guarantees firm closure of the suture line, and prevents the suture breakdown which accounts for 40-60% of the fatal complications following other procedures.

5984 **RESULTS OF ENZYME THERAPY WITH ENZYMED IN GENERAL PRACTICE.** *(Ger.)* Benzing, H. (Vogelsangweg 16, Ohringen, Germany). *Deutsch Med J* 19(17):611-613, 1968.

Clinical tests of Enzymed were run on 5 groups of patients: 1) 60 with a combination of symptoms referable to the cardia, meteorism, and a feeling of fullness; 2) 90 with gastritis sometimes associated with anorexia and hyper- or hypoacidity; 3) 31 with complaints referable to the liver and bile ducts; 4) 4 with pancreas diseases; and 5) 15 with disturbances in enzyme synthesis following stomach or gallbladder surgery. One tablet of Enzymed (40 mg sodium dehydrocholate, 50 mg pancreatin, 0.8 mg trypsin, 50 mg pepsin, 20 mg ascorbic acid, 1 mg thiamine mononitrate, 2 mg sodium riboflavin-5'-phosphate, 0.5 mg folic acid and 2 mg dried aqueous extract of jambul) was given with each meal. If no improvement occurred the dose was increased to 2 tablets with each meal. Subjective improvements were obtained in all but 9 cases in groups (2) and (3). These patients included a 56-yr-old woman in menopause living under mental stress; a 50-yr-old man later diagnosed as having stomach cancer; and 2 patients who refused to give up drinking.

5985 **SUGAR INTOLERANCE IN MALNOURISHED INFANTS AND CHILDREN.** *(E.)* Chandra,

R. K. (All-India Inst. Med. Sci., New Delhi), R. R. Pawa and O. P. Ghai. *Brit Med J* 4(5631):611-613, 1968.

Half of 100 malnourished children (3 months to 5 yr of age) were found to have fecal pH's below 6. Of these 50, 39 showed lactose intolerance, 16 did not tolerate glucose or galactose well, sucrose intolerance was demonstrated in 25 children, and maltose intolerance in 15. The D-xylose test was abnormal in all 16 children with poor glucose-galactose absorption. Daily fecal fat excretion exceeded 6 g in 7 out of 18 children tested. Nutritional recovery and weight gain were noted after 3 months of dietary therapy. Carbohydrate tolerance tests then showed normal disaccharide absorption in all except 4 patients; 3 of these 4 children showed evidence of a familial lactose intolerance resulting from a primary enzymic defect. The other child had a persistent disaccharide and monosaccharide intolerance, experienced recurrences of diarrhea, and showed poor weight gain.

5986 **DIARRHOEA IN KWASHIORKOR.** *(E.)* Wharton, B. (Makerere Med. Sch. Kampala, Uganda), G. Howells and I. Phillips. *Brit Med J* 4(5631):608-611, 1968.

In a study of 71 children with kwashiorkor, diarrhea was found to be severe in 14, moderate in 30, and mild in 27. Of the 14 with severe diarrhea, 5 were found to have *Salmonella* enteritis complicated by septicemia and intestinal sugar intolerance; 1 patient with moderate diarrhea also had salmonellosis. Three other children had stool pathogens but their diarrhea was very mild and no sugar intolerance was demonstrated; they were regarded as carriers. Sugars were found in the stools of 15 children devoid of intestinal pathogens; 5 had severe, 8 had moderate, and 2 had mild diarrhea. When sugar tolerance tests were performed in 16 of the 21 patients with demonstrable fecal sugars, intolerance to lactose only was found in 5 children; 7 showed intolerance to glucose, galactose, sucrose and lactose; and 4 were intolerant of all sugars tested. The prognosis was most grave in patients with multi-sugar intolerance. The substitution of sucrose or fructose for lactose in the diet of kwashiorkor patients, rather than antibiotic therapy, is recommended to relieve diarrheal symptoms.

5987 **THE EXCRETION OF PORPHYRIN-PEPTIDE CONJUGATES IN PORPHYRIA VARIEGATA.** *(E.)* Rimington, C. (U. Coll. Hosp. Med. Sch., London, England), W. H. Lockwood and R. V. Belcher. *Clin Sci* 35(2):211-247, 1968.

The separation and purification of water-soluble porphyrin X conjugates isolated from the urine and feces of members of a large family (106 members) and several other unrelated patients with variegate porphyria revealed significant variations in molecular size. Conjugates were also isolated from the bile and liver. The porphyrin in plasma, however, is largely bound to protein and unextractable by the usual methods. Analysis showed that porphyrin is bound by thio-ether linkages to cysteine units of the amino acid sequence; these linkages can be split by silver or mercury salts yielding hematoporphyrins. A large, alkali-soluble, acid-insoluble peptidoporphyrin, with an approximate molecular weight of 8000, yielded 15 different amino acids upon hydrolysis. Smaller acid-soluble fractions contained lysine and phenylalanine in proportions of 3:2 and 1:1. Their spectra closely resembled those of simple porphyrins. The amounts of porphyrin X conjugates found in the urine and fecal specimens of afflicted patients varied greatly with the type of porphyria; this may be useful in differential diagnosis. The similarity between the thio-ether linkages of porphyrin and cytochrome c is discussed and a possible connection between porphyrin and cytochrome c metabolism is mentioned.

5988 **A NOTE ON THE DISTRIBUTION OF FAECAL PORPHYRIN X VALUES.** *(E.)* Riley, P. A. (U. Coll. Hosp. Med. Sch., London, England) and R. V. Belcher. *Clin Sci* 35(2):249-250, 1968.

In order to explain the irregular rather than bimodal distribution pattern of fecal porphyrin values in the porphyric family discussed in the previous paper, it is porposed that although the production of porphyrin X is governed by a single dominant gene, its excretion is affected by a different gene, such that individuals with a homozygous recessive complement at this second locus excrete much of the porphyrin X generated while the remainder retain it. Making the assumption that 2 alleles at 2 independent loci are responsible for the effect, and assigning arbitrary values to porphyrin X production and excretion, it is possible to calculate the distribution pattern of porphyrin X output for a large population. Values calculated in this way approximate the distribution of observed values. It also follows from these calculations that 25% of nonporphyric individuals will excrete relatively large amounts of porphyrin and 25% of the affected population will have a low porphyrin output, possibly explaining some of the apparent discrepancies in the transmission of porphyria.

5989 **HYPOTHESES ON THE MIGRATORY PATHWAYS OF ASCARID LARVAE.** *(Ger.)* Matoff, K. (Central Lab. Helminthol., Sofia, Bulgaria). *Z Parasitenk* 31(2):137-154, 1968.

Of 173 mice, 65 were first immunized p.o. once or twice with *Ascaris suum* eggs, 10 with *Toxocaris canis* eggs, and 5 with *A. suum* antigen. The other 93 served as controls. Animals were killed 17-38 hr after challenge or infection and examined for larvae. After washing the abdominal cavity with 10-20 ml of physiological saline only a few larvae were found, but hundreds were present in the intestinal wall and liver. *A. suum* larvae were found primarily in the wall of the cecum and *T. canis* larvae in all parts of the intestine, but particularly in the cecum and colon. Smaller numbers of larvae were found in these organs in mice immunized with *A. suum* antigen. Smaller numbers of larvae were found in the mesentery lymph nodes, the mean number of *T. canis* being about 4 times larger than that of *A. suum.* It is concluded that *T. canis* and *A. suum* migrate primarily through the blood and into the liver via the portal vein. Both species, particularly *T. canis*, also migrate through the blood and lymph to the mesentery lymph nodes.

5990 **RADIOLOGICAL CHANGES IN THE ESOPHAGUS STOMACH, DUODENUM, AND ILEOJEJUNUM IN PATIENTS WITH SCLERODERMA.** *(Por.)* Bettarello, A. (Med. Fac., U. São Paulo, Brazil) and J. C. Oliveira Lins. *Rev Hosp Clin Fac Med S Paulo* 23(3):142-149, 1968.

Radiological studies with $BaSO_4$ contrast were made on 24 patients (4 men and 20 women, including 16 Caucasians, 5 Negroes, and 3 Mongolians) with scleroderma. The diagnosis was based on clinical findings and was confirmed in 10 cases by skin biopsies. The duration of the disease ranged from 6 months to more than 10 yr; the mean duration was 3 yr. In all but one patient the joints, blood vessels, and skin were affected before the digestive tract. Morphological changes in the esophagus included dilatation of the lower 1/3 in 4 of the 24 patients examined, mucosal changes in this region in 2 patients, and annular constriction of the distal extremity in 3. None of these patients had a hiatus hernia or gastroesophageal reflux. Functional disturbances included tertiary waves in 1 patient and delayed esophageal emptying in a standing position in 11. The stomach was hypotonic and flaccid in 16 of the 22 patients examined and the duodenal arch was dilated in 4. Functional disturbances included stasis of the gastric contents in 5 patients, delay in gastric emptying in 10, and slow duodenal filling in 8. Morphological changes in the small intestine included hypotonia of the ileojejunum in 7 out of the 16 patients examined, intestinal dilatation in 2, and poor contrast in 2. Intestinal emptying was delayed in 4 patients. These findings are accounted for on the basis of physiological changes found in scleroderma.

5991 IMMUNOLOGICAL FEATURES OF *SALMONELLA MÜNCHEN* INFECTIONS IN NEWBORNS AND INFANTS.

(It.) Marzetti, G. (1st Pediat. Clin., U. Rome, Italy), G. Turbessi, F. Laurenti and F. Aiuti. *Minerva Pediat* 20(40): 2075-2080, 1968.

A study was made of 19 patients, aged 8 days to 2.5 yr, who contracted salmonellosis while hospitalized for other conditions. Diarrhea was noted in all 19, fever in 18, vomiting in 6, splenomegaly and/or hepatomegaly in 5, and roseola in 1. Chloramphenicol, ampicillin, and colimycin were administered i.v., i.m., or p.o., depending upon the severity of the symptoms. *Salmonella paratyphi* C_2 (*S. münchen*) was isolated repeatedly from the stools of 15 of these patients and from those of 19 other asymptomatic subjects. Serological tests for *S. typhi* and H antigen were positive in 17 out of 19 cases; those for O antigen were negative in all 19. Anti-H agglutinins were found in the IgM fraction in 8 out of 10 newborns and in the IgG fraction in 4. Among the 6 patients, aged 2-6 months, these antibodies were present in the IgG fraction in all and in the IgM fraction in 3. Only 1 of the 3 children aged 6 months or more had anti-H agglutinins in the IgM fraction. The majority of these patients had elevated IgM and IgA levels, while IgM levels were within normal limits.

5992 COPROLOGIC AND SEROLOGICAL FINDINGS IN CHILDREN ADMITTED TO THE DEPARTMENT OF PEDIATRICS OF THE UNIVERSITY OF CAGLIARI FOR ACUTE ENTERITIS AND IN THEIR RELATIONS.

(It.) Bo, G. (Pediat. Clin., U. Cagliari, Italy), W. Tangheroni, R. Corda, V. Badas and V. Cattaneo. *Minerva Pediat* 20(28):1429-1450, 1968.

Laboratory tests for parasites, bacteria, viruses, and serum antibodies were run on 33 children (18 males and 15 females, aged 2 months to 8 yr) with acute enteritis and on 25 of their family members (12 mothers, 13 siblings). Hygienic conditions in the home, diet, and general health were poor in most cases. Enteritis was considered secondary to other infections in 19 patients, including 8 with pharyngitis and tonsillitis, 3 with bronchopneumonia, 2 with bronchitis, 4 with urinary tract infections, and 2 with otitis. Only 3 children from one family had intestinal parasites (*Giardia lamblia*). A total of 567 strains of enterobacteria were isolated from the stools of these 58 subjects (359 from the patients, 208 from their relatives). Among the patients, these included 5 strains of *Salmonella* from 2 patients, 2 strains of *Shigella* from 2 patients, and only 1 strain of *Escherichia coli*; 24 strains of staphylococci were isolated from 24 patients and 18 strains of *Streptococcus* from 18 patients. However, only 9 patients had positive hemagglutinin tests for strains of enterobacteria isolated from their own stools during convalescence. These findings seem to indicate that this gastroenteritis, which is endemic in Sardinia, is caused chiefly by poor diet and resulting disturbances in the intestinal flora. Symptoms are aggravated by delayed hospitalization and by the hot climate.

5993 AMEBIASIS IN NORTHERN SASKATCHEWAN: EPIDEMIOLOGICAL CONSIDERATIONS. *(E.)*

Eaton, R. D. P. (Macdonald College P.O., Quebec, Canada). *Canad Med Ass J* 99(14):706-711, 1968.

Attempts to control infections with *E. histolytica* on the Indian reservations of northwestern Saskatchewan are described. Although many of the reports were questionable many other cases probably went unreported, 110 cases were recorded in a population of approximately 1000 from July 1959 to June 1967, with 8 deaths. The incidence of dysentery was twice as high in females as in males, and 49 of 103 cases occurred in children below the age of 10. Mass medication programs with iodochlorhydroxyquin-phanquone (Mexaform) and diloxanide furoate (Furamide) were carried out in Nov. 1964, April 1965 and Nov.-March 1966-1967. Neither these measures, nor public health education and improvements in housing and sewage disposal, had succeeded in permanently reducing the incidence of infection or controlling clinical disease at the time of this report.

5994 MODERATE GASTRIC HYPOTHERMIA IN THE TREATMENT OF MASSIVE HEMORRHAGES OF THE ESOPHAGOGASTRODUODENAL TRACT.

(It.) Rovati, V. (Gen. Surg. Clin., U. Milan, Italy), E. Mirelli, S. B. Doldi, G. Fichera and M. Galeone. *Arch Ital Chir* 94(1): 103-124, 1968.

The physiological effects of gastric cooling, equipment and techniques used, complications, indications, and published results obtained with this method are discussed. The authors used gastric cooling to arrest severe bleeding in 23 patients with duodenal ulcers (4 cases), hemorrhagic gastritis (6 cases), esophageal varices (3 cases), gastric carcinoma (1 case), postoperative hemorrhages (3 cases), postanastomotic ulcer (2 cases), regional vascular ectasia of the stomach (2 cases), and other causes (2 cases). In all but 2 cases (the patient with gastric carcinoma and one with a duodenal ulcer) cooling was successful in arresting bleeding until specific treatment could be given. Four illustrative case reports are presented.

5995 EFFECT OF A PROTEASE INHIBITOR ON THE FIBRINOLYTIC ACTIVITY IN ACUTE PERITONITIS.

(It.) Rubino, M. (Inst. Gen. Clin. Surg., U. Bari, Italy), M. Conte, A. Brienza and F. Selvaggi. *Rass Int Clin Ter* 48(10):587-596, 1968.

In 26 patients subjected to surgery for acute peritonitis (appendectomy in 13 cases, suture of a perforated duodenal ulcer in 9, gastrectomy for ulcer in 1, suture of resection of the

arge intestine for diverticulitis in 2, and cholecystostomy in
), the serum fibrinolytic activity as measured by the diameter
f the zone of fibrinolysis on a fibrin plate (method of Astrup)
veraged 19.1 mm (range 8.0-38.5 mm) compared to a normal
alue of 5 mm or less. Immediate postoperative treatment
vith the trypsin inhibitor Trasylol at a dose of 200,000 U/day
v. (plus antibiotics and supportive therapy) decreased the av-
rage fibrinolytic activity to 12.3 mm after 2 days and 8.5 mm
fter 7 days. In 10 patients studied after 20 days, the average
ibrinolytic activity was only 5.35 mm. In one patient, who
ied on the 3rd postoperative day, the fibrinolytic activity in-
reased from 14 to 23 mm in 2 days. The clinical course in
he other 25 patients was satisfactory, while in 5 cases of peri-
onitis not treated with Trasylol the fibrinolytic activity and
ever both remained elevated for prolonged periods. Studies
n 4 other controls showed that surgery *per se* did not result
n increased fibrinolytic activity. It is assumed that Trasylol
nhibits the formation of vasoactive kinins which are responsible
or some of the clinical manifestations of endotoxin shock.

996 **THE TREATMENT OF GASTROENTERITIS IN CHILDREN: CLINICAL TRIAL OF A PREPARA-
ION CONTAINING AMINOSIDIN, CHLORAMPHENICOL
ND SULFAGUANIDINE.** *(It.)* Chiossi, F. M. (Giannina
aslini Inst., Genoa, Italy). *Lattante* 39(2):68-81 1968.

abroral Complex was used in the form of an oral suspension
ontaining 25 mg of aminosidin, 25 mg of chloramphenicol ·
innamate, 75 mg of sulfaguanidine, 15 mg of tannalbin and
5 mg of kaolin in 5 ml of syrup in the treatment of 21 chil-
ren (5 yr old or less, including 15 infants below the age of
yr) with gastroenteritis. The dosage varied from 1 teaspoon-
ul every 8 hr to 3 every 6 hr, for periods of from 2 days to
weeks. There were no side effects, and the results were ex-
ellent in 19 of the 21 cases, stool cultures becoming negative
vithin a few days in most. Among 12 cases in which the causa-
ive organism could be isolated, only one strain of pathogenic
taphylococcus and one of *Escherichia coli* 0 119:B 14 were
ound to be resistant to the preparation; other isolated strains
f pathogenic *Staphylococcus, Salmonella typhi murium,*
seudomonas aeruginosa, and *Escherichia coli* 0 125:B 15 and
86:B 7 were sensitive.

997 **TOASTED FULL-FAT SOYA FLOUR IN TREATMENT OF KWASHIORKOR.** *(E.)*
utishauser, I. H. E. (Med. Res. Counc. Infant Malnutr. Res.
Jnit, Kampala, Uganda) and B. A. Wharton. *Arch Dis Child*
3(230):463-467, 1968.

en children (14-34 months old) with mild kwashiorkor were
reated with a diet based on soya flour. Subsequently, 17 out
f 32 children (8-50 months old) admitted with moderate or
evere kwashiorkor were treated in a similar way, while the
emainder were given a diet based on cow's milk. Both diets
vere fed at a level of approximately 4 g protein and 100 cal-
ries/kg daily. Progress was assessed clinically and in terms
f the changes in weight, total serum protein and serum albumin
luring the first 14 days of treatment. Nitrogen balance studies
vere carried out in the children with mild kwashiorkor. In the
nild cases, both diets were well accepted and tolerated. All the
hildren made good clinical progress, although those receiving
oya lost more weight initially and their subsequent rate of
ain was less than in those receiving milk. The increase in
total serum protein and albumin was slightly larger, and nitrogen absorption and retention were consistently higher, in the children receiving milk. In the moderate and severe cases there were greater differences. The soya diet was taken reluctantly by a number of patients, and in them tube-feeding was necessary for a longer period than in those receiving milk. Vomiting was more troublesome in the soya-fed children, but this was reduced by introducing the diet more gradually. Clinical progress in all the milk-fed children was good, but in 4 children treated with soya it was poor. In 3 of these there was an immediate improvement when milk was given. The children receiving soya lost more weight initially and the subsequent rate of gain was considerably less than in those receiving milk. The increase in total serum protein and serum albumin was greater in the milk-fed children. A diet based on milk seems to be the treatment of choice for children with moderate and severe kwashiorkor; though basically more expensive, such diets will prove less costly in terms of length of treatment.

5998 **PATIENT ISOLATOR SYSTEMS IN CANCER CHEMOTHERAPY: THE USE OF HIGH-DOSE "NONABSORBABLE" ANTIBIOTICS IN THE REDUCTION OF BOWEL FLORA.** *(E.)* Hazen, J. G. (Natl. Cancer Inst., Bethesda, Md.), A. A. Levitan and L. D. Bourgeois. *Amer J Gastroent* 50(3):195-201, 1968.

A patient isolator system is described which is designed to protect a highly susceptible subject against both external infection and "autoinfection" from the gastrointestinal tract during cancer chemotherapy. The system consists of a modified hospital bed surrounded by a large plastic canopy and designed to allow sterile air and food to be received by the patient. Before being placed in the isolator, the patient undergoes one week of preparation which consists of antibacterial baths and intensive doses of multiple nonabsorbable antibiotics (bacitracin, kanamycin, neomycin, nystatin, paromomycin, polymyxin, sulfathalidine) which are taken continuously for the duration of the stay within the enclosure. In the 14 patients treated utilizing this system, 60% of the aerobic stool cultures were sterile and similar results were obtained with anaerobic and fungal cultures. Control studies revealed very little absorption of the antibiotics (usually less than 1%), even in patients with chemotherapy-induced oral or gastrointestinal ulceration or radiation-induced enteritis.

5999 **CHANGES IN THE PHAGOCYTIC ACTIVITY OF THE LEUKOCYTES AND IN PROTEIN FRACTIONS IN DYSENTERY PATIENTS TREATED WITH ANTIBIOTICS AND BY A COMBINED METHOD (ANTIBIOTICS, PENTOXYL, AND BACTERIOPHAGE).** *(Rus.)* Sarkisian, S. A. (Yerevan State Med. Inst., USSR). *Zh Eksp Klin Med* 8(2):99-103, 1968.

Studies on 215 patients with acute or chronic dysentery (185 given antibiotics and 30 given symptomatic treatment) demonstrated that therapeutic doses of tetracycline or biomycin had no significant effect on the phagocytic activity of the leukocytes, which increased uniformly during the 4-week observation period regardless of treatment. The phagocytic activity was consistently higher in acute than in chronic dysentery. In a subsequent study, therapeutic doses of tetracycline or biomycin alone were given to 69 patients with acute dysentery and to 115 with chronic dysentery, while a combination of antibiotics with pentoxyl and bacteriophage (administered p.o. or rectally) was used in

24 patients with acute dysentery and 19 with chronic dysentery. Symptomatic treatment was given to 23 controls with chronic dysentery. Antibiotic therapy alone had no appreciable effect on the concentration of total serum proteins or their fractions. Combined therapy with antibiotics, pentoxyl and bacteriophage still had no significant effect on the serum proteins, but in patients with chronic dysentery given combined treatment the serum γ-globulin level was somewhat higher than with antibiotic therapy alone (1.39 g% at the end of 4 weeks compared to 1.31 g% with antibiotics alone and 1.11 g% in controls).

6000 ZOLLINGER-ELLISON SYNDROME: AN ANALYSIS OF TWENTY-FIVE CASES. *(E.)*

Way, L. (U. California Sch. Med., San Francisco), L. Goldman and J. E. Dunphy. *Amer J Surg* 116(2):293-304, 1968.

Multiple endocrine adenomatosis was identified in 12 of 25 patients with the Zollinger-Ellison syndrome (11 men and 14 women, 15-80 yr old). This is twice the incidence seen in Ellison's series. Eleven patients had hyperparathyroidism, 5 had adrenal hyperplasia, 4 had pituitary tumors, 3 had thyroid tumors, 1 had carcinoid tumors, and 2 had hyperinsulinism. It is felt that a patient with hyperparathyroidism and peptic ulcers is likely to have pancreatic tumors, and that all patients with the Zollinger-Ellison syndrome should be investigated for hyperparathyroidism. Symptoms included abdominal pain (24 cases), severe bleeding (10), perforation (9), and watery diarrhea (7). Gastric analysis was diagnostic in 12 cases, and an upper gastrointestinal series was highly suggestive in 14. Diagnosis is often difficult with presently available techniques, but a high basal-to-maximal acid output ratio, a bioassay positive for gastrin, or demonstration of an islet cell tumor are quite reliable. Total gastrectomy was highly successful in the treatment of 10 of these patients; since it is curative and is associated with little nutritional morbidity, total gastrectomy is the only recommended operation except in patients with primary ectopic tumors which can be treated successfully by tumor excision. Results after vagotomy or partial gastrectomy were variable.

6001 DIGESTIVE TRACT TUMORS AND EXTRACELLULAR ALKALOSIS. *(Por.)*

Do Vale, J. C. (Nat. Inst. Cancerol., Rio de Janeiro, Brazil). *Rev Brasil Cancerol* 23(35):7-38, 1967.

The nomogram of Siggaard-Andersen and Engel was used to study the acid-base equilibrium in the blood of 20 patients (11 women and 9 men, aged 36-80 yr) with cancers of the digestive tract. A total of 37 determinations were run. Patients included 6 with epidermoid carcinomas of the esophagus; 3 with adenocarcinomas and 1 with an infiltrating blastoma of the stomach; 3 with adenocarcinomas of the gastric antrum; 1 with a blastoma and 1 with an adenocarcinoma of the head of the pancreas; 1 with a leiomyoma of the small intestine; 1 with an adenocarcinoma of the rectum and one of the hepatic angle of the colon; 1 with an adenocarcinoma of the rectum, one of the stomach and peritoneal metastases; and 1 with an adenocarcinoma of the gallbladder. The results of these 37 determinations showed that respiratory alkalosis was present 12 times, mixed alkalosis 11 times, and metabolic alkalosis 6 times. In 3 determinations it was impossible to distinguish between respiratory alkalosis and metabolic alkalosis. The only patient with metabolic acidosis apparently had a mixed alkalosis. No abnormalities in acid-base equilibrium were found in 4 examinations. Case reports and results of acid-base studies are presented for all 20 patients.

6002 FURTHER EXPERIENCE WITH GENERAL BOD HYPOTHERMIA IN THE TREATMENT OF MASSIVE GASTROINTESTINAL HEMORRHAGE. *(E.)*

Mark, J. B. D. (Stanford U. Sch. Med., Palo Alto, Calif.), W. D. Gaisford, C. M. Knauer and R. A. Chase. *Amer J Surg* 116(2): 286-292, 1968.

Moderate general hypothermia (30-32C) was induced by external cooling under sedation with chlorpromazine and pentobarbital sodium in 23 patients with gastrointestinal and 2 patients with intraperitoneal bleeding. However, an adequate duration of hypothermia for a meaningful trial (at least 12 hr) was achieved in only 20 cases. At least temporary cessation of bleeding occurred in 18 of these, and in 12 bleeding did not recur; 3 of the 6 patients with recurrences and 4 of the 5 who could not be treated adequately died. Bleeding usually stopped within 4 hr, and hypothermia was continued for at least 24 hr more. The only complications were 3 cases of aspiration pneumonitis and one of atrial fibrillation. Experimental studies in 11 dogs revealed a decrease in gastric pepsin during hypothermia but no change in gastric acid secretion or volume output. Detailed studies in 3 patients revealed a 25% decrease in cardiac output and a 35% increase in systemic vascular resistance, without evidence of anaerobic metabolism. General hypothermia may be useful in selected patients with massive gastrointestinal bleeding, particularly if the source of bleeding is unknown or in the presence of cardiopulmonary, renal or musculoskeletal problems which increase the risk of surgery.

6003 ASPECTS OF FAT METABOLISM IN ANOREXIA NERVOSA. *(E.)*

Crisp, A. H. (St. George's Hosp. Med. Sch., London, England), L. M. Blendis and G. L. S. Pawan. *Metabolism* 17(12):1109-1118, 1968.

Serum cholesterol levels and their relation to thyroid function and other aspects of fat metabolism were investigated in 37 female patients (13-33 yr of age) with anorexia nervosa in comparison with 37 normal women without a personal history of thyroid disease or diabetes mellitus. Significantly higher serum cholesterol levels were observed in the anorexia nervosa patients than in controls ($P < 0.002$). Protein-bound iodine levels, basal metabolic rates, 24-hr ^{131}I uptake and response to thyrotropin were used as indices of thyroid function in the patient group. Protein-bound iodine and ^{131}I uptake were independent of serum cholesterol levels; basal metabolic rates returned to normal as serum cholesterol levels rose, and in 3 patients there was a 40-120% rise in ^{131}I uptake 24 hr after thyrotropin stimulation. During a 2-3-month study period in a psychiatric ward, 7 anorexia nervosa patients were given up to 500 mg/day chlorpromazine and a 2500-3000 calorie daily diet; weekly blood samples were collected after an overnight fast, and 24-hr urine collections were made at intervals throughout treatment. Although all patients gained weight, blood lipid values exhibited a characteristic and striking lability and extended over a wide range. Fat-mobilizing-substance (FMS) activity, absent in the urine of 5 patients at the start of the study, appeared transitorily 2-4 weeks after weight gain commenced. It is suggested that anorexia nervosa patients provide a valuable means of investigating some of the aspects of lipid metabolism.

004 **GLUCOSE AND STARCH TOLERANCE AFTER RECOVERY FROM KWASHIORKOR.** *(E.)* ook, G. C. (Dept. Med., Makerere U. Coll., Kampala, Uganda). *letabolism* 17(12):1073-1083, 1968.

oth i.v. and oral glucose tolerance tests and oral starch toler- nce tests were performed on 31 African children (18 males nd 13 females) who had been treated for kwashiorkor 5.2-12.2 r before and 21 children (13 males and 8 females) who did not ave a history of kwashiorkor but who lived in a similar envir- nment. Oral carbohydrate usually produced an early maximum lood glucose level, but the curves were more often relatively at in the post-kwashiorkor group; after oral glucose, 21 had peak at 30 min, 7 at 60 min, and 1 at 90 min, while after ral starch 8, 11 and 1 had maximum levels at these times. In e control group, after oral glucose, 15 had a peak at 30 min, at 90 min and 1 at 120 min; after oral starch 11, 5 and 1 ad maximum levels at these times. There was a significant cor- lation between the individual K values and the ratio of the aximum rise in blood glucose after oral starch to that after ral glucose. The mean K values after i.v. glucose were sig- ficantly lower in the post-kwashiorkor group than in the con- ol group. There was no significant correlation between the dividual K values and body weight in either the post-kwashior- or or the control group. There was a significant inverse cor- lation between the individual K values and the rise in blood ucose 2.5 hr after oral glucose, and the mean rise was sig- ficantly greater in the post-kwashiorkor group than in ontrols. It is concluded that children who have had kwashiorkor, en if adequately treated, probably have a higher incidence of ncreatic disease than those who have not had kwashiorkor.

005 **CLINICOPATHOLOGIC STUDIES ON LARVAL ANISAKIASIS IN JAPAN.** *(E.)* Yokogawa, M. Sch. Med., Chiba U., Japan) and H. Yoshimura. *Amer J Trop Ied* 16(6):723-728, 1967.

he clinicopathological features in 92 Japanese cases of anisak- sis are presented. Threse cases were widely distributed, but ost were found in the coastal areas. There were 65 males nd 24 females (3 unrecorded), and more than 50% of the atients were between 30 and 50 yr old; 7 were less than 20 r old. In 65%, the lesions occurred in the stomach wall; in 0% they were similarly distributed in the small or large bowel. he preoperative diagnosis was gastric tumor in 34 (37%), ap- endicitis or acute abdomen in 15 (16%), ileus or invagination 3, biliary tract disease in 2, ileitis in 2, and tuberculous peri- onitis, pancreatic carcinoma, and diverticulitis in 1 case each. the 25 cases in which stool examinations were made, no elminth eggs were found. Occult blood in the gastric juice r stools was present in 70% of the cases. An average eosinophil ount of 10% was found in 17 patients. Two case reports are resented; in 1 case, an intact *Anisakis*-like larva was found in n eosinophilic phlegmon of the intestine at surgery. Compari- on with *Anisakis* larvae collected from mackerel identified the testinal lesion as such. In Japan, the infection is probably cquired by eating raw marine fish and squid.

006 **THERAPEUTIC TRIALS WITH ORAL DEHYDROEMETINE IN INTESTINAL AMOEBIASIS.** *(E.)* iswanathan, M. (Stanley Med. Coll., Madras, India) and C. V. rishnaswami. *J Indian Med Ass* 51(8):381-383, 1968.

n a group of 50 patients with intestinal amebiasis, 25 were iven 25 mg of dehydroemetine twice a day p.o. and 25 were given 10 mg of the drug three times daily p.o., for 7 days. In the group receiving a total daily dose of 50 mg, 3 patients had to be eliminated from the study due to anemia and severe malnutrition, resulting in inability to complete the full course of treatment. In the remaining 22 patients, the results of treatment, as judged by clinical improvement and negative stool examinations, were: very good, 13; good, 4; fair, 4; and poor, 1. In 2 cases, evidence of secondary bacterial infection necessitated concomitant treatment with antibiotics. Side effects noted were: nausea and vomiting, 5; abdominal distension, 1; increase in number of stools, 2; slight drop in blood pressure, 2; and burning sensation of the face, 1. In the group receiving a total daily dose of 30 mg, the results of treatment were: very good, 21; and good, 4. Side effects were: nausea and vomiting, 4; increase in number of stools, 1; and moderate asthenia, 1. The results indicate that an optimum dosage of the drug is between 30 and 50 mg daily for 7 days. An overall cure rate of 95% was seen in this series.

6007 **TREATMENT OF HYPERCHOLESTEROLEMIA: COMBINED DIETARY, SURGICAL, AND BILE SALT-BINDING RESIN THERAPY.** *(E.)* Buchwald, H. (U. Minnesota Hosps., Minneapolis), R. B. Moore, G. B. Lee, I. D. Frantz Jr. and R. L. Varco. *Arch Surg* 97(2):275-282, 1968.

Serum cholesterol concentration data are presented for 38 patients who underwent partial ileal bypass operations for the treatment of hypercholesteremia. All of these patients were on a low-cholesterol, low-saturated-fat diet for at least 3 months prior to the operation. In 24 patients, the response to dietary management prior to bypass was followed: in 8 cases, diet failed to lower the cholesterol level at all, while the remaining 16 showed an average reduction of 17%. The average diet-induced cholesterol reduction for the entire group was 11%. The same hypocholesteremic diet was continued in all patients after partial ileal bypass, which lowered the serum cholesterol in all cases. The average cholesterol reduction 3 months after ileal bypass was 40% of the post-dietary baseline value, and 61% of the patients had a serum cholesterol level below 200 mg/100 cc at this time. In the 16 patients who responded to diet as well as to bypass, the average total cholesterol reduction was 53%. At least 2 yr after operation, 5 patients were placed on 32 g of cholestyramine daily for 3 weeks while being maintained on the same low-cholesterol, low-saturated-fat diet. Addition of the bile salt-binding resin failed to induce any further lowering of the serum cholesterol values.

6008 **GASTROINTESTINAL HEMORRHAGE CAUSED BY SURGICAL STRESS: PRESENTATION OF FOUR CASES.** *(It.)* Fani, C. (S. Giovanni Hosp., Rome, Italy), F. Puntillo and G. Zeri. *Gazz Int Med Chir* 73(20): 1780-1802, 1968.

Severe, fatal gastrointestinal bleeding occurred 15, 5 and 7 days after prostatectomies performed on 3 men with prostate hypertrophy, aged 63, 72, and 75 yr, resp. Two of the 3 had histories of duodenal ulcers: one had responded to conservative treatment and the other had undergone a gastroenterostomy at age 25 yr. An autopsy performed on 1 of these men showed nonspecific gastroenteritis and membranous colitis. A 47-yr-old woman who had undergone a gastroduodenal resection for duodenal ulcer at age 20 recovered from severe bleeding which developed 8 days after a cholecystectomy. Only mild gastritis, which had been present before surgery, was detected by a radiological examination 33 days after the operation. Severe gastrointestinal bleeding after surgery is considered to be a manifestation of stress in predisposed patients.

6009 INVOLVEMENT OF THE DIGESTIVE TRACT DURING MALIGNANT DISEASES OF THE LYMPHATIC SYSTEM (LYMPHATIC LEUKEMIA AND LYMPHORETICULOSARCOMA). *(Fr.)* Revol, L., J. J. Viala, P. A. Bryon, P. Coeur and P. Noel. *Lyon Med* 220(41): 835-852, 1968.

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6011 IDIOPATHIC "ULCERATIVE" ILEOCOLITIS AND COLITIS: RELATIONS WITH CROHN'S DISEASE AND HEMORRHAGIC RECTOCOLITIS. *(Fr.)* Vachon, A., G. Guillemin, P. Paliard, J. Gauthier, R. Tete, J. Cuilleret, G. Spay, Guillaud, P. Maillet and J. Cuche. *J Med Lyon* 49(1146):1177-1200, 1968.

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6017 MORTALITY DUE TO ACUTE DIARRHEA IN CHILDREN'S HOSPITALS AND REHYDRATION CENTERS. *(Sp.)* Mourigan, H. (Pablo de Maria 1588, Montevideo, Uruguay). *Arch Pediat Uruguary* 39(3):224-236, 1968.

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See also: 5734, 5803

6108 **THE EFFECTIVENESS OF CYCLIC X-RAY THERAPY IN CANCER OF THE ESOPHAGUS.** *(Fr.)* Cukiersztajn, W. (Inst. Oncol., Warsaw, Poland). *J Radiol Electr* 49(5):331-338, 1968.

Results of cyclic X-ray therapy are presented for 153 patients (36 women and 117 men, aged 31-85 yr) with esophageal cancer confirmed by biopsy. Squamous cell carcinomas were found in 104 (68%), solid carcinomas in 31 (20%), and various other tumors in 18 (12%). Therapy was curative in 134 cases and palliative in 19. Doses of 100-150 r exposure were administered over a period of 35-48 days so that total doses ranged from 4180 to 6700 r for curative therapy. Of the 153 patients treated, 118 (77%) died. The mean survival time for patients who died after palliative therapy was 5 months and for those who died after curative therapy, 9.76 months (9.85 months for men, 9.5 months for women). Only 3 patients given curative therapy survived for 5 yr or longer. The mean survival time was about the same for patients with squamous cell and solid carcinomas and shorter for those with other varieties. Mean survival times were significantly longer among patients with tumors 5 cm long or less, those whose symptoms had developed 6 months or less before institution of therapy, those who received doses of 5500 r or less, and those who were treated for more than 55 days. Complications included esophageal fistulas n 14 cases and esophageal edema and dysphagia necessitating a gastrostomy in 26.

6109 **INDICATIONS FOR OROPHARYNGEAL INTUBATION IN CANCER OF THE ESOPHAGUS.** *(Fr.)* Mounier-Kuhn, P., J. Gaillard, J. P. Haguenauer and B. Le Champs. *J Franc Otorhinolaryng* 17(6):491-500, 1968.

During the period 1954-66, metal Souttar tubes were used for he palliation of esophageal neoplasms in 453 patients; since 101 f these had ŕepeated intubation (2-5 times), a total of 618 ubes were inserted. Addition of 56 interventions for the removal of obstructions or for repositioning of a descended tube esults in a total of 674 endoscopic procedures performed durng this period. Examinations performed on 325 patients showed hat the lesions were in the cervical or cervicothoracic region f the esophagus in 85 cases, in the sub- or retroaortic region n 216, and in the subdiaphragmatic region in 23. Esophageal perforation occurred in 14 intubated patients and led to death n 8 (2% of the total). Follow-up studies were possible in 290 patients. The Souttar tube was used for an average of 3.5 months by the 153 patients who were followed during the entire period of their intubation; 58 of these patients later required gastrostomies, and 17 died shortly after surgery. Although none of these patients survived long (only 46% survived 3 months and 5% survived a year or longer), intubation with the Souttar ube is preferable to gastrostomy since it enables the patient to eat comfortably and the risk is slight. A detailed review of the ndications and techniques for palliative therapy in esophageal cancer is included.

6110 **THE LOWER ESOPHAGEAL SPHINCTER IN SCLERODERMA.** *(Por.)* Bettarello, A. (Fac. Med., U. São Paulo, Brazil) and H. W. Pinotti. *Arq Gastroent* 5(3):101-108, 1968.

Electromanometric pressure measurements at rest and during swallowing were made at the level of the lower esophageal sphincter in 18 patients (15 women and 3 men, aged 20-56 yr) with scleroderma and in 10 controls with no gastrointestinal symptoms and negative Guerreiro-Machado reactions. Eleven of the patients with scleroderma had esophageal dysphagia. The maximum pressure of the sphincter at rest was lower in patients with scleroderma than in the controls, but this difference was statistically significant only when values for patients with dysphagia were compared with those of the controls. Achalasia was present in 81.5% of the measurements made on patients with dysphagia and in 51.4% of those on patients without dysphagia. No achalasia was found in any of the measurements made on controls. These changes in pressure at the sphincter in patients with scleroderma are attributed to changes in the musculature at the juncture of the esophagus and stomach.

6111 **THE TECHNIQUE OF PNEUMATIC DILATATION AND RESULTS OBTAINED FROM ITS USE IN MEGAESOPHAGUS.** *(Fr.)* Lequerler, Y. *Vie Med (Spec)* 49:1367-1373, 1968.

A balloon-tipped catheter was used for esophageal dilatation in 55 patients with idiopathic megaesophagus. No premedication or laryngeal anesthesia was used. Pressures of 50-90 mm Hg were used in the first dilatation and 150-180 mm Hg in the second or third. The number of dilatations varied from 2-12 or more. Results were excellent in 5 cases, good in 32, and fair or poor in 10. Two complications occurred. Several hours after dilatation one patient had a hemorrhage which required a blood transfusion. Another patient developed epigastric and dorsal pain, a fever, and a high WBC without signs of emphysema. An esophageal fissure was suspected and the patient was given antibiotics. An operation performed several weeks later showed no signs of inflammation and a Heller esophagogastrostomy was performed without difficulty. Operations were performed on 6 patients who did not improve permanently after repeated esophageal dilatation.

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6117 TWO CASES OF ESOPHAGEAL LEIOMYOMA. *(It.)* Gaetini, A. (Inst. Surg. Path., U. Turin, Italy), M. Dei Poli and L. Piazza. *Minerva Chir* 23(14):753-763, 1968.

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6119 RADIOTHERAPY OF CANCER OF THE ESOPHAGUS. (STUDIES ON TELECOBALT-THERAPY 31ST REPORT). *(JAP.)* Koga, S. (Nagoya U. Sch. Med., Japan). *Nippon Acta Radiol* 28(4):473-478, 1968.

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See also: 5698, 5929, 5957, 5962, 6158, 6253

STOMACH
(Gastric ulcer is placed with peptic ulcer)

6142 **THE SEX RATIO IN GASTRIC CANCER AND HYPOTHETICAL CONSIDERATIONS RELATIVE TO AETIOLOGY.** *(E.)* Griffith, G. W. (Min. Health, Alexander Fleming House, London, England). *Brit J Cancer* 22(2):163-172, 1968.

An analysis of data previously published by Segi and Kurihara on the mean annual mortality from gastric cancer in 5-yr age groups in 24 countries for the period 1958-63, separated according to sex, indicates that the ratio of male to female death rates for gastric cancer varies with age, following the same trend in all countries for which information was available. The mean ratio increased from about 1 in subjects aged 25-29 yr to a maximum of 2.23 at ages 55-59 yr, and then decreased progressively to 1.46 in the oldest age group. It is hypothesized that gastric cancer may be caused by a dietary carcinogen, and that the differences in morbidity between the sexes may be accounted for by the lower total caloric consumption of females. Since adenocarcinoma of the glandular stomach can be produced in rats by feeding contaminated peanuts, it is suggested that the dietary factor producing gastric cancer is the toxic metabolite of a soil fungus which grows on any one of a variety of carbohydrate substrates.

6143 **ANTRO-PYLORIC HYPERTROPHY IN PATIENTS WITH LONGSTANDING GASTRO-ENTEROSTOMIES–A STUDY OF THIRTEEN CASES.** *(E.)* Hajdu, N. (St. George's Hosp., London, England), I. Hyde and V. Riddell. *Brit J Radiol* 41(481):49-54, 1968.

Among 132 patients in whom gastroenterostomies had been performed at least 15 yr before, 2 developed carcinoma of the pyloric antrum and radiological examination revealed 13 cases of benign antropyloric muscular hypertrophy; this incidence of 10% compares to one of 0.32% in a series of 6546 random barium meals. Of the 13 patients (8 men and 5 women, 47-75 yr old, with gastroenterostomies of 15-53 yr duration), at least 10 had originally been treated for peptic ulcers and in 6 the diagnosis of antropyloric hypertrophy was confirmed by subsequent partial gastrectomy; 3 of these 6 specimens also showed fibrosis and inflammation and one more showed only inflammation. The principal symptoms were dyspepsia, melena, hematemesis, and abdominal pain or distension. The radiological picture was characterized by a relatively smooth, concentric narrowing of the pyloric antrum, sometimes proceeding to total occlusion, and accompanied in some cases by bulging of the hypertrophied muscle back towards the body of the stomach. The pathological criteria and possible etiological factors in antropyloric muscular hypertrophy are discussed and 3 illustrative cases are described in detail. Existence of pyloric hypertrophy before the gastroenterostomy is considered unlikely in these patients since the length of the narrowed segment was greater than in the usual type of pyloric hypertrophy and, in 3 cases, the hypertrophy was confined to the pyloric antrum. Based on the frequent finding of inflammation, it is suggested that antral gastritis may play a role, both in the development of postoperative muscular hypertrophy and pyloric stenosis in adults and in the persistence of pyloric hypertrophy in infants treated by gastroenterostomy.

6144 **THE REGENERATIVE CAPACITY OF STOMACH TISSUE IN ACUTE RADIATION SICKNESS.** *(Rus.)* Stankevich, N. V. (Cent. Sci.-Res. Inst. Roentgenol. Radiol., Leningrad, USSR). *Biull Eksp Biol Med* 66(10):80-84, 1968.

In cats suffering from acute radiation sickness of moderate severity (death in 3-30 days) due to whole-body irradiation with 208 r 30-168 hr before, full-thickness sections of stomach wall near the pylorus (2.5 x 3.5 cm in area) were excised and replaced with an omental graft strengthened by a musculotendinous homotransplant. Regeneration of the stomach tissue was then observed histologically during the following 445 days. In both control and irradiated cats, and regardless of the time between irradiation and surgery, the stomach wall was completely regenerated without deformation in the form of a plaqu of connective tissue containing elements of smooth muscle; the gastric mucosa was also completely regenerated, with norma mucus-producing glands. It is concluded that acute radiation sickness has no effect on the regenerative capacity of the stomach.

6145 **GASTRIC ACID PRODUCTION IN MILK ALLERGIES.** *(Ger.)* Werner, M. (District Hosp., Pinneberg, Germany) and L. Wettwer. *Deutsch Med Wschr* 93(49):2372-2376, 1968.

Gastric acid production was studied by continuous intragastric pH measurements with a radiosonde in 10 males and 5 females, aged 22-62 yr, with milk allergies and in 34 nonallergic male controls, aged 17-64 yr. Most subjects in both groups had peptic ulcers or other gastrointestinal disorders. The time required for the pH of the gastric contents to return to its initial value was determined after ingestion of 50 ml cow's milk or 5 ml saturated bicarbonate solution, and the ratio of these time was calculated. This ratio ranged from 2.19 to 6.67 among patients with milk allergies and from 0.45 to 1.91 in patients without such allergies. These findings show that considerably less acid was produced after ingestion of milk than after ingestion of bicarbonate in patients with milk allergies. This may help to explain the malabsorption and allergies to other foods frequently associated with milk allergy.

6146 **STUDY OF CHRONIC AND EXPERIMENTAL GASTRITIS: GASTRIC LESIONS PRODUCED BY EPINEPHRINE AND PILOCARPINE.** *(It.)* Arrigo, F. (Inst Med. Path., U. Messina, Italy) and L. Scaffidi. *Boll Soc Ital Biol Sper* 44(12):989-991, 1968.

The etiology of experimental gastritis was studied by dividing 54 rats and 39 guinea pigs into 3 groups. Animals in the first group were kept as controls, those in the second were given 15 μg/kg epinephrine s.c. twice a day for 30 days, and those in the third were given 0.15 mg/kg pilocarpine s.c. twice a day for 30 days. Histological examinations of the stomach were made 10, 20, and 30 days after the beginning of the experiment Epinephrine produced vascular changes with hyperemia of the mucosa and small subepithelial hemorrhages and glandular chang consisting of hyperplasia and hypertrophy of delomorphous cell Pilocarpine produced trophic changes consisting of a considerabl reduction and degeneration in adelomorphous cells and the cove ing epithelium and of marked edema dissociating the glandular structure.

7 **FECAL MICROFLORA IN PATIENTS WITH GASTRIC RESECTIONS.** *(Rus.)* Mikelsaar, :. (Dept. Microbiol., Tartu State Univ., USSR), A. A. zner, J. J. Sarv and S. R. Veldre. *Uch Zap Tartu Gos Univ led* 18(215):180-187, 1968.

l cultures were taken from 2 groups of patients: 1) 23 ents (17 men and 6 women, aged 24 to 64 yr) with dump- syndrome following Billroth II gastrectomies, and 2) 10 ents (9 men and 1 woman, aged 35 to 62 yr) with gastro- oduodenoplasties performed by a modification of riianov-Zakharov's operation. Reconstructive gastrojejuno- lenoplasty had been performed by Sarv's method on 20 of patients with dumping syndrome. After surgery, patients dumping syndrome had significantly larger numbers of bic bacteria, coliforms, staphylococci, and lactobacilli in : stools than normal controls. There were also significant erences between the ratios of anaerobic to aerobic bacteria, *oli* to lactobacilli, and bacteroids to lactobacilli. These ges in the bacterial composition of the stools persisted after symptoms of dumping syndrome had disappeared, there was a definite relation between changes in bacterial position and changes in the mucosa of the small intestine. ents in group (2) had larger numbers of anaerobic, aerobic, orm bacteria and lactobacilli than controls, but about the e number of staphylococci. It is suggested that changes in erial composition of the stools in patients with dumping lrome are due to a decrease in the excretion of mucoid tances and that this change in bacterial composition pro- es changes in the mucosa of the small intestine.

8 **CORRELATIONS BETWEEN STRUCTURE AND ENZYME ACTIVITIES IN GASTRIC CAR- OMA.** *(Ger.)* Heber, J. (Inst. Path., Karl Marx U., Leipzig, many) and H. Wolff. *Exp Path* 2(5):305-314, 1968.

ological features and enzyme activities were compared in phologically normal and tumor tissue obtained from 20 ents who had undergone gastrectomies for gastric carcinoma. normal tissue was taken from the middle of the stomach. ending upon their structure the tumors were classified as nocarcinomas or solid carcinomas, and significant differences nzymatic activity were found between them. In adenocar- omas the activities of enzymes involved in glycolysis and the tose shunt and malic dehydrogenase were significantly higher n in normal tissue from the stomach wall. In solid carcinomas, the other hand, the activities of all enzymes but pyruvate ase were equal to or lower than those in the normal stomach l. The extractable protein fraction from both types of tumors ; smaller than that from normal tissue. In the extractable tein fraction from adenocarcinomas the activities of all en- nes but GOT and GPT were significantly different from those his fraction from normal tissue. Activities of enzymes in- ved in glycolysis, the citric acid cycle, and the pentose shunt e very much higher. In the extractable protein fraction from d carcinomas only the pyruvate kinase, lactate dehydrogenase, glucose-6-phosphate dehydrogenase activities were signific- ly increased. Since stomach tumors are sometimes difficult classify with histological methods, it is suggested that deter- iation of enzyme activities might be of assistance in disting- ing between the various types.

19 **CLINICAL EXPERIENCE WITH THE USE OF SINULENE IN PEPTIC ULCER AND GASTRODUODENITIS.** *(It.)* Siciliani, M. (Hosp. Incurables, Naples, Italy). *Minerva Chir* 23(15):810-814, 1968.

Five to 6 Sinulene tablets/day (containing 50 mg of the aluminum salt of 18β-olean-3β-ol-11-keto-12-ene-30-carboxylic acid, 400 mg colloidal $Al(OH)_3$ and 0.125 mg belladonna alkaloids/tablet) were given p.o. after meals to 3 groups of patients: 1) 20 patients, aged 18-70 yr, with radiologically confirmed peptic ulcer; 2) 10 patients after stomach surgery who had epigastric pain accompanied by nausea and vomiting; and 3) 40 patients, aged 15-68 yr, with radiologically confirmed acute or chronic gastritis or gastroduodenitis. Sinulene gave good results in 12 ulcer patients and unsatisfactory results in 8. It was more effective in the treatment of stomach ulcers than in duodenal ulcers. Good results were obtained in slightly more than half of the postoperative patients. Among patients with gastritis or gastroduodenitis, good results were obtained in 26 cases and unsatisfactory results in 14, Sinulene proved very effective in the treatment of cortisone- or tetracycline-induced changes of the gastroduodenal mucosa. No side effects or signs of intolerance were seen in any of these 70 patients.

6150 **STUDY OF THE BOAS-OPPLER BACILLUS IN SOME GASTRIC DISEASES.** *(Rus.)* Voronina, M. N. (Dept. Microbiol., Tartu State U., USSR) and A. A. Lenzner. *Uch Zap Tartu Gos Univ Tr Med* 18(215):21-32, 1968.

Gastric juice was obtained under sterile conditions by gastric puncture at surgery or by a special catheter from 80 subjects, including 16 with stomach cancer, 4 with gastric polyposis, 16 with chronic gastritis, 14 with gastric ulcer, 12 with duodenal ulcers, and 18 normal controls. Specimens from 38 subjects (10 with stomach cancer, 15 with chronic gastritis, 3 with peptic ulcers, and 9 controls) yielded 256 *Lactobacillus* strains belonging to 11 different species. Gastric acid concentrations were low or nil in 24 and high or normal in 14. There was no relation between the occurrence of lactobacilli and increased gastric acid secretion or acidity. The species found most often, in both patients and controls, were *L. fermenti* and *L. acidophilus. L. casei* was found significantly less often, and unidentified betabacteria more often, in patients with stomach cancer than in those with chronic gastritis. Gastric juice from patients with polyposis and peptic ulcer had the least abundant and varied flora. Metachromatic granules were found more often in *L. fermenti* from controls than from patients with stomach cancer.

6151 **ULCER SYNDROME WITHOUT ULCERS: CLINICAL AND SECRETORY FINDINGS IN 8 CASES.** *(Por.)* Zaterka, S. (Fac. Med., U. São Paulo, Brazil), A. C. Torres da Costa and A. Bettarello. *Arq Gastroent* 5(3):115-117, 1968.

Radiological examinations of 8 patients (6 males and 2 females, aged 13-53 yr) were negative for ulcers although all complained of recurrent epigastric pain and/or pyrosis; 2 had hematemesis and melena, and 1 had melena. Values for gastric acidity under basal conditions and 30 min after Histalog (betazole) stimulation were comparable to those found in patients with duodenal ulcers. No evidence of peptic ulcers was found during laparotomies performed on 2 patients. Stomach biopsies from 2 patients showed mild superficial gastritis in one and interstitial bleeding in the other. Most of these patients had vagal hypertonicity and 4 of the 6 males had an increase in the population of parietal cells similar to that found in most patients with duodenal ulcers. It is recommended that patients with the ulcer syndrome should be regarded as ulcer-prone, followed carefully, and given the diet and drugs used in the treatment of patients with peptic ulcers.

6152 CLINICAL EXPERIENCE WITH CORRECTIVE SURGERY FOR THE DUMPING SYNDROME.

(E.) Nagel, C. B. (Dept. Surg., U. California, Irvine) and J. M. Farris. *Amer J Surg* 116(2):229-236, 1968.

Corrective surgery was performed in 13 patients with dumping syndromes, all of whom had undergone extensive subtotal gastrectomy of the Billroth II type for duodenal ulcer disease. Postgastrectomy distress had persisted for periods of 3-12 yr prior to reoperation. Time and diet had brought no relief, and all patients were malnourished. Three remedial operations were used: 5 patients were converted from a Billroth II to a Billroth I gastroduodenostomy (group I), 5 patients underwent reversed or antiperistaltic jejunal interposition between the gastric remnant and duodenum (group II), and in 3 patients isoperistaltic jejunal interposition was performed (group III). Truncal vagotomy was added at the time of reconstructive surgery if not previously performed. The results in group I were good in 2, fair in 1, and poor in 2; in group II there was 1 excellent, 2 good, 1 fair, and 1 poor result; and in group III there was 1 excellent result and 2 fair results.

6153 GASTRECTOMY-INDUCED MALABSORPTION AND ITS TREATMENT.

(Rus.) Sitenko, V. M. (S. M. Kirov Acad. Milit. Med., Leningrad, USSR) and V. I. Samokhvalov. *Klin Med (Moskva)* 46(10):111-116, 1968.

Gastrectomy-induced malabsorption was seen in 11 patients (9 men and 2 women, aged 37-61 yr) in whom modifications of the Billroth II procedure had been used to remove 2/3-4/5 of the stomach; 9 of these patients had peptic ulcers and 2 had stomach cancer. The first symptoms of malabsorption developed 6 months to 20 yr after surgery. In addition to typical symptoms, findings included decreased urinary excretion of 17-ketosteroids in 3 cases, paranoid psychosis and suicidal tendencies in 1, and rapid emptying of the stomach in 5 of the 9 studied. None of the 11 had ever had dumping syndrome and only 1 had pancreatic damage due to surgical trauma to the duct of Wirsung which occurred during removal of a duodenal papilla; this patient improved with large doses of pancreatin. Liver biopsies performed in 6 cases showed diffuse lipid deposits in 3, chronic hepatitis in 2, and dystrophic changes in 1. The mucosa of the small intestine was atrophic in 1 of 6 cases and villi were hypertrophic in 3. Since medical treatment of malabsorption proved ineffective, Henley's procedure for reconstructing the duodenum was performed on 5 of these patients. These included 1 in serious condition who died of heart failure 2 days after surgery. Excellent results were obtained in the other 4 cases.

6154 GASTRIC MOTILITY IN CHRONIC CHAGAS' DISEASE.

(Sp.) Freytes, M. A. (Dept. Symptomatol., Natl. U. Córdoba, Argentina) and L. M. Amuchastegui. *Prensa Med Argent* 55(26):1239-1243, 1968.

Recordings were made of gastric motility under basal conditions and 15-25 min after s.c. injection of 5 mg bethanechol chloride in 20 patients (11 men and 9 women, mean age 47 yr) with chronic Chagas' disease and compensated cardiopathies and in 20 normal controls (7 men and 13 women, mean age 51 yr). Gastric secretion was also measured before and after histamine stimulation. Planimetric evaluation of motility measurements showed that bethanechol chloride produced a mean increase of 77% in controls and 132% in patients with Chagas' disease. Under basal conditions type I waves accounted for a mean of 16.77% of total activity in controls and 25.38% in patients; the corresponding values for type II waves were 33.35% and 16.8%, resp. After stimulation, type I waves constituted 9.18% of the activity in controls and 13.91% in patients; the corresponding values for type II waves were 40.25% and 38.12% resp. In 3 patients, gastric activity became completely disordered after administration of bethanechol chloride. Thus, bethanechol chloride produces changes in gastric activity in patients with Chagas' disease which are similar to, but less pronounced than, changes seen in vagotomized patients. This suggests that chronic Chagas' disease produces disorders in the parasympathetic innervation of the stomach. No correlation was found between motility and gastric secretion.

6155 SERUM PEPSINOGEN IN GASTRECTOMIZED PATIENTS.

(Fr.) Chariot, J. *Vie Med (Spec)* 49:1357-1364, 1968.

Serum pepsinogen was determined by Mirsky's method in 51 patients who had undergone gastrectomies (20 total gastrectomies and 31 subtotal in which as much as 2/3 of the stomach was removed) and in 32 controls with endoscopically normal stomachs. Serum pepsinogen was absent in 18 (and very low in 2) patients who had undergone total gastrectomies. In some of these patients it had disappeared completely one week after surgery. The mean serum pepsinogen value for 15 patients wit partial gastrectomies and endoscopically normal anastomotic openings and stumps was lower than that of the controls and significantly lower than the mean for 9 patients with partial gastrectomies and inflammation of the anastomotic opening or gastritis of the stump. Serum pepsinogen levels were very low in 4 patients with partial gastrectomies and atrophic gastr of the stump and much higher than control levels in 3 cases o anastomotic peptic ulcer. These results suggest that serum pe sin activity is gastric in origin, that the serum pepsinogen level is an indication of the number of pepsin-producing cells in the gastric mucosa, and that inflammation produces an increase in the serum pepsinogen level.

6156 5-FLUOROURACIL THERAPY FOR CANCER OF THE STOMACH.

(E.) Abasov, I. T. (Azerbaidzhan Inst. Radiol. Oncol., Baku, USSR) and A. G. Nadzharov. *Oncology* 22(2/3):159-165, 1968.

Improvement in general health, increased performance, relief or cessation of pain, subsidence or cessation of dysphagia, weight stabilization or gain, and decrease in tumor size were evaluated in 54 male and 18 female patients (30-78 yr of age with stomach cancer after 1-3 courses (2-6 months apart) of 5-fluorouracil treatment (12-15 mg/kg i.v. on alternate days; 2.5-7 g/course); palpation and X-ray examination were used to evaluate gross tumor changes. Of 8 patients with recurren or metastatic cancer, no improvement was noted in 3, genera improvement with decreased pain and weight gain was observ in 4, and general improvement with decreased tumor size was seen in 1 patient also receiving 120 mg thiophosphamide and radiotherapy. Of 64 patients with primary cancers, 6 showe only progressive deterioration while 16 showed no changes at all; 24 showed subjective improvement with decreases in pai

l dysphagia and better general health, while both subjective
l objective improvement were noted in the other 18. Re-
sions were generally of short duration, however, and 33
ients received repeated treatment. The frequent passage of
iid stools (30 patients), nausea (7 patients), mild stomatitis
patients), and leukopenia (17 patients) were the only side
ects noted. No serious complications were observed in any
ient. These results show that the drug not only suppresses
nor growth, but may also lead to reversal of tumor develop-
nt.

57 THE DUMPING REACTION STUDIED BY EEG AND RESPIRATORY VARIABLES. *(E.)*

gström, S. G. (Dept. Surg., U. Lund, Malmö, Sweden) and
Bülow. *Scand J Gastroent* 2(4):237-245, 1967.

27 patients (25 men and 2 women, 28-61 yr old) with
nping complaints 1-16 yr after partial gastrectomy (Billroth
n 23 cases) for ulcer disease (19 duodenal, 8 gastric), studies
e made before and during dumping provocation with hyper-
ic glucose solution. The electroencephalogram and respira-
n, including spirography and capnography, were recorded
tinuously and simultaneously, and the state of wakefulness,
athing pattern, and sensitivity to CO_2 were studied and re-
d to individual symptoms. The patients showed a lower
an standard threshold of ventilatory response to CO_2 than
control subjects. Following glucose, a biphasic change in
piration and state of wakefulness was observed. During the
t 20 min, a disturbed irregular ventilatory pattern was present
rked hyperventilation in severe cases), associated with signs
increased wakefulness. This was followed by a decrease in
level of wakefulness. Drowsiness and sleep patterns were
ed in both the electroencephalogram and respiration, cul-
ating 1 hr after glucose intake. It is felt that nervous struc-
es in the brain stem are primarily involved in the dumping
ction.

58 A CLINICAL AND EXPERIMENTAL STUDY OF CORROSIVE BURNS OF THE STOMACH.

Ritter, F. N. (Dept. Otorhinolaryng., U. Michigan, Ann
oor), M. H. Newman and D. E. Newman. *Ann Otol* 77(5):
-842, 1968.

ere stomach burns were sustained by 2 patients (16-yr-old
and 15-month-old infant) who ingested a commercial liquid
ali, Liquid Plumr, containing 30% NaOH. The older patient
s treated successfully by gastric irrigation, but the infant died.
e mechanisms responsible for damage to the gastric mucosa
re then investigated by administering 15-50 cc of the same
stic by gastric tube to 5 randomly selected mongrel dogs
fasted and 2 fed). The initial response was observed fluoro-
pically and the damage was assessed at autopsy 24 hr later.
each case, the caustic caused intense emesis within 20 sec
er initial contact with the stomach. Due to esophageal peri-
lsis, this was followed by a to-and-fro motion of the gastric
itents between the stomach and esophagus, lasting about 5
n and followed by evacuation of the stomach contents into
duodenum. Autopsy findings in the infant and the dogs
re very similar, except that in the dogs the burns were smaller
l were always located in the body of the stomach. The
phageal mucosa showed only superficial ulceration and coag-
tion necrosis, while the gastric mucosa showed a full-thick-
s third-degree burn with submucosal hemorrhage. It is con-
ded that the stomach burn takes place during initial contact with the caustic. Since gastric peristalsis is not affected by this caustic, the best initial treatment in patients who ingest it might be to withhold liquid neutralizing solutions, which seem to induce regurgitation which could lead to further esophageal, laryngeal, and oral cavity damage. Endoscopy should be performed to assess the degree of damage and a plastic tube should be passed into the stomach for aspiration and irrigation.

6159 A HISTO-AUTORADIOGRAPHIC STUDY OF THE DISTRIBUTION AND ABSORPTION OF ^{14}C-ACETYLSALICYLIC ACID IN THE RAT STOMACH.

(E.) Noujaim, A. A. (Sch. Pharm. and Pharm. Sci., Purdue U., Lafayette, Ind.), E. E. Morris, J. E. Christian and T. S. Miya. *Canad J Pharm Sci* 3(3):55-58, 1968.

The mechanism of the production of gastric lesions by acetylsalicylic acid was investigated by administering a 50 mg/kg dose of ^{14}C-labeled acetylsalicylic acid suspension to adult male rats by stomach tube, sacrificing animals at 5, 10, 15, 30, 60, 90, and 120 min, and using histo-autoradiographic techniques to identify the sites of localization and absorption of the labeled drug. A definite difference in the rate of drug absorption in the various parts of the stomach was noted with varying time intervals. No localization of radioactivity or absorption of the drug was observed in the rumen portion of the stomach. Localization of the drug in the submucosal region of the corpus portion of the stomach was noted after 60 min, and could still be observed up to 2 hr after drug administration. These findings tend to refute some of the previous hypotheses in which the production of gastric lesions was attributed to a concentration gradient in specific foci inside the stomach wall; such lesions are probably related more closely to induced biochemical changes rather than to the physical effects of the drug on the stomach wall.

6160 ALTERATIONS DURING THE REPAIR OF INJURY INDUCED BY GASTRIC FREEZING.

(E.) Klavins, J. V. (Brooklyn-Cumberland Med. Ctr., Brooklyn, N. Y.), W. W. Shingleton and R. L. Fuson. *Cryobiology* 4(6): 309-316, 1968.

Functional and anatomical changes during the healing phase after gastric freezing and the comparative effects of different types of instruments used to freeze the stomach were determined in mongrel dogs selected at random. Ten dogs were subjected to gastric freezing with the Swenko instrument, 10 with the Temtrol instrument, and 10 dogs were used as controls. The stomachs were perfused through a rubber balloon with 95% ethanol at inflow temperatures ranging from -10 to -18C and outflow temperatures ranging from -7 to -13C for 50 min with a constant balloon volume of 700 cc. In 4 other dogs, 20 units of pitressin were administered i.v. during the freezing procedure. Before and after gastric freezing, measurements were made of volume, free acidity, pH, and total acidity. In animals sacrificed 2 weeks after gastric freezing, there was a statistically significant decrease in volume, but the total free acid and pH were unchanged. In focal areas the parietal and chief cells had not regenerated. The Temtrol instrument gave the best temperature control and resulted in less damage to the gastric mucosa than with the other instruments. Extremely low freezing temperatures and lower weights of the animals were more frequently associated with the development of gastric ulcers. Pitressin inhibited the incidence of gastric lesions. No lesions were found in dogs sacrificed 4 weeks after gastric freezing. It is suggested that the gastric freezing procedure should be modified to induce more extensive selective parietal cell damage in order to achieve inhibition of gastric acidity.

6161 HISTOLOGIC TYPES OF GASTRIC CARCINOMA IN HIGH- AND LOW-RISK AREAS. *(E.)* Muñoz, N. (Fac. Med., U. del Valle, Cali, Colombia), P. Correa, C. Cuello and E. Duque. *Int J Cancer* 3(6):809-818, 1968.

The histological types of gastric carcinoma found in Cali, Colombia, a region of high risk for this type of cancer, were compared with the types found in Cartagena and Barranquilla, coastal cities of Colombia, and Mexico City, which are all regions of low risk. Among 191 cases of gastric cancer from Cali, 51.8% were of the intestinal type, 34% were of the diffuse type and 14.1% were classified as being of neither the intestinal nor the diffuse type. The ratio of intestinal to diffuse was 1.52. In the 3 low-risk areas, 35.9% of the 131 cases were of the intestinal type, 48.9% were of the diffuse type and 15.2% were classified as "other". The mean ages of the patients with intestinal and diffuse types in Cali were 55.8 and 53.5 yr, resp., which was similar to the age distribution in the low-risk areas. In Cali, the male/female ratio for the intestinal type was 2:7 and that for the diffuse type was 1:4. In the low risk areas, the corresponding ratios were 2:4 and 1:9. The male excess for intestinal type gastric cancer was greater in Cartagena and Barranquilla than in Mexico City. In Cali, the ratio of intestinal to diffuse types increased with age in males but remained stable in females. A similar tendency was observed in the low incidence areas, with the qualification that the data for females under 50 yr suggested a low ratio. A total of 56% of the cases were suitable for evaluation of intestinal metaplasia in the noncancerous mucosa. A higher frequency of intestinal metaplasia was observed in the intestinal type than in the diffuse type in both the high and low incidence areas. These data suggest that the intestinal and diffuse histologic types may be connected with different precancerous lesions, since a significant proportion of the intestinal type carcinomas arose from intestinal metaplasia of the gastric mucosa, while the diffuse type showed no such origin.

6162 CHRONIC GASTRITIS AND AUTOMMUNITY. *(Rev.) (Ger.)* Fixa, B. (1st Med. Clin., U. Hosp. Hamburg-Eppendorf, Germany) and K. Krentz. *Med Klin* 63(27):1061-1067, 1968.

6163 GASTRIC SYPHILIS: REPORT OF A CASE. *(Sp.)* O'Farril, G. (Nov. 20 Hosp. ISSTE, Mexico City) and G. Santoscoy G. *Rev Mex Radiol* 22(2): 85-90, 1968.

6164 INVOLVEMENT OF THE REGIONAL LYMPH NODES IN STOMACH CANCER. *(It.)* Donini, I. (Inst. Gen. Clin. Surg., U. Genoa, Italy) and B. Borreani. *Rass Arch Chir* 1(3):152-156, 1967.

6165 BENIGN TUMORS OF THE STOMACH. *(Sp.)* Vidal-Colomer, E. (Santa Cruz y San Pablo Hosp., Barcelona, Spain). *Rev Esp Enferm Apar Dig* 27(6):817-860, 1968.

6166 THE CONTENT OF FREE AMINO ACIDS IN THE GASTRIC JUICE. *(Rus.)* Shevchenko, I. A. (S. M. Kirov Acad. Milit. Med., Leningrad, USSR). *Lab Delo* (9):557-559, 1968.

6167 PROCEDURE FOR SECURING THE END-TO-END ANASTOMOSIS BETWEEN THE ABDOMINAL ESOPHAGUS AND THE JEUUNUM FOLLOWING TOTAL GASTRECTOMY BY THE METHOD OF ROUX. *(Ge*
Von Elzenbaum, H. (Schlanders Gen. Hosp., South Tyrol, Italy
Zbl Chir 93(35):1193-1197, 1968.

6168 THE TECHNIQUE OF RADICAL SURGERY FOR CARCINOMA OF THE GASTRIC STUMP. *(Ger.)* Kühlmayer, R. (Gen. Polyclin., Vienna, Austria). *Chirurg* 39(11):522-525.

6169 GASTRIC POLYPS-LONG-TERM FOLLOW-UP. REPORT OF TWO CASES. *(E.)* Keller, R. J. (Mount Sinai Hosp., New York City). *J Mount Sinai Hosp NY* 35(6):622-628, 1968.

6170 RADIOLOGICAL NOTES. GASTRIC CARCINOMA: 3½ YEAR FOLLOW-UP. *(E.)* Bloch, C. (Mount Sinai Hosp., New York City) and H. M. Peck. *J Mount Sinai Hosp NY* 35(6):630-634, 1968.

6171 RADIOLOGICAL NOTES. METASTATIC LESIO
TO THE COLON-PRIMARY IN THE STOMACH
(E.) Bloch, C. (Mount Sinai Hosp., New York City) and H. M
Peck. *J Mount Sinai Hosp NY* 35(6):635-638, 1968.

6172 ADULT HYPERTROPHIC PYLORIC STENOSIS
PRESENTATION OF A CASE INCLUDING CINÉGASTROSCOPIC FINDINGS. *(E.)* Meltzer, S. S. (36th Tactical Hosp., Bitburg, Germany), R. R. Price and R. M. Maurer. *Amer J Gastroent* 50(5):382-386, 1968.

6173 THE EXPERIMENTAL STUDIES ON SPHINCT
RESERVING DISTAL GASTRECTOMY. *(E.)* Nakao, Y. (Wakayama Med. Coll., Japan), M. Katsumi, A. Nishigaki, Y. Kobayashi, T. Nakasuji, K. Tsuhada and S. Okamura. *Wakayama Med Rep* 13(10):35-48, 1968.

6174 VARIOUS TYPES OF POUCH REPLACEMENT
FOLLOWING TOTAL GASTRECTOMY: HISTO
ICAL DATA AND CURRENT THOUGHTS REGARDING TOTAL GASTRECTOMY. *(E.)* Herrington, J. L. Jr. (Vande
bilt U. Med. Ctr., Nashville, Tenn). *Amer Surg* 34(12):879-887, 1968.

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181 RETROANASTOMOTIC HERNIA FOLLOWING GASTRIC RESECTION. *(It.)* Leone, A. (Inst. en. Clin. Surg., U. Naples, Italy). *Chir Gastroent* 2(1):58-75, 968.

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6205 TOXIC GASTRITIS AFTER INHALATION OF AMMONIA. *(Fr.)* Dupuy, R. (Beaujon Hosp., Clichy, Paris, France), A. d'Oblonsky and L. Chauveinc. *Arch Franc Mal Appar Dig* 57(10):819-820, 1968.

6206 GASTRIC VOLVULUS AFTER LOBECTOMY. *(Sp.)* Marí Balcells, V. (Red Cross Hosp., Barcelona, Spain). *An Med [Cir]* 54(2):78-87, 1968.

6207 GASTRIC DIVERTICULA. *(Rus.)* Rits, I. A. and E. M. Pishchin. *Vestn Khir Grekov* 101(9): 79-84, 1968.

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6209 NEW EFFECTIVE METHOD FOR LONG-TERM APPLICATION OF CARCINOGENS TO THE GASTRIC MUCOSA OF RATS. *(Rus.)* Arkhipov, G. N. (Inst. Nutrit., Moscow, USSR). *Pat Fiziol Eksp Ter* 12(4):73-77, 1968.

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6211 COW'S MILK IN THE TREATMENT OF CHRONIC GASTRITIS AND GASTRODUODENAL ULCER. *(Ser.)* Kanlić, M. and D. Dukanović. *Med Glas* 22(4):108-111, 1968.

6212 THE EARLY DIAGNOSIS OF STOMACH CANCER. *(Ger.)* Witte, S. (Med. Clin., U. Erlangen, Germany). *Therapiewoche* 18(12):495-497, 1968.

6213 EXPERIMENTAL STUDIES ON THE MECHANIS OF ACTION OF A SEROTONIN ANTAGONIST ON GASTRIC SECRETION. *(It.)* Antonelli, A. (Inst. Gen. Clin. Med., U. Siena, Italy), M. Montagnani, G. Nieri and P. F. Bayeli. *Arch Ital Mal Appar Dig* 34(6):580-587, 1967.

See also: 5735, 5835, 5839, 5852, 5863, 5879, 5883, 5884, 5885, 5894, 5900, 5902, 5903, 5912, 5923, 6147, 5939, 5940, 5942, 5945, 5946, 5962, 6217, 6262, 6264

214 **RESPONSE OF THE CANINE DUODENUM TO INTRALUMINAL CHALLENGE WITH CHOLERA XOTOXIN.** *(E.)* Carpenter, C. C. J. (Johns Hopkins U. Sch. led., Baltimore, Md.) and W. B. Greenough III. *J Clin Invest* 7(12):2600-2607, 1968.

n 10 adult dogs, Thiry-Vella duodenal loops consistently pro- uced isotonic fluid for 24-36 hr after intraluminal challenge ith crude cholera exotoxin. Fluid production usually began ithin 15 min after challenge. The mean bicarbonate concen- ation of this fluid was 24 ± 6 mEq/liter. Perfusion of exo- oxin-treated duodenal loops with an isotonic electrolyte solu- on containing 60 mOsm/liter of glucose caused a significant ecrease in exotoxin-induced isotonic fluid output. The net ffects of glucose on isotonic fluid absorption by perfused uodenal loops were not significantly different before and after dministration of crude cholera exotoxin. The response of anine duodenal loops to challenge by cholera exotoxin differs om the responses of jejunal and ileal loops in the absence of detectable lag period between exotoxin administration and itiation of net fluid output, a longer period of fluid produc- ion after exotoxin administration, and a significantly greater et fluid output per unit length of gut. The mean bicarbonate oncentration of fluid produced by the duodenum is less than hat produced by the ileum, but is not significantly different om that produced by the jejunum. The duodenal response is imilar to that of the more distal small bowel segments because n effect on isotonic fluid movement is seen shortly after exo- oxin administration and the maximum rate of exotoxin-induced sotonic fluid production is not reached until 4-5 hr after exo- oxin administration. The reason for a consistent delay of 4-5 r between intraluminal exotoxin administration and maximum ut fluid production has not yet been determined. These find- ngs are consistent with the hypothesis that the rate of secre- ion of isotonic fluid induced by cholera exotoxin is not sig- ificantly different per unit length in the duodenum and ileum nd that the lower *net* fluid output in the ileum is due to its reater capacity for isotonic fluid absorption.

215 **THE EFFECT OF X-RAY ON THE ULTRASTRUCTURE OF THE IMMUNO- OGICALLY ACTIVE CELLS OF MOUSE INTESTINAL UBMUCOSA.** *(Fr.)* Deby, C. (U. Hosp. Ctr., Algiers, lgeria) and K. Malkani. *Path Biol* 16(15-16/17-18):739- 49, 1968.

The submicroscopic changes produced in duodenal macrophages, ymphocytes and plasmocytes of Paris II albino mice (40) y total-body irradiation with a single dose of 800 r were tudied. Radiation was administered at a distance of 50 cm nd a rate of 45 r/min. Duodenal tissue was fixed with smic acid following prefixation *in situ* with dilute formal- lehyde at intervals of 1-7 days after irradiation. Ultra- nicrotome sections were prepared from alcohol dehydrated issue embedded in Epon 812 and Araldite Cy212 and tained with a saturated solution of uranyl acetate (5 hr) nd lead citrate (10 min). In comparison to nonirradiated ontrols (10), there was an increase in the number and the hagocytic activity of macrophages in the first 24 hr after rradiation, followed, in 48 hr, by the complete disappear- ance of cells with phagocytosed material. Lymphocytes showed massive destruction in 6 hr and disappeared completely in 24 hr. Plasmocytes showed progressive degeneration for 5 days, beginning with destruction of mitochondria at 48 hr. Replacement of mature cells began on day 5 and continued until the termination of the experiment on day 7. Control animals (40) irradiated in the same manner and allowed to live showed 50% mortality after 8 days, 100% mortality after 12 days.

6216 **THE ENTEROPATHY ASSOCIATED WITH HERPETIFORM DERMATITIS.** *(Sp.)* Schaposnik, F. (Fac. Med., La Plata Natl. U., Argentina), R. Echeverría, R. Laguens, A. Conti and R. Calafell. *Prensa Med Argent* 55(26):1235-1238, 1968.

Reports are presented on 3 patients (38, 52, and 74 yr of age) with herpetiform dermatitis and gastrointestinal symptoms. In 1 case a jejunal biopsy showed a reduction in the height of the villi, an increase in the number of goblet cells, and the absence of Paneth's cells; in another case there was massive atrophy of the villi and flattening of the mucosal relief. The only abnormality noted in the third case was lymphocytic infiltration of the epithelium. Fecal fat excretion was elevated in the first 2 cases. A gluten-free diet, prescribed in 1 case, produced both a remission in the dermatitis and an improvement in the appearance of the intestinal mucosa.

6217 **ROENTGENOLOGICAL TRANSIT TIME THROUGH THE SMALL INTESTINE IN THE IMMEDIATE POSTOPERATIVE PERIOD.** *(E.)* Noer, T. (Ulleval Hosp., Oslo, Norway). *Acta Chir Scand* 134(7):577-580, 1968.

The passage of radiopaque material from the duodenum to the cecum was followed postoperatively in 45 patients having had either gallbladder or gastric operations. The patients were divided into 4 groups: Group A (13 patients) in which barium sulfate suspension (Mixobar) was injected at the end of the operation; Group B (13 patients) in which barium was injected 19-25 hr postoperatively; Group C (8 patients) in which barium was injected 42-46 hr postoperatively; and Group D (11 patients) in which diluted Gastrografin (1 volume Gastrografin:4 volumes tap water) was injected at the end of the operation as in Group A. The contrast medium reached the cecum within 26 to greater than 46 hr in Group A, within 10 to greater than 24 hr in Group B, in less than 13 hr in Group C, and within 29-44.35 hr in Group D. Of the 9 patients in which transit time was abnormally slow, the delay occurred in the distal ileum or at the ileocecal junction in 5, in the jejunum in 3, and along the whole length of the samll intestine in 1. There seemed to be no substantial difference in transit times between gallbladder and gastric operations, although the latter generally lasted almost twice as long as the former. There was also no marked difference in the transit times of Groups A and D, despite the physical characteristics of the barium sulfate suspension as compared to Gastrografin.

6218 **CYTOKINETICS OF THE KREBS 2 CARCINOMA. I. ITS EFFECT ON THE CELL POPULATION KINETICS OF DUODENAL CRYPT PROLIFERATION.** *(E.)* Schmeer, A. C. (U. Colorado Med. Ctr., Denver). *Int J Cancer* 3(6):829-837, 1968.

The effect of the Krebs 2 transplantable solid carcinoma on the cell cycle of the duodenal cryptal progenitor population as determined by ^{3}H-thymidine uptake and radioautography, was studied in 184 female CFl mice, 5-7 weeks old. Each animal received approximately 3 x 10^{7} K2 ascites cells s.c. in the right axillary region. Four days after implantation, when a palpable solid tumor with a diameter of 5-8 mm was evident in 100% of these animals, a single pulse label of tritiated thymidine (specific activity 20,000 μC/mM, methyl labelled, diluted to a concentration of 18 μC/per animal) was injected. The mice were then serially killed from 15 min to 24 hr later and data were recorded in the form of a curve describing the rate of appearance and disappearance of labelled mitoses in the tumor and duodenal crypt cells. The turnover rate of the cryptal progenitor population was found to be essentially the same in tumor-bearing mice and controls. Thus, the mean DNA synthetic index was 37.0 and 38.8%, resp., the mean mitotic index was 5.7 and 5.5%, resp., and the estimated time between 2 waves of mitoses was 14.0 and 14.5 hr, resp. In the tumor itself, on the other hand, these 3 parameters were 49.4%, 4.2% and 12.0 hr, resp.

6219 **EXPERIMENTAL BLIND LOOP SYNDROME IN ALBINO RATS: A PRELIMINARY COMMUNICATION ON SURGICAL TECHNIQUE.** *(E.)* Kumar, D. (J. Nehru Med. Coll., Aligarh Muslim U., India) and M. C. Gupta. *J Indian Med Ass* 51(8):381-386, 1968.

Self-filling, peristaltic blind loops were produced in the small intestines of 25 fasting albino rats anesthetized with pentobarbitone i.p. and subsequent ether inhalation. Ten animals were maintained as controls. The operative technique is described in detail. Twenty rats (80%) have survived for more than 30 weeks, while 2 died during the operation, 2 on the first postoperative day, and 1 on the second postoperative day. Milk feeding for 2 days preoperatively and overnight fasting aided in preventing spillage of fecal matter into the peritoneal cavity. This experimental preparation is intended for use in studies of the anemia and steatorrhea often following gastrointestinal surgery.

6220 **SMALL BOWEL IN HYPOPROTEINEMIC STATES.** *(E.)* Chuttani, H. K. (Maulana Azad Med. Coll., New Delhi, India), M. L. Mehra and R. C. Misra. *Scand J Gastroent* 3(5):529-536, 1968.

The effect of protein deficiency on the structure and function of the small bowel mucosa was studied in 18 patients with primary malnutrition (13-80 yr of age), 10 patients with cirrhosis (13-60 yr of age), 17 patients with nephrotic syndrome (16-50 yr of age), and 20 normal controls. Total serum protein levels in all 45 patients were less than 6 g/100 ml. Fecal fat determinations, the D-xylose test, and gross and microscopic examination of the jejunal mucosa were performed in all patients Mild steatorrhea was found in 4 cases of malnutrition and 2 cases of cirrhosis; fecal fat was normal in the nephrotic syndrome. Urinary excretion of D-xylose was abnormally low (less than 1.0 g/24 hr) in 9 patients with malnutrition and 6 with cirrhosis. Mild to moderate abnormalities in jejunal mucosal morphology were found in about 67% of the 38 patients in which specimens could be evaluated, as well as in 3 of the 20 controls. All patients were found to be anemic, although the degree of anemia was greater in primary malnutrition than in cirrhosis or nephrotic syndrome. A megaloblastic bone marrow was found in 1 case of malnutrition and 1 case of cirrhosis. Bulky, frothy stools were often observed in the malnutrition patients. *Entamoeba histolytica* cysts were observed in the stools of 1 nephrotic syndrome patient and 1 malnutrition patient. No other parasites were found, and all stool cultures were negative.

6221 **MEGADUODENUM: REPORT OF AN UNUSUAL CASE.** *(Sp.)* Fernández-Nogués, F. (Fac. Med., Barcelona, Spain), J. Garcés Brusés, J. Condomines Pereña and R. Nolla Panadés. *Med Clin (Barcelona)* 51(1):72-81, 1968.

6222 **PRIMARY CARCINOMA OF THE DUODENUM: REPORT OF TWO CASES.** *(Ger.)* Kramer, A. (Elisabethen Home, Darmstadt, Germany). *Z Gastroent* 6(4):266-270, 1968.

6223 **PRIMARY TUMORS OF THE SMALL INTESTINE.** *(E.)* Thomas, D. (Alfred Hosp., Melbourne, Australia). *Ans N Zeal J Surg* 37(4):359-362, 1968.

6224 **GRANULAR CELL MYOBLASTOMA OF DUODENUM. A REVIEW OF LITERATURE AND CASE REPORT.** *(E.)* Lee, B. Y. (VA Hosp., Castle Point, N. Y.) and J. L. Madden. *Amer J Gastroent* 50(5):397-401, 1968.

6225 **LEIOMYOSARCOMA OF THE DUODENUM. A CASE REPORT.** *(E.)* Litvin, L. E. (Cobb Med. Ctr., Seattle, Wash.). *Northwest Med* 67(12):1193-1195, 1968.

6226 **VOLVULUS OF INTESTINE. A CLINICAL STUDY.** *(E.)* Jain, B. L. (S. S. Med. Coll., Rewa, India) and K. K. Seth. *Indian J Surg* 30(3):239-246, 1968.

6227 **DUODENAL CARCINOMA. [CASE REPORT].** *(Ger.)* Maurer, P. (Surg. Clin., Tech. Hochschule, Munich, Germany). *Chirurg* 39(11):527-528, 1968.

6228 **EXPERIMENTAL STUDIES ON SUGAR METABOLISM IN ILEUS - ESPECIALLY ON THE BEHAVIOR OF ENZYMES OF GLUCOLYTIC SERIES IN THE LIVER.** *(E.)* Ogasawara, E. (Sch. Med., Tokushim U., Japan). *Shikoku Acta Med* 23(5):445-462, 1967.

6229 **HODGKIN'S DISEASE OF THE SMALL INTESTINE.** *(Fr.)* Warter, J. (U. Hosp. Ctr., Strasbourg, France), A. Sibilly and B. Hammann. *Strasbourg Med* 19(4):371-383, 1968.

6230 **REPORT OF A PATIENT WITH A RELATIVE LARGE DIVERTICULUM IN THE REGION OF THE DUODENOJEJUNAL FLEXURE.** *(Jap.)* Umeda, K. (Tokyo Med. Coll., Japan), S. Akiya, S. Yamamoto, Y. Hirabayashi, S. Hasegawa and S. Katsuta. *J Tokyo Med Co* 26(3/4):487-490, 1968.

6231 **SUB-MESOCOLIC TRANSPOSITION OF THE EFFERENT DUODENAL LOOP IN THE DUMPING SYNDROME.** *(Fr.)* Patel, J. C. *Presse Med* 76(38): 1829, 1968.

232 **OBSTRUCTIONS DUE TO DUODENAL AND PANCREATIC MALFORMATIONS.** *(Fr.)* arlioz, H., J. Gubler, J. Borde and P. Petit. *Ann Chir fant* 9(2):111-123, 1968.

233 **HEMANGIOPERICYTOMAS OF THE SMALL INTESTINE: ONE CASE.** *(Fr.)* Veyssiére, C. (New Hosp., Amiens, France), A. Smadja, P. Vives and J. Baillet. *Arch Franc Mal Appar Dig* 57(10):801-808, 1968.

6234 **DUODENAL SARCOMAS: A CLINICAL, ANATOMICAL AND RADIOLOGICAL STUDY.** *(Fr.)* Varay, A. (Beaujon Hosp., Paris, France), A. Hauert, P. Vigneron, M. Méot and M. P. Lefebvre. *Arch Franc Mal Appar Dig* 57(10):769-788, 1968.

See also: 5789, 5851, 5853, 5887, 5930, 5963, 6147

6235 ANTISECRETORY EFFECTS OF LIBRAX IN PATIENTS WITH DUODENAL ULCER. *(E.)* Head, H. B. (Northwestern U. Med. Sch., Chicago, Ill.) and J. B. Hammond. *Amer J Dig Dis* 13(6):540-550, 1968.

Fasting gastric secretion before and after administration of 10 mg chlordiazepoxide hydrochloride and 5 mg clidinium bromide (Librax) or a placebo was determined in 20 patients with radiologic evidence of a deformed duodenal bulb and basal gastric secretion with a pH consistently below 4.0. Significant reductions occurred after Librax in mean hourly pH, mean hourly secretory volume, hourly total acid output, hourly volume (percentage of control), and hourly acid output (percentage of control), while after placebo all parameters increased. The results indicate that the antisecretory effect was due to a reduction in both volume and acid concentration. The reduced acid concentration was reflected by the change in pH; titratable acidity was changed only slightly but in the same direction. The antisecretory effects, which were of the same magnitude in the 10 subjects with the lowest basal gastric secretion and in the 10 with the highest, are attributed to clidinium bromide, an anticholinergic agent. There were no side effects.

6236 THE EFFECT OF UNSATURATED FATTY ACIDS IN FOOD PRODUCTS ON GASTRIC ACIDITY, GASTRIC MOTILITY, AND BLOOD LIPIDS. *(Ger.)* Keel, H. J. (Grad. Hosp., U. Pennsylvania, Philadelphia), M. L. Goldstein, W. S. Haubrich and J. L. A. Roth. *Schweiz Med Wschr* 98(8):288-289, 1968.

Studies were run on 30 patients with duodenal ulcers before and after hourly ingestion of 4 oz whole milk (ratio of unsaturated to saturated fatty acids 0.06:1) and 4 oz of Mead Johnson 7216 (ratio of unsaturated to saturated fatty acids 9:1). A significantly longer time was required to neutralize stomach acid and for the stomach to empty after ingestion of the test product. This slower emptying, combined with the product's higher protein content and greater buffering capacity, accounts for more effective neutralization of the stomach contents. Delayed emptying of the stomach is ascribed to the product's high fat content and high osmolarity. The test product was substituted for whole milk in the Sippy diet used to treat 12 out of 35 patients with acute ulcers. Serum cholesterol increased 11.1% in the first 10 weeks among patients receiving whole milk and decreased 11.6% in the first 2 weeks among those receiving the test product. However, substitution of whole milk for 4 weeks after 10 weeks on the test product produced an average increase in the cholesterol levels of 19.6% over those at the beginning of therapy. The same effect was seen, but to a lesser extent, in serum levels of total lipids, free fatty acids, phospholipids, and triglycerides.

6237 CLINICAL STUDIES ON THE TREATMENT OF PEPTIC ULCER. PART 2. THE AMBULATORY TREATMENT OF DUODENAL ULCER WITH BISMUTH SUBNITRATE. *(Ger.)* Berndt, H. (Inst. Cancer Res., German Acad. Sci., Berlin, Germany), H. J. Gütz, K. H. Jacobasch, B. Prahl and G. Wolff. *Deutsch Gesundh* 23(20):913-916, 1968.

In a controlled clinical trial of bismuth subnitrate ($BiOH(NO_3)_2$), the subjective and radiological response to ambulatory treatment was compared in 2 groups of 24 duodenal ulcer patients (average age 45.1 yr) who were given either $CaCO_3 + MgO_2$ (80:15) or $CaCO_3 + MgO_2 + BiOH(NO_3)_2$ (40:15:40) at a dosage of 1 tablespoon daily (suspended in water and drunk *ad lib.* during the day); both groups were told to stay home from work and to avoid smoking, spices, alcohol, coffee and difficultly digestible foods, but were not put on a strict diet. Pretreatment biopsy findings in the 2 groups were similar, with gastritis being noted in 10 of 16 control patients and 10 of 12 given bismuth subnitrate. Weekly questionnaires revealed essentially the same subjective response in both groups, with improvement at the end of 3 weeks in 19 of 24 controls and 18 of 21 on bismuth; 6 of 19 and 7 of 18 were entirely free of pain. Radiological follow-up after 20-24 days showed disappearance of the ulcer in 13 of 24 and 10 of 21 and a decrease in ulcer size in 6 of 24 and 7 of 21. It is concluded that there was no significant difference between the results in the 2 groups and that the addition of bismuth subnitrate to the conventional antacids is unnecessary.

6238 PEPTIC ULCERS AMONG RAILROAD PERSONNEL. *(Fr.)* van Roy, R. *Arch Belg Med Soc* 26(5):321-330, 1968.

Statistics on the percentage of absenteeism due to peptic ulcers were compared among the approximately 2,300,000 railway workers in 19 European countries. It was highest among crews of locomotives followed by train crews and office workers. The percentage of workers unable to go back to their old jobs range from 2.6% in Belgium to 25% in the Netherlands. The percentage of patients receiving surgical treatment varied from 12% in Belgium to 75% in Luxemburg. In the author's experience, some patients developed low blood pressure shortly before recurrences. In addition to predisposition and environmental factors, a minor role is played by irregular hours, overwork, vibration of the locomotives, rapid eating of meals, poor eating habits, and excessive cigarette smoking. In Belgium the replacement of steam locomotives had no effect on the incidence of ulcers among railroad personnel.

6239 PERSONAL EXPERIENCE WITH VAGOTOMY AND PYLOROPLASTY FOR PERFORATED DUODENAL ULCERS. *(Fr.)* Stoppa, R. (Fac. Med., Amiens, France), M. Quintyn, H. Abourachid, K. Henry and C. Duclaye. *Lille Chir* 23(1/2):29-36, 1968.

A group of 42 patients with perforated duodenal ulcers was treated by pyloroplasty and vagotomy. Of these patients, 4 were very obese, 3 had diabetes, 3 hepatosplenomegaly, 4 hepatic cirrhosis, and 1 pulmonary tuberculosis. Six were considere to be high-risk cases. Surgical findings in 32 cases showed 22 perforated ulcers in the center of the anterior side of the duodenal bulb, 6 ulcers more than 3 mm in diameter and requiring resection of more than 2/3 of the bulb, 1 large perforation at the base of the bulb, 2 stenosing ulcers with periduodenal lesio and 1 recurrent bulbar perforation. Ulcers of the lesser curvat of the stomach were found in 2 cases. Histological examinatio of resected material showed aberrant pancreatic islets near the ulcer in 2 cases. The only postoperative complications were a residual Douglas abscess evacuated by rectotomy and 1 moderately severe and 2 mild parietal infections. Only 4 of 12 ltures of peritoneal effusions were positive. Only 22 of these tients could be followed for more than 18 months. Clinical sults were good or very good in 17, fairly good or acceptable 4, and poor in 1. Radiological follow-up showed no evidence ' recurrences. The 2 stomach ulcers found associated with rforated duodenal ulcers had healed 3 months after surgery.

240 GASTRIC SECRETION IN FAMILIAL AND NONFAMILIAL ULCERS.

(Fr.) Bettarello, A. 'ac. Med., U. São Paulo, Brazil) and S. Zaterka. *Arq astroenterol* 5(2):47-50, 1968.

modification of Kay's histamine loading test was performed 9 members of a single family with an abnormally high in- dence of duodenal ulcers, 10 patients with uncomplicated, onfamilial duodenal ulcers, and 5 controls. Student's *t* test owed no significant differences in the maximum hourly secre- on between patients with familial and nonfamilial ulcers. How- er, patients with familial ulcers secreted much more HCl under asal conditions and secreted a much higher percentage of their aximal capacity under basal conditions than did patients with onfamilial ulcers. These findings suggest that patients with milial duodenal ulcers have a high basal secretion of gastrin d vagal hypertonicity. It is unlikely that the patients with milial ulcers had Zollinger-Ellison syndrome since they im- oved after surgery.

241 A CONTRIBUTION TO THE STUDY OF GASTROINTESTINAL HEMORRHAGE DUE TO LCERS: STATISTICS ON 123 OUT OF A TOTAL OF 475 ASES OF PEPTIC ULCER.

(Fr.) Fraisse, H. (St. Etienne osp. Ctr., Lyon, France), J. P. Etaix and N. Livoireau. *J Med yon* 49(1149):1339-1366, 1968.

f 473 patients with peptic ulcers, 123 (84 men, 39 women) ad hemorrhages severe enough to require hospitalization. hese patients accounted for 24.3% of all patients admitted for astrointestinal hemorrhages. In the 120 cases in which the xact site of the ulcer was known, the incidence of hemorrhages as highest among women with stomach ulcers (47.27%) followed y men with stomach ulcers (34.6%), women with duodenal ul- ers (21.8%), and men with duodenal ulcers (17.33%). Hemor- hages were particularly frequent among patients with postbulbar lcers (4 out of 10) and those with multiple duodenal ulcers 5 out of 9). Of the 123 patients with peptic ulcers and hemor- hages, 81.2% were over 40 yr and 56.8% ranged from 60-80 yr. he 12 patients with familial ulcers made up less than 10% of hese patients, and they had fewer severe hemorrhages than av- rage. Only 9% of North African Muslims developed hemor- hages; this is attributed to their abstinence from alcohol. Alco- olism was definitely implicated in 41 patients, 16 of whom had irrhosis. Drugs, most often aspirin, were considered to be the ause of bleeding in 21 cases. Seasonal peaks in gastrointestinal emorrhages occurred in August and November, but there was o significant seasonal variation. In the 110 cases in which the everity of bleeding was evaluated, hemorrhages were judged to e severe in 40%, moderate in 37.2%, and mild in 22.7%. The ncidence of severe hemorrhages was especially high among wo- nen, patients over 40 yr, and alcoholics. Bleeding occurred at he onset of ulcer development in 33.3% and during the first ear after appearance of symptoms in 61.6%. Recurrent bleed- ng accounted for 33 of the cases and was particularly frequent among alcoholics and those over 40 yr; 6 of these patients died. Medical treatment was used on 107 patients, 13 of whom died; 25.9% of these patients had recurrent hemorrhages. Surgery was performed on 38 of these 123 patients, usually 3-4 weeks after the hemorrhage. Only 1 patient died and none of the survivors had recurrences of bleeding or severe functional disorders. Emergency surgery was performed on 16 patients, 2 of whom died. None of the survivors had recurrences of bleeding, but functional disorders, particularly the dumping syndrome, were severe and persistent in a relatively large percentage of cases.

6242 A STATISTICAL STUDY OF THE FACTORS INFLUENCING THE ASSOCIATION OF CHRONIC LUNG DISEASE AND PEPTIC ULCER.

(It.) Grassi, V. (Inst. Spec. Med. Path., U. Perugia, Italy), G. Fornari, N. Marchesi and G. Duranti. *Rass Fiziopat Clin Ter* 40(2):160-174, 1968.

Of 455 patients with chronic lung disease, (11.9% with stomach ulcers, 7.2% with duodenal ulcers) 19.1% were found to have peptic ulcers; this included 17.1% among 308 with only moderate hypoxemia and 23.3% among 146 with severe hypoxemia and hypercapnia. Results of the ^{2}-test show that this difference is not significant. However, the incidence of peptic ulcers was significantly higher among patients who had chronic lung disease for more than 10 yr and among males (22.6% vs 7.5% for females). The incidence of peptic ulcers was also significantly higher among patients with severe lung disease who had polycythemia. Medications used in the treatment of lung disease had no appreciable effect on the incidence of peptic ulcers. It is concluded that the association of peptic ulcer and pulmonary disease may result from the common embryologic origin of the lung and gastrointestinal tract, their common control by the autonomic nervous system, the many external factors (such as smoking) to which both areas are exposed, and the long-term effects of pulmonary emphysema and hypoxia on gastric secretion and the resistance of the gastroduodenal mucosa.

6243 GASTROINTESTINAL ULCERATION AND HEMORRHAGE ASSOCIATED WITH SEVERE BRAIN INJURY.

(E.) Heiskanen, O. (Neurosurg. Clin., U. Cent. Hosp., Helsinki, Finland) and T. Törmä. *Acta Chir Scand* 134(7):562-566, 1968.

Among 960 patients treated for severe head injuries during the period 1960-66, 11 (10 adult males, 16-59 yr old, and a 4-yr-old girl) developed signs of gastrointestinal ulceration and/or bleeding while they were still alive and unconscious. The group included 8 cases of cerebral contusion, 2 of extradural hematoma and one of compound skull fracture, and 2 cases were complicated by shock. Hemorrhage was noted 3-220 days after the injury (3-14 days in 8 of 11), and unconsciousness lasted for from 11 days to over 1.5 years. Four of the 8 patients treated conservatively and 1 of 3 treated surgically died 2-64 days after the onset of bleeding, but death was ascribed to the bleeding in only 1 case (perforated duodenal ulcer). Of the 6 survivors, 3 were totally disabled, 1 was partially disabled, and 2 returned to work. The results of autopsy studies in 4 cases showed that no particular area of the brain can be implicated in the production of gastroduodenal ulcers. Since the eventual degree of recovery may be surprisingly good in these cases, an active approach to their treatment is recommended, especially in young patients.

6244 **CONTRIBUTION TO THE STUDY OF GASTRIC MOTILITY IN PATIENTS WITH GASTRODUODENAL ULCERS.** *(Cz.)* Jablonská, M. (Fac. Gen. Med., Charles U., Prague, Czechoslovakia), E. Součková, V. Skop and L. Stamidis. *Sborn Lek* 70(10):293-300, 1968.

Radiological studies of antral peristalsis in 100 patients (63 men and 37 women) with peptic ulcers (52 duodenal, 33 mediogastric, 11 at other sites on the stomach, and 4 with both duodenal and gastric ulcers) revealed statistically significant differences only between males with mediogastric or duodenal ulcers. The average height of the X-ray polygraph wave varied from 2 to 46 mm and averaged 20.4 mm for duodenal ulcer (21.2 mm in males and 18.6 mm in females), 16.2 mm for mediogastric ulcer (14.4 mm in males and 19.1 mm in females), 6.6-9.3 mm for other gastric ulcers, and 19.5 mm for combined gastric and duodenal ulcers. The results suggest that gastric ulcer may be charcterized by antral hypomotility and support the concept that motility disorders may play a role in the etiology of gastric ulcer, although secondary changes in motility following the development of an ulcer are also possible.

6245 **RELATION BETWEEN ANTRAL MOTILITY AND MAXIMAL STIMULATED GASTRIC SECRETION IN PATIENTS WITH GASTRODUODENAL ULCERS.** *(Cz.)* Jablonská, M. (Fac. Gen. Med., Charles U., Prague, Czechoslovakia), E. Součková, V. Skop and L. Stamidis. *Sborn Lek* 70(10):301-306, 1968.

Radiological studies of antral peristalsis revealed a significant correlation between antral motility and maximal histamine-stimulated gastric acid secretion (r = 0.78) among 27 males with duodenal ulcers, although there was no such correlation among 14 females with duodenal ulcers or 10 males and 10 females with mediogastric ulcers, and no correlation between antral motility and basal secretion in either group. The basal acid secretion (mEq/hr) averaged 6.22 in males with duodenal ulcers, 3.95 in females with duodenal ulcers, 0.90 in males with mediogastric ulcers, and 0.33 in females with mediogastric ulcers. The corresponding figures for maximal histamine-stimulated acid secretion (mEq/hr) were 23.57, 14.16, 8.80 and 4.47, resp., and the average height of the motility wave on the X-ray polygraph was 22.2, 20.3, 15.2 and 17.1 mm, respectively. Nine of the men with duodenal ulcers showed marked antral motility (peaks above 28 mm) along with an increase in both basal and histamine-stimulated gastric secretion (5-24 mEq/hr and above 25 mEq/hr, resp.). It is suggested that these differences may play a role in the pathogenesis of different types of peptic ulcer in men and women.

6246 **THE SURGICAL TREATMENT OF GASTRODUODENAL ULCERS (EXCLUDING THE ZOLLINGER-ELLISON SYNDROME): INDICATIONS AND RESULTS IN A SERIES OF 392 PATIENTS.** *(Fr.)* Roux, M. (Vaugirard Hosp., France), P. Vayre and L. Debbasch. *Rev Prat* 18(24):3441-3451, 1968.

Among 392 patients treated for peptic ulcers during a 10-yr period, 115 were given emergency surgery (23 for hemorrhage and 92 for perforation) and 31 of these were operated on twice (suture followed by gastrectomy or vagotomy); the resultant total of 423 operations included 219 subtotal gastrectomies, 115 vagotomies (104 complemented by a drainage procedure and 11 by gastrectomy), 2 gastroenterostomies, 82 sutures for perforation, and 5 procedures for bleeding (hemostasis or simple resection of the ulcer). Among the 92 patients treated for perforated ulcers, 3 died and 42 have so far been followed for 3 yr or longer after simple suture; of these, 27 have had a recurrence and 15 are apparently cured, indicating that simple suture is a valid emergency procedure for perforated ulcer. Among 27 patients presenting with severe hemorrhage, 4 were treate medically and 23 came to surgery; of the latter, 3 died The overall mortality among the 392 patients was 3.7% (16 cases), including 8 deaths after elective surgery and 8 after emergency surgery; 11 of these deaths followed a gastrectomy (emergency gastrectomy in 33%) and 3 fo lowed vagotomy and drainage. The 308 elective procedures performed included 195 gastrectomies and 109 vagotomies. The former included 96% of the gastric ulce patients who came to surgery (40% of the total), and in recent years usually involved the Pean technique; in contrast, only 30% of all duodenal ulcer patients were treated surgically, and vagotomy has gradually been replacing gastrectomy as the procedure of choice. The indications for these various methods of treatment in cases of gastric and duodenal ulcer are discussed in detail. Immediate postoperative complications developed in 32 of 219 patients subjected to gastrectomy and includ duodenal fistulas, parietal suppuration, thromboembolism, hemorrhage, and subhepatic abscess. Complications in patients treated by vagotomy included visceroplegia, diarrhea and biliary dystonia. The long-term results were good in 83-84% of those treated by gastrectomy, fair in 15-16% and poor in 1.0-1.3% (recurrences), compared to 88-89%, 6-8% and 3.5-4.6% of those treated by vagotomy and drainage. Late sequellae developed in abou 15% (5% severe) and 5% of the two groups. Despite th higher risk of recurrence, vagotomy and drainage is favored for the surgical treatment of uncomplicated duodenal ulcer.

6247 **THE EFFECT OF ALDOSTERONE ON EXPERIMENTAL ULCERS INDUCED BY CINCHOPHEN.** *(Rum.)* Groza, P. (Inst. Norm. Path. Physiol., Acad. Sci., Bucharest, Romania), V. Buzoianu, F. Constantinescu and S. Ionescu. *Stud Cercet Fiziol* 13(5):391-395, 1968.

When 3 dogs were given cinchophen (Atophan) at a dos of 0.5 g/kg, one died of a perforated ulcer after 7 day and the 2 survivors (sacrificed at 10 days) both showed multiple, mucosal and submucosal, prepyloric ulcers of varying dimensions combined with severe hepatic necrosis dilatation of the sinusoidal capillaries and proliferation o the Kupffer cells. In 5 dogs given both cinchophen and aldosterone (100 μg/day) for 10 days, the gastric ulcers were more localized and superficial, and none perforated hile the liver showed only moderate dystrophy without crosis. The fact that an effective antisecretory agent ch as aldosterone had only a partial protective effect ainst cinchophen-induced ulcers indicates that trophic ctors, rather than peptic secretion, may play the main le in the pathogenesis of these experimental ulcers. ie trophic nature of the protective effect of aldosterone is also confirmed by the reduced hepatic toxicity of ichophen when combined with aldosterone. These re- lts indicate that aldosterone may not have the same otective effect against peptic ulcers in man.

48 ANASTOMOTIC PEPTIC ULCER: ETIOLOGICAL AND THERAPEUTIC CONSIDERA- ONS BASED ON 22 CASES. *(Fr.)* Marchal, G., H. Baumel, Olivier, M. Balmes and M. Bousquet. *Montpellier Chir* (6):853-869, 1967.

ver a 10-yr period, anastomotic ulcers developed in 22 tients (19 men, 3 women) 1-36 yr after peptic ulcer surgery gastric, 17 duodenal). Gastroenterostomies had been perrmed in 9 cases, gastrectomies with gastrojejunal anastomoses 6, gastrectomies with gastroduodenal anastomoses in 4, a strectomy with a Roux-en-Y anastomosis in 1, and a gastro- terostomy and vagotomy in 2. The average age of these tients was 45 yr. Six patients had had severe ulcers which d been considered likely to recur. None had Zollinger- lison's syndrome. Symptoms of anastomotic ulcers included in in 18 cases, hemorrhage in 3, stenosis of the anastomosis 1, and gastrojejunocolonic fistulas in 2. Diagnosis was nfirmed by analysis of the gastric juice and radiological and uoroscopic examinations. Medical treatment was inadequate most cases. The following operations were performed: classical degastrogastrectomy alone in 5 cases (1 recurrence yr later); 2) classical degastrogastectomy combined with gotomy in 12 (no recurrences); 3) vagotomy alone in 2 ery good results with disappearance of the ulcers 12 days ter surgery).

49 CONSERVATIVE TREATMENT OF PEPTIC ULCERS WITH SPECIAL REFERENCE TO NTIINFLAMMATORY AGENTS: PERSONAL EXPERIENCE ITH SODIUM OXYFERRISCORBONE. *(Sp.)* Galán Navarro, L. (Dig. Dis. Clin., INP, Madrid, Spain) and E. Arias Vallejo. *ev Esp Enferm Apar Dig* 27(8):1217-1234, 1968.

48 outpatients (33 men and 15 women, 20-61 yr old) with ptic ulcers (45 duodenal and 3 gastric), sodium oxyferris- rbone (sodium salts of complexes of iron with diketogluconic d ascorbic acids) was injected daily i.m. for 20 days at the ginning of treatment. A second series of injections was given r 20 days beginning with the 40th day of treatment. Associ- ed conditions among patients with duodenal ulcers included cases of pyloric stenosis, 5 of gastroduodenitis, 3 of duoden- is, and 2 of severe gastritis. The clinical picture was typical peptic ulcer in 36 cases, dyspeptic and nonspecific in 3, and ore characteristic of gastritis than ulcers in 7. Other drugs lministered included modifiers of gastroduodenal motility, nticholinergics, tranquilizers and anxiolytics, bismuth, antacids, d others. Treatment was continued for 2 months and followed clinical examinations every 10-15 days and radiological ex- minations once a month. Treatment had to be discontinued in cases because the patients' symptoms remained unchanged. Response to therapy was so good in 37 cases that the patients became asymptomatic. Poor results were obtained in 7 cases and satisfactory ones in 4. Five illustrative case reports are presented along with X-rays.

6250 PEPTIC ULCERS IN CHILDHOOD AND ADOLESCENCE. *(Sp.)* Olmo Fabra, A. (La Paz Soc. Sec. Clin., Madrid, Spain) and E. R. Lucio. *Rev Esp Enferm Apar Dig* 27(8):1147-1160, 1968.

Of 968 patients with peptic ulcers, 8 (3 males, 5 females) were 8-14 yr old and 8 others (7 males, 1 female) were 17-21 yr old. Three of the children in the younger age group had a family history of peptic ulcer. All 8 of these children had duodenal ulcers, which were associated with stomach ulcers in 2 cases, and 7 of 8 were admitted to the hospital with hematemesis. Four of these patients had taken 1-2 aspirin tablets some hours before bleeding developed. All 8 of the older age group also had duodenal ulcers, and 4 were admitted with hematemesis which followed ingestion of salicylates in 2 cases. Patients in both groups were treated medically with bedrest, a mild diet, antacids, anticholinergics and, in some cases, transfusion of small quantities of blood. Response was good: hemorrhages were quickly controlled and anemia disappeared in 10-12 days. No surgery was required. Brief case reports are presented for all 8 of the younger patients.

6251 PEPTIC ULCERS IN ATYPICAL LOCATIONS. *(Por.)* San Juan, F. (Hosp. State Employees, Rio de Janeiro, Brazil), M. Treiger and J. I. C. Rabelo. *Hospital (Rio)* 74(2):443-446, 1968.

Atypical locations were found in 7 out of 680 cases of peptic ulcer (1.02%). These 7 included 3 postbulbar ulcers, 3 in the second part of the duodenum and 1 association of ulcers in the duodenal bulb and proximal jejunum in a 60-yr-old man with Zollinger-Ellison syndrome. Patients included 6 males and 1 female and ranged in age from 20-60 yr. Clinical symptoms had been present for from 10 days to 15 yr. Except for 1 patient with a history of stress, all complained of epigastric or mesogastric pain. Other symptoms included hematemesis, melena, and anemia in 3 cases and melena alone in 1. However, these atypical ulcers can only be diagnosed radiologically. In the subjects studied the basal acidity and acidity after histamine stimulation were greater than in normal individuals or patients with typical peptic peptic ulcers. Conventional medical treatment with diet, antacids, and anticholinergics was effective in only 2 of the 3 patients with ulcers located in the second part of the duodenum. The patient with Zollinger-Ellison syndrome died after gastrectomy and resection of the jejunal loop. A subtotal gastrectomy was performed in 1 case, an antrectomy and vagotomy in 1, and pyloroplasties and vagotomies in 2.

6252 VAGAL INFLUENCE IN GASTRIC ACID SECRETION IN NORMALS AND IN DUODENAL ULCER PATIENTS. *(E.)* Singh, H. (Maulana Azad Med. Coll., New Delhi, India), R. K. Goyal, D. S. Ahluwalia and H. K. Chuttani. *Gut* 9(5):604-608, 1968.

Gastric acid secretory responses were investigated before and after vagal block with a combination of hexamethonium bromide (50 mg) and atropine sulfate (0.325 mg) in 24 duodenal ulcer patients and 25 healthy subjects. The mean values for acid secretion, volume, and acid output in mEq/hr were significantly higher in duodenal ulcer patients than in the normal controls both before and after vagal block. The percentage reduction after vagal block was 78% in normals and 68% in ulcer patients for basal acid output and 67% in normals and 64% in ulcer patients for maximal acid output. These differences are not statistically significant. The ratio between basal maximal acid output was 21% and 19% before vagal block and 17% and 19% after vagal block in normals and duodenal ulcer patients, resp.; these differences are also not statistically significant. These findings negate the thesis of vagal hypertonicity as a cause of hypersecretion in duodenal ulcer patients.

6253 THE "TRIDENT" CARDIA IN ULCERATIVE DUODENAL LESIONS. *(It.)* Mattii, M. (Inst. Radiol., U. Bologna, Italy) and C. Pecunia. *Riv Gastroent* 19(6, Suppl.):751-757, 1967.

A systematic study of the esophagus and cardia showed that a surprisingly large number of patients with duodenal ulcers had trident cardias. This was particularly true in patients with long histories of peptic ulcer disease and active ulcers. This trident image is attributed to simultaneous opacification of the anterior marginal fold of the esophagus, with its characteristic spiral course, and the 2 adjacent paramarginal folds. At the same time, the muscularis mucosae must be in a tonic state so that the folds are prominent and the mucosal relief is not flattened.

6254 RESULTS FROM EMERGENCY SURGERY FOR MASSIVELY BLEEDING DUODENAL ULCER. *(E.)* Snyder, E. N. Jr. (Los Angeles County Gen. Hosp., California) and C. A. Stellar. *Amer J Surg* 116(2):170-176, 1968.

Suture ligation of the bleeding vessel, pyloroplasty and vagotomy were used on an emergency basis to control massive bleeding from a duodenal ulcer in 131 patients (99 men and 72 women). There was a major episode of rebleeding after surgery in 13 cases and 4 of these patients died. There were 22 deaths (17%) within 30 days postoperatively in the whole series, all occurring in the 97 patients who were 50 yr of age or older; 14 of these deaths were due to cardiovascular disease, 4 to sepsis, and 4 followed rebleeding. There were also 69 nonfatal postoperative complications (mostly pulmonary) in 52 patients. The overall mortality was 30% among the 30 patients with giant ulcers (2.5 cm or larger) and 13% among the 101 with smaller ulcers. These results lead to the conclusion that the operation is suitable for the critically ill patient with massive bleeding from a duodenal ulcer. It is recommended that the operation be performed sooner after initial massive hemorrhage than was done during the period covered by this study.

6255 PHARMACOLOGICAL STUDIES ON AN ANTIGASTRIN AND ANTIULCER AGENT, SC-15396. *(E.)* Bianchi, R. G. (Dept. Pharmacol., G. D. Searle and Co., Chicago, Ill.) and D. L. Cook. *Fed Proc* 27(6):1331-1336, 1968.

Oral administration of the nonanticholinergic antisecretory agent, SC-15396 (2-phenyl-2-(2-pyridyl)-thioacetamide), markedly reduced basal gastric secretion in unanesthetized rats (adult male Charles River) with gastric fistulas or ligated pylori. Gastric ulcers induced by pyloric ligation (19 hr) or Δ^1-cortisol (4 mg/day s.c. x 4) in rats as well as histamine-induced duodenal ulceration in guinea pigs (10 mg/kg histamine diphosphate s.c. x 3) could be largely prevented by treatment with the compound at oral doses of 10-25 mg/kg t.i.d. In dogs with gastric fistulas or Heidenhain pouches, oral administration of SC-15396 inhibited the gastric secretory responses to the C-terminal gastrin tetrapeptide (0.1 mg s.c.), and secretion stimulated by food was also markedly inhibited. Insulin-induce gastric secretion (0.75 U/kg i.v.) was moderately inhibited and histamine stimulation (0.05 mg/kg s.c.) was not significantly affected by SC-15396.

6256 THE INCIDENCE OF PEPTIC ULCER IN *SCHISTOSOMA MANSONI* INFESTATIONS. *(Por.)* Mincis, M. (São Paulo Med. Sch., Brazil) and M. P. Vilela. *Rev Ass Med Brasil* 13(10):341-346, 1967.

Peptic ulcers were diagnosed in 260 patients (5.8%) among a total of 4424 with gastrointestinal diseases; in 8 (5%) of 160 patients with schistosomiasis; and in 10 (20%) of 50 patients with chronic liver disease not caused by schistosomiasis. The difference between the incidence of peptic ulcer in the total population and that in patients with schistosomiasis was not significant. Among the 180 patients with the hepatosplenic form of schistosomiasis, 3 had peptic ulcers (2 with confirmed portal hypertension), as did 5 of the 52 with hepatointestinal form. This difference is also not significant. The incidence of peptic ulcers in patients with chronic liver disease was significantly higher than in patients with schistosomiasis. These findings suggest that the high incidence of peptic ulcers in chronic liver disease is due to liver failure rather than to portal hypertension.

6257 EVALUATION OF THE PROGNOSTIC SIGNIFICANCE OF VARIOUS FACTORS IN MASSIVE ULCER HAEMORRHAGE. *(E.)* Andersen, D. (Bispebjerg Hosp., Copenhagen, Denmark), J. G. Klebe and A. Nielsen. *Scand J Gastroent* 3(5):537-544, 1968.

The influence of complicating diseases, nature of the blood passed, age, sex, and ulcer site on the prognosis of massive gastrointestinal bleeding was studied in 102 patients (61 males and 41 females) with gastric (52) and duodenal (50) ulcers. The overall mortality rate was 32%, the cause of death being cardiac complications in 7 patients, pulmonary embolism in 6, exsanguination in 5, respiratory complications in 4, hepatic insufficiency in 4, cerebral complications in 3, surgical complications in 3, and sepsis in 1. The 33 deaths included 28 of 59 patients with complicating diseases and 5 of 43 without, and the difference between these 2 groups was statistically significant ($P < 0.005$). Red hemorrhage occurred in 60 patients, 26 of whom died, while only 7 of 42 patients with black hemorrhage died; this difference was also statistically significant ($P < 0.005$). The difference between the mortality rates of patients younger and older than 70 yr was also statistically significant; 13 of 58 patients under 70 yr died and 20 of 44 patients 70 yr or older died. The difference between male and female mortality rates was not significant, nor was the difference between patients with gastric and duodenal ulcers. There was no evidence proving that older patients bleed more severely than younger patients.

6258 GASTRIC SECRETORY PATTERNS BEFORE AND DURING TREATMENT WITH 1-HYOSCYAMINE. *(E.)* Christiansen, P. M. (Bispebjerg Hosp., Copenhage

enmark) and P. Rødbro. *Scand J Gastroent* 2(4):251-256,
967.

en male patients with chronic duodenal ulceration were ex-
nined with the augmented histamine test (40 µg/kg s.c.) before
d during treatment with optimal effective doses of long-acting
hyoscyamine (0.6-1.2 mg t.i.d.). The outputs and secretory
itterns for volume, acid, pepsin, and intrinsic factor were de-
rmined. The basal and stimulated output of volume and acid
ere significantly reduced during treatment, while the pepsin
d intrinsic factor secretion were essentially unchanged. There
as also no significant change in acid concentration, and 2
tients showed an inverse response (higher acid output than
fore treatment). The reduction in acid output after vagotomy
d pyloroplasty was more pronounced than during treatment
ith 1-hyoscyamine in 8 of the 9 patients tested.

59 **MECHANISM OF THE VAGOTOMY-INDUCED
SUPPRESSION OF THE MAXIMAL ACID RE-
PONSE TO HISTAMINE IN ANTRECTOMIZED DUODENAL
LCER PATIENTS.** *(E.)* Broomé, A. (Cent. Hosp., Boras,
veden). *Scand J Gastroent* 2(4):275-282, 1967.

fter resection of the antrum and duodenal bulb in 6 duodenal
cer patients, the maximal acid response to histamine (MHR)
as determined with and without conduction anesthesia of
e vagus nerves and after vagotomy. Blocking of the vagus by
nduction anesthesia was found to be complete by means of
insulin test. The MHR during conduction anesthesia, im-
ediately after, and 6-8 weeks after vagotomy showed a similar
d significant reduction. Following resection of the antrum
d duodenal bulb in 10 other duodenal ulcer patients, the
HR was determined before and after vagotomy and thereafter
ring simultaneous administration of carbacholine and hist-
nine. A carbacholine-induced increase in MHR was noted after
gotomy. Although there were individual variations in the
ecessary dose of carbacholine, the increase in the MHR took
ace in the subthreshold area for carbacholine even up to pre-
gotomy values for the MHR. Suprathreshold doses of carba-
oline resulted in no further increase in the MHR. This study
dicates that the reduction in MHR after vagotomy is not
used by atrophy of the parietal cells and that parietal cells,
the absence of gastrin-producing areas, are quantitatively
ependent on cholinergic activity in their response to maximal
stamine stimulation.

260 **THE ACETYLATOR PHENOTYPES OF DUO-
DENAL ULCER PATIENTS.** *(E.)* Evans, D. A. P.
Dept. Med., U. Liverpool, England). *Scand J Gastroent* 2(4):
39-292, 1967.

nce rats with diminished acetylating powers due to coenzyme
deficiency develop hypersecretion of gastric acid and duo-
enal ulcers, and since it is known that human beings are gen-
ically constituted as either rapid or slow acetylators (due to
polymorphism of acetyl transferase), 50 male patients with
uodenal ulcers were acetylator phenotyped to determine
hether slow acetylators are more susceptible to peptic ulcer
isease. The results were compared with phenotypes found in
9 healthy male subjects. Phenotyping was performed with
ne sulfamethazine test. The distribution of acetylator pheno-
ypes was essentially the same in duodenal ulcer patients (slow
8, fast 22) as in healthy controls (slow 50, fast 49). There is thus no well-marked predisposition for either acetylator phenotype to develop duodenal ulcer disease. Studies were also done on 7 female duodenal ulcer patients (2 slow, 5 fast) and 13 patients with gastric ulcers (9 slow, 4 fast), but the numbers were too small for statistical evaluation.

6261 **INDICATION OF A POSSIBLE ASSOCIATION BETWEEN PEPTIC ULCER AND VASCULAR LESION OF THE CENTRAL NERVOUS SYSTEM.** *(E.)* Stocks, P. (Colwyn Bay, North Wales). *Brit J Prev Soc Med* 22(4):206-211, 1968.

A comparison of the standardized death rates from peptic ulcer and vascular lesions of the central nervous system revealed a high correlation between the two in both males and females in 28 countries and throughout the rural districts of southern Scotland. High death rates from peptic ulcer in females were prevalent in the 6 countries on consuming more than 2 lb tea/person, although they were not prevalent in either sex in countries with high coffee consumption. During 1949-1953, death rates from both diseases correlated significantly with the physical activity required by English and Welsh men (20-64 yr of age) in 93 occupations, as well as with the mortality from cirrhosis of the liver. This latter fact suggests that alcohol addiction is a common factor in the causation of the two diseases.

6262 **GASTRIC ULCERATION OCCURRING DURING INDOMETHACIN THERAPY.** *(E.)* Taylor, R. T. (Westminster Hosp., London, England), E. C. Huskisson, G. H. Whitehouse, F. D. Hart and D. H. Trapnell. *Brit Med J* 4(5633): 734-737, 1968.

Case histories are presented for 10 patients (6 males and 3 females, 40-93 yr old) who had been treated with either capsules or suppositories of indomethacin for rheumatoid arthritis, gout, ankylosing spondylitis or osteoporosis over a period of 7 weeks to 2 yr. The patients, presenting with anemia, epigastric pain, hematemesis, anorexia, vomiting or melena, were diagnosed as having gastric ulcers via barium meals (9 patients) or at autopsy. The ulcers tended to be prepyloric and were often suspected as being malignant. Symptoms were promptly relieved and rapid radiological healing occurred upon withdrawal of the drug.

6263 **A COMPARISON OF VAGOTOMY PLUS PYLOROPLASTY WITH BILLROTH I GASTRECTOMY IN THE TREATMENT OF GASTRIC ULCER: AN INTERIM REPORT.** *(E.)* Duthie, H. L. (Royal Infirm., Sheffield, England) and G. H. Smith. *J Roy Coll Surg Edinb* 13(6):324-329, 1968.

Among 53 patients operated on for gastric ulcer during a 28-month period, 23 (30-74 yr old) underwent vagotomy with pyloroplasty, 24 (40-73 yr old) underwent Billroth I gastrectomy, 5 showed malignant changes in the ulcer, and 1 could not be followed. No mortality occurred, and postoperative gastric symptoms--graded according to the Visick classification--were similar in the two main groups. Although both operations relieved the symptoms of gastric peptic ulceration, residual symptoms seemed more prominent after vagotomy with pyloroplasty; the difference, however, was not significant. In view of the incidence of episodic diarrhea occurring after total vagotomy, selective gastric vagotomy combined with pyloroplasty is suggested to reduce this symptom.

6264 POST-VAGOTOMY DIARRHOEA EXAMINED BY MEANS OF A NUTRITIONAL CONTRAST MEDIUM. *(E.)* Madsen, P. (Bispebjerg Hosp., Copenhagen, Denmark) and G. Pedersen. *Scand J Gastroent* 3(5):545-552, 1968.

A fast gastric emptying rate, a short small-intestine transit time, and filling of the colon were characteristic radiological findings in 47 patients in whom a vagotomy and pyloroplasty had been performed for peptic ulcer disease and who had been given a nutritional contrast medium containing 60 g Eledon Nestlé, 20 g sucrose, 40 g micropaque, and 200 ml water after at least an 8-hr fast. When these patients were compared with 77 patients, 67 of whom had postgastrectomy symptoms, a more uniform distribution of fast and slow gastric emptying and fast and slow small-intestine transit time, along with less frequent colon filling, were observed in the latter group. Rapid gastric emptying, short small-intestine transit times and colon filling were more common in the 24 patients suffering from diarrhea following vagotomy and pyloroplasty than in the 23 patients without diarrhea. Filling of the colon was noted in 12 patients with diarrhea (4) or a frequent desire to defecate (8). If a nutritional contrast medium offers a more reliable picture of gastrointestinal motility after food intake than barium sulfate, it is possible that rapid gastric emptying, short small-intestine transit times, and rapid colon filling are important factors in the pathogenesis of post-vagotomy diarrhea.

6265 RESULTS OF THE SURGICAL TREATMENT OF PERFORATED GASTRIC AND DUODENAL ULCERS. *(Ger.)* Heller, J. (Distr. Hosp., Nordenham, Germany). *Chirurg* 39(7):315-317, 1968.

6266 PERSONAL EXPERIENCE WITH POSTGASTRECTOMY SYNDROMES. *(Sp.)* Loyarte, H. F. (Mar del Plata Hosp., Argentina) and C. Cavagnaro. *Rev Esp Enferm Apar Dig* 27(6):873-881, 1968.

6267 THE TREATMENT OF GASTRODUODENAL ULCERS WITH LARGE DOSES OF VITAMIN C. *(Fr.)* Debray, C. (Bichat Hosp., Paris, France), F. Pergola, J. P. Hardouin, J. André and Y. Lequerler. *Sem Ther* 44(6/7): 393-398, 1968.

6268 REPORT OF 12 CASES OF DUODENAL ULCER IN ALGERIAN CHILDREN. *(Fr.)* Benallegue, A. (Parnet Hosp., Algiers, Algeria), M. Dubois, S. Benabdallah, S. Nahon and J. P. Grangaud. *Tunisie Med* 46(2):163-179, 1968.

6269 THE TECHNIQUE OF PEAN GASTRODUODENOSTOMY FOLLOWING EXTENSIVE GASTRECTOMY: RESULTS OBTAINED IN THE AREA OF EXTREME INDICATIONS. *(Fr.)* Kuhlmann, N. (U. Hosp. Ctr., Oran, Algeria), A. Kazdali, S. Hamou, A. Aissaoui, F. López-Rodríguez, M. Billieres and F. Renaud. *Tunisie Med* 46(2): 181-187, 1968.

6270 THE SURGICAL TREATMENT OF DUODENAL ULCERS. *(Sp.)* Pi-Figueras, J. (Santa Cruz y San Pablo Hosp., Barcelona, Spain). *Medicina (Mex)* 48(1041): 398-401, 1968.

6271 CURRENT PROBLEMS IN THE TREATMENT OF PEPTIC ULCER. *(Rus.)* Burchinskii, G. I. (A. A. Bogomolets Med. Inst., Kiev, USSR). *Ter Arkh* 40(7): 3-10, 1968.

6272 PERFORATION OF A DUODENAL ULCER IN A NEWBORN INFANT. *(It.)* Proscia, N. (Munic. Hosp., Asti, Italy) and F. Sorisio. *Minerva Chir* 23(13):695-698 1968.

6273 THERAPEUTIC USE OF HIGH-FREQUENCY PHYSIOTHERAPEUTIC AGENTS (INDUCTOTHERMY, MICROWAVES AND ULTRASOUND) IN ULCER DISEASE AND GASTRODUODENITIS. *(Rus.)* Tsimmerman, Ia. S. (Med. Inst., Perm, USSR). *Vop Kurort Fizioter* 33(5): 424-431, 1968.

6274 MASSIVE HEMORRHAGE WITH PEPTIC ULCERS. *(Ger.)* Zukschwerdt, L. (Surg. Clin., U. Hamburg, Germany) and E. Farthmann. *Chirurg* 39(11):491-495, 1968.

6275 THE CHOICE OF OPERATIONS IN UNCOMPLICATED GASTRIC AND DUODENAL ULCERS. *(Ger.)* Zenker, R. (Surg. Clin., U. Munich, Germany), K. Reichel, W. Lorenz, H. Haendle and G. Feifel. *Chirurg* 39(11): 488-490, 1968.

6276 CONSERVATIVE TREATMENT OF ULCER DISEASE. *(Ger.)* Demling, L. (Med. Clin., U. Erlangen, Germany). *Chirurg* 39(11):485-488, 1968.

6277 THE PATIENT'S OPINION OF THE BILROTH II GASTRECTOMY. *(Ger.)* Henne, H. F. (Evangelical Hosp., Oldenburg in Oldenburg, Germany) and E. Kröger. *Chirurg* 39(11):519-522, 1968.

6278 LONG-TERM RESULTS OF BILLROTH I AND BILLROTH II GASTRECTOMIES IN PATIENTS WITH PEPTIC ULCERS. *(Ger.)* Lázár, D. (Munic. Hosp., Nagykanizsa, Hungary). *Chirurg* 39(11):517-519, 1968.

6279 NONRESECTING OPERATIONS FOR GASTRIC AND DUODENAL ULCERS. *(Ger.)* Schreiber, H. W. (Marienkrankenhaus, Hamburg, Germany), H. von Ackere K. Dahm and W. Koch. *Chirurg* 39(11):499-505, 1968.

6280 LEARNING TO LIVE WITH AN ULCER. *(E.)* Fenn, A. K. (Christ. Med. Coll. Hosp., Vellore, India), M. S. George, M. Eapen, K. S. N. Raju and F. H. Garlick. *J Christ Med Ass India* 43(8):387-390, 1968.

6281 MEDICAL MANAGEMENT OF PEPTIC ULCER. *(E.)* Konar, N. R. (Med. Coll., Calcutta, India). *Calcutta Med J* 65(9):354-356, 1968.

6282 THE EFFECT OF DRINKING MERICHLERI MINERAL WATER ON THE MOTOR AND EVACUATORY FUNCTION OF THE STOMACH IN PATIENT WITH ULCER DISEASE. *(Rus.)* Gladilov, N. (Res. Inst. Balneol. Physiotherapy, Sofia, Bulgaria). *Vop Kurort Fizioter* 33(5):431-435, 1968.

283 EXPERIENCES WITH THE SURGICAL MANAGEMENT OF BLEEDING PEPTIC ULCER N SINGAPORE. *(E.)* Ong, S. C. (Dept. Surg. U. Singa- ore) and N. K. Yong. *Aust N Zeal J Surg* 37(4):366- 70, 1968.

284 DUODENAL ULCER TREATMENT WITH AN ANTICHOLINERGIC-ANTACID DRUG 'NEU- RADONNA'. *(E.)* Durrani, H. A. (S. M. H. S. Hosp., ashmir, India) and R. S. Manhas. *Indian Pract* 21(8): 37-544, 1968.

285 THE CHOICE OF DRAINAGE OPERATION IN SUBDIAPHRAGMATIC VAGOTOMY. *(Ger.)* ckmann, L. (Tiefenau Hosp., Bern, Switzerland) and R. irardin. *Helv Chir Acta* 35(1/2):64-68, 1968.

286 SERUM ENZYME LEVELS IN PATIENTS GASTRECTOMIZED FOR PEPTIC ULCERS. *Pol.)* Woszczyk, J. (2nd Surg. Clin., Acad. Med. Poznan, oland). *Pol Przegl Chir* 40(9):996-1002, 1968.

287 CHANGES IN GASTRIC SECRETION DURING THE ADMINISTRATION OF PHENYLQUINO- INECARBOXYLIC ACID (CINCHOPHEN): AN EXPERI- MENTAL STUDY IN RATS. *(It.)* Biliotti, G. (Inst. Spec. urg. Path., U. Florence, Italy), F. Andreoli, A. Pirani and . Bucciarelli. *Arch De Vecchi Anat Pat* 50(3):697-707, 967.

6288 VAGOTOMY AND EXTRAMUCOSAL PYLOROMYOTOMY IN THE RAT. *(It.)* Biliotti, . (Inst. Spec. Surg. Path., U. Florence, Italy) and V. Mori. *rch De Vecchi Anat Pat* 50(1/2):405-412, 1967.

289 INITIAL LONG-TERM RESULTS OF VAGOTOMY IN THE TREATMENT OF DUODENAL JLCER. *(It.)* Rovati, V. (Gen. Surg. Clin., U. Milan, Italy) nd A. Bastagli. *Arch Ital Chir* 94(1):75-90, 1968.

290 THE STRUCTURE AND FUNCTION OF THE KIDNEYS IN PATIENTS WITH GASTRO- DUODENAL ULCERS. *(Pol.)* Urban, A. (2nd Surg. Clin., cad. Med. Krakow, Poland), A. Marczynska, A. Gorniak, . Kulpa and J. Oszacki. *Pat Pol* 19(1):143-152, 1968.

291 STRESS ULCERS IN THORACIC SURGERY: A REPORT OF 14 CASES. *(Sp.)* Chávez spinosa, J. I. (Gen. Hosp., Mexico City), O. Rivero Serrano, 1. Hernández González, M. S. Miner Avila and A. Celis alazar. *Rev Fac Med (Mexico)* 11(2):28-40, 1968.

292 EFFECT OF MEDICAL VAGOTOMY ON GASTRIN- AND HISTAMINE-INDUCED ASTRIC SECRETION IN PATIENTS WITH DUODENAL JLCERS. *(Pol.)* Konturek, S. (1st Clin. Int. Dis., Acad. led. Krakow, Poland) and J. Oleksy. *Pol Tyg Lek* 23(40): 515-1518, 1968.

293 PATHOGENETIC EFFECT OF THE TYPE OF SUTURE ON THE GASTROINTESTINAL NASTOMOSIS AND THE DEVELOPMENT OF RECURRENT ULCERS. *(Sp.)* Diez Gascón, A. (Generalísimo Milit. Hosp., Barcelona, Spain). *Med Cir Guerra* 30(7/8):21-30, 1968.

6294 CLINICAL AND RADIOLOGICAL BEHAVIOR OF GASTRODUODENAL ULCERS TREATED WITH METRONIDAZOL (8823 RP): A REPORT OF THE FIRST CLINICAL TRIALS OF ITS EFFECTS ON ULCERS. *(Sp.)* Sancho-Miñano Padilla, J., M. Remartinez Buera and E. Remartinez Buera. *Med Cir Guerra* 30(7/8):33-39, 1968.

6295 THE ANATOMICAL AND SURGICAL BASIS OF SELECTIVE VAGOTOMY. *(It.)* Paletto, A. E. (Inst. Surg. Anat., U. Turin, Italy). *Minerva Med* 59(83):4391-4404, 1968.

6296 PERFORATED GASTRIC ULCERS. *(Fr.)* Lavigne, J. C. (Surg. Clin., U. Liege, Belgium), J. Leclercq and G. Rogister. *Rev Med Liege* 23(20):642-644, 1968.

6297 VAGOTOMY AS A SURGICAL PROCEDURE FOR THE TREATMENT OF PEPTIC ULCER. *(Bul.)* Atanasov, A. (Inst. Postrad. Med., Sofia, Bulgaria) and G. Ganchev. *Khirurgiia (Sofiia)* 21(4):329-333, 1968.

6298 VAGOTOMY IN THE PREVENTION OF GASTRIC ULCERS DUE TO CINCHOPHEN: AN EXPERIMENTAL STUDY IN RATS. *(It.)* Bucciarelli, G. (Inst. Spec. Surg. Path., U. Florence, Italy), F. Andreoli, G. Biliotti and V. Mori. *Arch De Vecchi Anat Pat* 50(1/2):413-416, 1967.

6299 VELAR AND PHARYNGEAL DYSFUNCTION FOLLOWING VAGOTOMY IN THE HIATAL REGION. *(Ger.)* Jeschek, J. (Prov. Hosp., Leoben, Austria) and R. Stauber. *Chirurg* 39(11):513-517, 1968.

6300 TECHNIQUE IN PYLOROPLASTY. *(Ger.)* Allgower, M. (Surg. Clin., U. Basel, Switzerland), C. Burri and K. Hell. *Chirurg* 39(11):505-510, 1968.

6301 ACUTE PERFORATION OF THE STOMACH AND DUODENUM. *(Ger.)* Dittrich, H. (Surg. Clin., U. Erlangen, Germany). *Chirurg* 39(11):495-498, 1968.

6302 CHANGES IN THE FUNCTIONAL STATE OF THE INTESTINE IN PATIENTS WITH GASTRIC AND DUODENAL ULCERS RECEIVING TREATMENT WITH MUD APPLICATIONS. *(Rus.)* Kafafova, E. D. (Central Inst. Balneol. Physiotherapy, Moscow, USSR). *Vop Kurort Fizioter* 33(5):435-439, 1968.

6303 CHANGES IN NEUROAUTONOMIC REACTIVITY FOLLOWING GASTRIC RESECTION IN PATIENTS WITH DUODENAL ULCER. *(Bul.)* Ganchev, G. (Higher Med. Inst., Sofia, Bulgaria). *Khirurgiia (Sofiia)* 21(2):115-121, 1968.

6304 UNUSUAL POSTOPERATIVE COURSE IN A YOUNG PATIENT WITH A PERFORATED DUODENAL ULCER. *(Rum.)* Arnautu, L. (Inst. Med. Pharm., Iasi, Romania), G. Kreisler, C. Dragomir and M. Diaconescu. *Rev Medicochir Iasi* 72(1):215-218, 1968.

6305 ULCERS OF THE SECOND DUODENAL SEGMENT. *(Fr.)* Dupuy, R. (Beaujon Hosp., Clichy, Paris, France), J. Vallin, H. Dauchy and R. Milcamps. *Entret Bichat [Med]* pp. 55-58, 1968.

6306 EXTERNAL DUODENOSTOMY AS AN AID IN CLOSING "DIFFICULT" DUODENAL STUMPS DURING PARTIAL GASTRECTOMY. *(Pol.)* Lotkowski, K. (Cent. Munic. Hosp., Bytom, Poland) and M. Rawa. *Pol Przegl Chir* 40(10):1067-1073, 1968.

6307 CONTENTS OF IRON, COPPER, COBALT AND ZINC IN THE GASTRIC JUICE OF DOGS WITH EXPERIMENTAL GASTRIC ULCER. *(Rus.)* Babenko, G. A. (Ivano-Frankovsk Med. Inst., USSR) and A. P. Parashchak. *Pat Fiziol Eksp Ter* 12(4):65-67, 1968.

6308 RADIOLOGICAL STUDY OF DUODENAL ULCERS TREATED BY VAGOTOMY COMBINED WITH DIFFERENT TYPES OF GASTRIC DRAINAGE. *(It.)* Slaviero, G. E. (Inst. Surg. Path. U. Pavia, Italy), G. Malvicini and R. Rodolico. *Riv Gastroent* 19(6, suppl.):980-994, 1967.

6309 POSTBULBAR DUODENAL ULCER. *(Por.)* da Costa, A. C. T. (Fac. Med., U. São Paulo, Brazil), A. de Godoy and A. Bettarello. *Arq Gastroent* 5(2):59-64, 1968.

6310 THE PRESENT POSITION OF THE SURGICAL TREATMENT OF PEPTIC ULCER. *(Ger.)* Kunz, H. (2nd Surg. Clin., U. Vienna, Austria). *Munchen Med Wschr* 110(29):1689-1696.

6311 DIFFERENTIAL DIAGNOSIS OF PEPTIC ULCERS OF VARYING ETIOLOGY DEVELOPING AFTER GASTRECTOMY AND TECHNIQUES FOR THEIR SURGICAL TREATMENT. *(Rus.)* Sitenko, V. M. (S. M. Kirov Acad. Milit. Med., Leningrad, USSR) and V. I. Samokhvalov. *Vestn Khir Grekov* 101(9):74-79, 1968.

6312 EARLY COMPLICATIONS IN PEPTIC ULCER SURGERY. *(Sp.)* de la Fuente Chaos, A. *An Acad Nac Med (Madrid)* 85:379-380, 1968.

6313 THE DIAGNOSIS OF CRATERS IN THE DUODENAL CAP. *(E.)* Sim, G. P. G. (Wellington Hosp., New Zealand). *Brit J Radiol* 41(490):792-794, 1968.

See also: 5757, 5758, 5763, 5764, 5840, 5879, 6149, 6151, 6152, 6157, 6160, 6208, 6211

314 **ULCERATIVE COLITIS AND REGIONAL ENTERITIS: RESULTS OF TREATMENT WITH MMUNOSUPPRESSIVE DRUGS (ANTIMETABOLITES).** *(E.)* ebbins, P. M. (Virginia Mason Hosp., Seattle, Wash.) and C. earson. *Bull Mason Clin* 22(3):112-121, 1968.

nmunosuppressive therapy (initially nitrogen mustard, 0.3 g/kg i.v. to total of 12-18 mg, followed by 6-mercaptopurine 0-100 mg/day) and in 2 cases by chlorambucil, 1-4 mg/day) as given to 2 women with regional enteritis and 4 patients (3 omen and 1 man) with ulcerative colitis who had relapsed ter or failed to respond to conventional medical treatment. ost also received concurrent prednisone, prednisolone, Azulfine and/or tranquilizers. Results were good in one patient with gional enteritis and 2 with ulcerative colitis, although in the rst case the dosage of 6-mercaptorpurine had to be reduced ecause of a skin rash and sore tongue and in one of the other cases the patient was lost to follow-up after initiation of 6-ercaptopurine. Another patient with ulcerative colitis showed adual improvement on 6-mercaptopurine over a 3-month period it a subsequent relapse failed to respons. The 2nd regional teritis patient showed no response to nitrogen mustard (no ntimetabolites were given), and the last patient with ulcerative olitis showed slight improvement on 6-mercaptopurine but eatment had to be interrupted because of leukopenia, after hich chlorambucil and prednisone had no effect. It is suggested at failures of immunosuppressive therapy may be due either the selection of medically unresponsive cases, the use of indequate or inappropriate drugs for immunosuppression, or positive antiinflammatory effect with no effect on the basic sease process.

315 **IgG AND ALBUMIN TURNOVER IN CROHN'S DISEASE.** *(E.)* Bendixen, G. (Bispebjerg Hosp., Copenhagen, Denmark), S. Jarnum, J. Soltoft, H. Westergaard, B. Weeke and M. Yssing. *Scand J Gastroent* 3(5):481-489, 1968.

^{125}I-Immunoglobulin G (IgG; 0.2 μC/kg), ^{131}I-albumin (0.5 μC/kg), and ^{59}Fe-iron dextran (0.1 μC/kg) were injected i.v. nto 10 patients (5 males and 5 females, 15-64 yr of age) with Crohn's disease of the terminal ileum, the colon, or both in order to study IgG and albumin turnover; similar studies were performed in 7 patients with intestinal lymphangiectasia and 11 control subjects without diseases affecting serum protein patterns. Serum IgG was normal in 5, decreased in 2 and elevated in 3 cases; while serum IgA was elevated in 1 and normal in the rest, serum IgM was normal in all cases. Serum albumin was depressed in all patients. Studies with ^{59}Fe-iron dextran showed an abnormal intestinal protein-loss in all patients, as evidenced by elevated fecal ^{59}Fe excretion; 1.6-7.7% of the injected dose was lost in 4-5 days, as compared to less than 1% in normal subjects. Fecal clearance of ^{59}Fe was also elevated. The fractional catabolic rate of IgG was highest in patients with high serum levels, while albumin catabolism was highest in patients with low serum albumin. The IgG synthesis rate was elevated in 7 of 10 patients and albumin synthesis was elevated in 2 patients. There was a significant correlation between albumin and IgG catabolism in Crohn's disease and intestinal lymphangiectasia. IgG distribution between intra- and extravascular masses was abnormal in all Crohn's disease patients but one, as evidenced by an elevated fraction of total protein mass intravascularly. Fecal radioiodine excretion from IgG and albumin increased in all patients, especially those with colonic involvement; it was lower in patients with terminal ileitis, but radioiodine from IgG exceeded that of albumin by a factor of 2-4. It is concluded that the ability to develop humoral hypersensitivity is probably normal in Crohn's disease.

6316 **CROHN'S DISEASE OF THE COLON.** *(Fr.)* Paris, J., A. Dupont and J. M. André. *Lille Med* 13(9):979-986, 1968.

Case reports are presented for 10 patients (6 males and 4 females) with Crohn's disease. Except for one 67-yr-old woman, all ranged in age from 17-38 yr. Negative tuberculin tests were obtained in 8 of the 9 patients examined, so it is considered unlikely that tuberculosis is involved in the etiology. After antibiotic and/or corticosteroid therapy had failed, intestinal resections were performed. Examination of the tissue obtained showed that 6 patients had Crohn's ileocolitis, 2 Crohn's colitis, and 1 Crohn's disease of the transverse colon associated with a mucoid epithelioma of the ileocecal valve. Of the 3 patients with apparently normal terminal ileums, 2 had functional impairment due to cecal stenosis or probable stenosis of the ileocecal valve. X-ray findings in 4 cases could be attributed either to Crohn's disease or to reflux ileitis. In all cases histological studies showed sarcoidotic nodules with epitheloid follicles having polynuclear giant cells without caseation. In 3 cases these lesions were not found in the ileum but did occur in the colon. Surgical complications occurred in 2 cases: 1) a stercoraceous fistula necessitating a second operation and 2) an acute stomach ulcer which was resected followed by ploroplasty.

6317 **CROHN'S ILEOCOLITIS.** *(Ger.)* Vandenbroucke, J. (St. Rafael Acad. Hosp., Louvain, Belgium), G. Vantrappen, G. Tytgat, L. Rutgeerts and E. Ponette. *Internist* 9(8):335-343, 1968.

6318 **PROBLEMS IN THE DIAGNOSIS AND TREAT MENT OF CROHN'S DISEASE (REGIONAL ENTERITIS).** *(Ger.)* Willam, K. (Prenzlauer Berg Munic. Hosp., Berlin, Germany). *Chirurg* 39(7):326-328, 1968.

6319 **REGIONAL ILEITIS AND CROHN'S DISEASE.** *(Fr.)* Mouchet, A. and M. Guivarc'h. *Rev Prat* 18(22):3115-3138, 1968.

6320 **REGIONAL ENTERITIS (CROHN'S DISEASE).** *(Ger.)* Wagner, A. (Med. Polyclin., U. Heidelberg Heidelberg, Germany). *Therapiewoche* 18(35):1390-1394, 1968.

6321 **INTESTINAL GANGLIONEUROMATOSIS IN A CASE OF REGIONAL ILEITIS.** *(It.)* Urbano, U. (Inst. Anat. Path. Histol., U. Genoa, Italy) and P. Farina. *Pathologica* 59(879/880):547-548, 1967.

See also: 6326, 6385

Malabsorption

6322 STEATORRHEA DUE TO BLOCKAGE OF LYMPHATIC RETURN IN A PATIENT WITH MALIGNANCY OF THE DEEP LYMPH NODES. *(Fr.)* Vigne, J., G. Marambat, S. Lechat, H. Fournier, C. Debonniere and L. Garetta. *Presse Med* 76(32):1601-1602, 1968.

6323 CURRENT VIEWS ON TROPICAL SPRUE AND A COMPARISON TO NONTROPICAL SPRUE. *(E.)* Corrada, R. M. (U. Puerto Rico, San Juan). *Med Clin N Amer* 52(6):1367-1385, 1968.

6324 INTESTINAL ABSORPTION OF PROTEINS IN DIABETICS. *(It.)* Martellotta, G. (Inst. Spec. Med. Path., U. Bari, Italy) and A. Patimo. *Arch Ital Mal Appar Dig* 34(4):410-415, 1967.

6325 WHIPPLE'S DISEASE. *(Sp.)* Torres, L. M. (José M. Fernández Hosp., Rosario, Argentina), S. Guassoni and J. Pigliacampo. *An Cir (Rosario)* 33(1/2): 35-38, 1968.

See also: 5864, 5867, 5886, 5910, 5973, 6029, 6145, 6153

LARGE INTESTINE

26 REGIONAL ENTERITIS OF THE COLON: A CLINICAL AND PATHOLOGIC COMPARISON

TH ULCERATIVE COLITIS. *(E.)* Farmer, R. G. (Cleveland)
n. Found., Ohio), W. A. Hawk and R. B. Turnbull Jr. *Amer
ig Dis* 13(6):501-514, 1968.

nong 151 patients initially diagnosed as having ulcerative
litis, 87 had pathologically confirmed regional enteritis of
colon, 48 had ulcerative colitis, and 16 had nonspecific
lammatory disease of the colon. The major pathologic feat-
distinguishing ulcerative colitis from regional enteritis is that
former affects only the mucosa while the latter is a trans-
ral disease. Clinical features found most commonly among
tients with regional enteritis included: 1) atypical sigmoido-
pic findings (patchy, irregular involvement with areas of
rmal mucosa between ulcers); 2) perianal fistulas and ab-
sses; 3) recurrent ileitis after colectomy and multiple opera-
ns; and 4) irregular, disproportionate, segmental involvement
the colon with deep strictures and fissures present on X-rays.
nical features found most commonly among patients with
erative colitis included: 1) carcinoma of the colon (8
tients); 2) rectal bleeding; and 3) toxic dilatation of the colon.

27 INVERSION OF AN ILEAL SEGMENT IN RECTOCOLECTOMY: AN EXPERIMENTAL

UDY. *(It.)* Tagliacozzo, S. (Inst. Spec. Surg. Path., U.
gliari, Italy) and G. Monaco. *Rass Med Sarda* 71(1-bis):115-
5, 1968.

ree groups of dogs were subjected to total rectocolectomy,
serving about 4-5 cm of rectum, followed by either simple
oproctostomy (controls), ileoproctostomy preceded by in-
sion of the terminal ileal segment (10-70 cm long), or ileo-
ctostomy preceded by inversion of an ileal segment (10-15
long) 60-80 cm proximal to the anastomosis. In both the
d group and especially the 3rd group, postoperative diarrhea
s less marked and normal defecation was reestablished earlier
an in the 1st group (controls). Initial postoperative weight
s was also less in the 3rd group and stablilization was achieved
thin a few weeks. Intestinal transit was accelerated post-
eratively in controls, delayed in group 2 (particularly in the
sence of long inverted segments), and was not measured in
oup 3. The results show that the interposition of an anti-
ristaltic loop of ileum, preferably 10-15 cm long, is effective
slowing intestinal transit and thus improving intestinal func-
n and nutrition. The best results are obtained when the
erted segment is separated from the anastomosis, presumably
cause the remainder of the ileum is then able to perform its
bsorptive functions more effectively.

328 THE SURGICAL TREATMENT OF BENIGN ANORECTAL LESIONS.

(Ger.) Deucher, F.
arau Cantonal Hosp., Switzerland) and W. Müller. *Helv Chir
ta* 35(1/2):190-223, 1968.

ie diagnosis, etiology and surgical treatment of a variety of
nmalignant conditions involving the anorectal area are dis-
ssed on the basis of experience with 871 patients treated be-
een 1959 and 1966. Operations performed during this period
cluded hemorrhoidectomy (318 cases), and sphincterotomy
77 cases), both together (45 cases), excision of polyps (44 cases), plastic or corrective surgery (43 cases), and operations for rectal prolapse (21 cases) and rectovaginal, rectovesicular or anal fistulas (223 cases). Particular attention is given to the surgical treatment of chronic anal fissures, internal hemorrhoids, rectal prolapse, anal malformations, anorectal abscess, anal fistulas, ulcerative colitis, Crohn's disease of the large intestine, suppurative hidradenitis, Hirschsprung's disease, habitual constipation, and large intestinal polyps. Among 109 cases of large intestinal polyps seen during recent years, 28 were found to have have cellular atypism or carcinoma *in situ* and 5 had invasive carcinomas; these 5 included 1 of 3 with familial polyposis, 2 of 8 with multiple but nonfamilial polyps, 1 of 29 with villous papillomas, and 1 of 69 with adenomatous polyps.

6329 DIAGNOSTIC AND THERAPEUTIC EXPERIENCES WITH 1800 CASES OF APPENDICITIS.

(Ger.) Garlipp, M. (Eisleben Miners' Hosp., Germany). *Zbl Chir* 93(30):1029-1034, 1968.

Among the 1887 patients who were either treated for appendicitis (1793 cases) or admitted with a diagnosis of appendicitis (1807 cases) during the period 1954-64, 316 were treated medically (including 59 patients originally diagnosed as having appendicitis but later found to have ulcers, gastroenteritis, cholelithiasis, cholecystitis or other diseases) and 1571 were treated surgically (including 35 cases in which the diagnosis of appendicitis was not confirmed and 4 in which appendicitis was found during laparotomy for other reasons); 80 patients eventually treated for appendicitis (76 medically and 4 surgically) were originally admitted with diagnoses of acute abdomen, gastroenteritis, ulcer, cholelithiasis, cholecystitis, ileus, peritonitis or other conditions. Among 1536 cases of operatively treated appendicitis, 770 were acute, 385 were subacute, 283 were chronic, and 98 had perforated (5.5% of the total). An inverse proportion was noted between the annual number of perforations and the number of early cases which could be treated conservatively. Only 7 of 1793 patients died of appendicitis, and all 7 were over 60 yr old; causes of death included diffuse peritonitis (in the 4 with perforation), myocardial infarction, acute heart failure, and apoplexy. Other postoperative complications included wound infection (which developed in 61 of the 98 cases of perforation), pneumonia, pleuritis, respiratory infection, urinary tract infection, thrombophlebitis, allergies and dermatoses.

6330 STATISTICAL ANALYSIS OF 200 ADULTS WITH RECTAL AND COLONIC ADENOMAS.

(Fr.) Duhamel, J. and Ngo-Quang-Binh. *Arch Franc Mal Appar Dig* 57(8/9):677-688, 1968.

Of 200 patients with adenomas of the colon and rectum, 79% were males. This percentage was relatively constant for all age groups between 40 and 79 yr. The incidence of differentiated polyps was highest among men (88%) and was followed by that of differentiated inflammatory polyps (74%), undifferentiated polyps (71%), villous polyps (64%), and rectal cancers (58%). The incidence of villous tumors was the same in males and females. Between 15 and 70 yr the number of cases increased progressively with age. Polyps were obviously malignant in 15 (7.5%) of these 200 patients. The percentage of suspect or malignant polyps increased from about 30% of those 5 mm or less in diameter to 86% in those 1 cm or more in diameter. No correlations were found between the presence of a pedicle, the external appearance of a polyp, or its site in the intestine and the benign or malignant nature of the neoplasm. There was no correlation between age and the incidence of multiple adenomas, but there was a correlation between multiple adenomas and cancer. Adenomas were associated with cancers in 24 cases. In 10 of these cases the adenomas were malignant or suspect and in 14 they were benign.

6331 **MANAGEMENT OF BILHARZIASIS OF THE APPENDIX, COLON AND RECTUM.** *(E.)* Ibrahim, A. A. (Fac. Med., Cairo, UAR), A. El Mazni and F. Bahnassy. *J Egypt Med Ass* 51(2/3):169-182, 1968.

The advisability of surgical treatment for prolonged or complicated schistosomiasis was confirmed by results in over 50 cases. The cases included in the study (77) were caused predominantly by *S. mansoni* in the region of lower Egypt, and affected males almost exclusively (74). Those recommended for surgery were 35-45 yr of age, showed malnutrition with enlarged liver and spleen, and had failed to respond to repeated medical treatment. The most common lesions were colonic growths (23), treated by anterior resection (19), or colostomy followed by resection (4). There were also rectal polyps (4), treated by rectal fulguration followed by anterior resection, rectal prolapse (4), treated by abdominal surgery (3) or rectosigmoidectomy (1), rectal strictures and ulcers (4), treated by abdomino-perineal surgery (2) or anterior resection (2), and intussusception (6), treated by right hemicolectomy. Cases of schistosomal appendicitis (12) were treated by appendectomy. In conjunction with antischistosomal drug treatment and restorative therapy, these measures resulted in permanent cure in all cases.

6332 **BLOOD LOSS DUE TO INFECTION WITH *TRICHURIS TRICHIURA*.** *(E.)* Layrisse, M. (Venezuelan Inst. Sci. Invest., Caracas), L. Aparcedo, C. Martinez-Torres and M. Roche. *Amer J Trop Med* 16(5):613-619, 1967.

Fecal blood loss was measured with ^{51}Cr in 9 girls, 3 to 14 yr old, with varying degrees of infection with *Trichuris trichiura* (passing 1-14 million eggs/day; 4 of the 9 also had *Ascaris* and 6 were anemic with hemoglobin levels of 4.3-11.2 g%. The ^{51}Cr studies revealed a fecal blood loss of 0.8-8.6 ml/day, which was roughly proportional to the number of parasites recovered (11-3009, average 1124) after treatment with stilbazium iodide (10 mg/kg b.i.d. for 3 days). The highest correlation coefficient ($r = 0.79 \pm 0.14$) was obtained between fecal blood loss and the number of female worms recovered. It can be calculated that patients lose 0.005 ml blood/worm/day (6-10 times less than from *Necator americanus* and 30-50 times less than from *Ancylostoma duodenale* infestation). It is estimated that infestation with over 800 parasites (producing more than 5 million eggs/day) can induce an iron-deficiency anemia in children.

6333 **THE SIGNIFICANCE OF PSYCHOSOMATIC FACTORS IN PAINFUL ANORECTAL DISEASES.** *(Fr.)* Vilotte, J. (Bichat Hosp., Paris, France), J. C. Hachette and S. Bonfils. *Gaz Med France* 75(30):6115-6122, 1968.

A psychological study of 13 men, aged 32-63, with idiopathic anal pruritis showed that all were dissatisfied with their sex life and complained of impotence, the lack of sexual relations or infrequent relations, ejaculatory disorders, and lack of sexual gratification. In addition, 5 were dissatisfied with their jobs and 2 had histories of insomnia which preceded the pruritis. The personalities of 7 were obsessive, 3 were hysterical, and 3 showed paranoid features. It is concluded that idiopathic anal pruritis is largely the result of an increase in the erogenous nature of the anal mucosa in response to dissatisfaction with the adult genital areas. Psychosomatic factors in anorectal neuralgia and transitory proctalgia are also discussed.

6334 **SUBMUCOUS LIPOMAS OF THE LARGE BOWEL: REPORT OF TWO CASES WITH A PREOPERATIVE DIAGNOSIS AND A REVIEW OF THE LITERATURE.** *(E.)* Mankowitz, B. J. (Newark Beth Israel Hosp., N. J.) and S. Diener. *J Newark Beth Is Hosp* 19(2): 88-99, 1968.

6335 **CASE REPORT OF ATRESIA OF AN EXTENSIVE PORTION OF THE COLON.** *(Ger.)* Kleint, Z. (Munic. Clin., Berlin-Buch, Germany) and R. Schumann. *Kinderaerztl Prax* 36(10):458, 1968.

6336 **POSSIBLE CAUSES OF THE RECTOSIGMOIDITI AND SIGMOIDITIS APPEARING AFTER RADIOTHERAPY FOR CANCER OF THE CERVIX.** *(Fr.)* Fletcher, G. H. (M. D. Anderson Hosp., Houston, Texas) and D. Chassagne. *J Radiol Electr* 49(8/9):639-641, 1968.

6337 **SUBMUCOSAL LIPOMA OF THE CECUM: THE ANATOMICAL AND CLINICAL FEATURES OF A CASE COMPLICATED BY INVAGINATION.** *(It.)* Azara, P. (Inst. Spec. Surg. Path., U. Cagliari, Italy). *Policlinico [Prat]* 75(44):1429-1440, 1968.

6338 **THIERSCH'S OPERATION FOR COMPLETE PROLAPSE OF THE RECTUM.** *(Dan.)* Marckmann, A. (Bispebjerg Hosp., Copenhagen, Denmark). *Nord Med* 80(40):1326-1327, 1968.

6339 **THE SURGICAL TREATMENT OF DIVERTICULAR DISEASE OF THE COLON.** *(Por.)* Pinheiro Magalhães, J. (Sch. Postgrad. Med., Cath. U. Rio de Janeiro, Brazil). *Rev Brasil Cir* 55(9/10):133-136, 1968.

6340 **AMEBIASIS OF THE CECUM AND VERMIFORM APPENDIX.** *(Fr.)* Bouvry, M. *Rev Prat* 18(22):3161-3167, 1968.

6341 **HEMOSTASIS BY MEANS OF SUBMUCOSAL CLIPS IN THE TREATMENT OF VILLOUS RECTAL TUMORS.** *(Fr.)* Gibert, T. (Paris, France). *Presse Med* 76(32):1603, 1968.

6342 **FATAL HEMORRHAGE DUE TO DIVERTICULOSIS OF THE COLON.** *(Ger.)* Bellmann, H. (Surg. Clin., Karl-Marx U., Leipzig, Germany), H. Graetz and R. Keitel. *Zbl Chir* 93(41):1442-1449, 1968.

6343 **THE TREATMENT OF THE COMPLICATIONS OF MEGACOLON.** *(Sp.)* Blando, V. R. O. (Fac. Med. Sci., Natl. U. Cordoba, Argentina) and L. Arata. *Prensa Med Argent* 55(16):719-721, 1968.

344 ANUS PRAETERNATURALIS AS A BORDERLINE SUBJECT INTERESTING BOTH THE URGEON AND THE GYNECOLOGIST. *(Ger.)* Knoch, H. G. Carl Gustav Carus Med. Acad., Dresden, Germany), P. Schneck nd H. Schreiber. *Zbl Chir* 93(42):1484-1488, 1968.

345 A STUDY OF 25 CASES OF COLONIC DIVERTICULOSIS. *(It.)* Galli Stampino, C. (S. Antonio bate Munic. Hosp., Gallarate, Varese, Italy) and G. Tacchi. *linerva Med* 59(74):3845-3867, 1967.

346 SIGMOIDITIS: 47 CASES. *(Fr.)* Guivarc'h, M. (Foch Ctr. Med. Surg., Suresnes, France), Marquand and A. Mouchet. *Ann Chir* 22(5/6):315-320, 968.

347 RADIOLOGICAL NOTES. METASTATIC LESIONS TO THE COLON–REPORT OF HREE CASES. *(E.)* Block, C. (Mount Sinai Hosp., ew York City) and H. M. Peck. *J Mount Sinai Hosp NY* 5(6):639-643, 1968.

348 IRRITABLE COLON SYNDROME (ON THE BASIS OF PRACTICAL SURGERY). *(E.)* akao, Y. (Wakayama Med. Coll., Japan), M. Katsumi, T. ragami, H. Utsunomiya, M. Okada, N. Nakamichi, T. akazawa, S. Ura and S. Fukatani. *Wakayama Med Rep* 3(1):11-18, 1968.

349 SPONTANEOUS PERFORATION OF THE BOWEL IN THE NEWBORN FOLLOWING XCHANGE TRANSFUSION. *(E.)* Castor, W. R. (Gen. losp. Port Arthur, Ontario, Canada). *Canad Med Ass J* 9(19):934-939, 1968.

350 CARCINOID TUMORS OF THE RECTUM. *(E.)* McNatt, M. (Med. Arts Building, Dallas, Tex.) nd C. Rosser. *Texas Med* 64(11):66-69, 1968.

351 PNEUMOCYST OF COLON. *(E.)* Finby, N. (St. Luke's Hosp. Ctr., New York City) and . F. Begg. *New York J Med* 68(22):2941-2943, 1968.

352 COLON PERFORATIONS OCCURRING DURING SIGMOIDOSCOPIC EXAMINATIONS AND ARIUM ENEMAS: A REVIEW. *(E.)* Kiser, J. L. (Ellis ischel State Cancer Hosp., St. Louis, Mo.), J. S. Spratt r and C. A. Johnson. *Missouri Med* 65(12):969-974, 1968.

353 "PROLAPSE OF RECTUM." *(E.)* Johari, S. L. (Bombay Hosp. India). *Bombay Hosp J* 9(2):29-35, 1968.

354 THE DIAGNOSIS AND CONSERVATIVE MANAGEMENT OF ACUTE CAECAL DIVERTICULITIS AND ACUTE CAECAL ULCER. *(E.)* Conrad, P. Middlesex Hosp., London, England). *Aust N Zeal J Surg* 37(4):351-353, 1968.

5355 TECHNIQUES, COMPLICATIONS AND POSTOPERATIVE TREATMENT IN COLOSTOMY. *(Fr.)* Adloff, M. (U. Hosp., Ctr., Strasbourg, France). *France Med* 30(6):295-304, 1967.

6356 THE AUTOPSY FINDINGS IN CROHN'S DISEASE OF THE COLON. *(Fr.)* Martin, E. D. (Bichat Hosp., Paris, France) and J. Delarue. *Mem Acad Chir (Paris)* 94(6/7):217-224, 1968.

6357 VILLOUS ADENOMAS OF THE LARGE INTESTINE. *(It.)* Gentileschi, E. (Surg. Clin., U. Rome, Italy), G. Ribotta, F. Carotenuto, I. Pagni and O. Maggi. *Gazz Int Med Chir* 73(19):1663-1690, 1968.

6358 THE MULTIFACTORIAL ETIOLOGY OF RECTAL CANCER: THE ROLE OF PRENEOPLASTIC CONDITIONS. *(Fr.)* Soullard, J. (Bichat Hosp., Paris, France) and F. Potet. *Entret Bichat [Med]* pp. 589-594, 1968.

6359 SECONDARY CANCERS OF THE RECTUM CAUSED BY METASTASIS OR SPREAD OF A GASTROINTESTINAL OR GENITAL CANCER. *(Fr.)* Debray, C. (Bichat Hosp., Paris, France), J. Leymarios, B. Gouin and C. Marche. *Entret Bichat [Med]* pp. 583-588, 1968.

6360 CHRONIC LARGE INTESTINAL DISEASE AND EXTRA-DIGESTIVE ALLERGIES. *(Fr.)* Mot, J. C. (St. Joseph Hosp., Paris, France), M. Kollitsch and J. Blamoutier. *Entret Bichat [Med]* pp. 577-582, 1968.

6361 THE RECIPROCAL INTERACTION BETWEEN ADENOMAS AND CANCERS OF THE COLON AND RECTUM. *(Fr.)* Duhamel, J. (Broussais Hosp., Paris, France) and Ngo Binh. *Entret Bichat [Med]* pp. 601-604, 1968.

6362 THREE CASES OF COLONIC OR RECTAL DUPLICATION. *(Fr.)* Jaubert de Beaujeu, M., P. Mollard, A. Campo-Paysaa and E. Etienne-Martin. *Ann Chir Infant* 9(2):103-110, 1968.

6363 INTRALUMINAL RECURRENCE DUE TO IMPLANTATION IN NEOPLASMS OF THE COLON. *(Sp.)* Giménez-Salinas, A. (Santa Cruz y San Pablo Hosp., Barcelona, Spain), F. M. Nogueras and R. Hortas. *An Med [Cir]* 54(3):173-180, 1968.

6364 EXTENSIVE RESECTION OF THE RECTUM FOR CANCER. *(Rus.)* Dedkov, I. P. (Kiev Inst. Postgrad. Med., USSR), D. V. Miasoiedov and V. L. Ganul. *Vestn Khir Grekov* 101(9):96-100, 1968.

6365 MODIFICATION IN THE TECHNIQUE OF ILEOTRANSVERSOSTOMY DURING THE TWO-STAGE PROCEDURE FOR RIGHT COLECTOMY. *(Sp.)* Kohan, S. (Rawson Hosp., Buenos Aires, Argentina), A Casoy and E. Fefer. *Prensa Med Argent* 55(11):496-499, 1968.

6366 CROHN'S DISEASE OF THE COLON AND ANAL REGION, OR CROHN'S PROCTOCOLITIS. *(Fr.)* Geffroy, Y. (U. Hosp. Ctr., Rouen, France), R. Laumonier, P. Jouanneau, J. Bourreille, R. Colin, A. Fondimare, J. Hemet and U. Marel. *Gaz Med France* 75(25):4769-4784, 1968.

6367 THE PROBLEM OF UNINTENTIONAL INJURY TO AND CONSCIOUS RESECTION OF THE URETER AND URINARY BLADDER DURING SURGERY FOR RECTAL AND RECTOSIGMOID CARCINOMAS. *(Ger.)* Lynen, F. K. (Med. Fac., Rhine-Westphalia Inst. Technol., Aachen, Germany) and R. Philipp. *Zbl Chir* 93(34):1168-1173, 1968.

6368 CARCINOID TUMORS OF THE RECTUM AND THE CARCINOID SYNDROME. *(Fr.)* Saegesser, F. (Surg. Clin., U. Lausanne, Switzerland), M. Gross and S. Vecerina. *Rev Med Suisse Rom* 88(9):597-609, 1968.

6369 STUDY OF A NEW METHOD OF POST-OPERATIVE ANESTHESIA FOR RECTAL SURGERY. *(It.)* Bardi, F. (G. Ronzoni Med. Surg. Inst., Milan, Italy) and G. Foroni. *Minerva Chir* 23(16):854-868, 1968.

6370 CURRENT STATUS OF THE TREATMENT OF RECTAL CANCER. *(Dut.)* Ceulemans, G. *T Gastroent* 11(1):61-66, 1968.

6371 HISTOCHEMICAL CHANGES IN THE RECTA MUCOSA FOLLOWING URETEROSIGMOIDOSTOMY. *(It.)* Sommo, G. (Munic. Hosp., Genoa, Italy) and G. B. Traverso. *Pathologica* 59(879/880):447-449, 1967.

6372 CLINICAL AND EXPERIMENTAL STUDIES ON HIRSCHSPRUNG'S DISEASE. *(Jap.)* Nakatani, G. (Osaka Munic. U. Grad. Sch. Med., Japan). *J Osaka City Med Cent* 17(3/4):237-260, 1968.

6373 SACRO-PUBIC AND PUBO-PUBIC ELECTRO-STIMULATION THERAPY IN PROCTOLOGY. *(It.)* Rabassini, A. (Inst. Gen. Clin. Surg., U. Bologna, Italy) and R. Bellomo. *Arch Ital Mal Appar Dig* 34(6):530-537, 1967.

6374 INFLAMMATORY TUMORS AFTER APPENDECTOMY. *(Bul.)* Atanasov, A. (Higher Med. Inst., Sofia, Bulgaria) and Kh. Mikhov. *Khirurgiia (Sofiia)* 21(2):133-139, 1968.

6375 AN EFFECT OF INTESTINAL LAVAGE ON CARDIAC FUNCTION IN PATIENTS WITH CHRONIC COLITIS. *(Rus.)* Karimdzhanov, A. K. (Tashsovet Munic. Hosp. Physiotherapy, Tashkent, USSR), Z. P. Filippova and Z. M. Tazieva. *Vop Kurort Fizioter* 33(15):420-424, 1968.

See also: 5845, 5935, 5943, 5975, 5978, 6316

76 **ATTEMPTS TO INDUCE ULCERATIVE HEMORRHAGIC RECTOCOLITIS EXPERIMENTALLY THE GUINEA PIG WITH IMMUNE SERA.** *(Fr.)* Terris, (St. Lazare Hosp., Paris, France) and J.-J. Bernier. *Actualites patogastroent Hotel Dieu* 4(3):A203-A207, 1968.

eterologous immune serum was obtained by injecting 6 ml a mixture containing 1 g homogenized guinea pig colon, eund's adjuvant, and chloramphenicol s.c. into rabbits once week for 10-15 weeks and bleeding them by cardiac puncture. single injection of 5 ml of rabbit anti-colon serum into 40 inea pigs produced congestive or hemorrhagic lesions in 20, cerative lesions in 7, and hypercrinism in 6. Repeated intions of 1 or 2 ml of rabbit anti-colon serum produced only percrinism in 16 out of 30 guinea pigs and congestive or morrhagic lesions in 5. Two injections of rabbit anti-colon rum and 2 lavages with 0.07% formol produced ulcerative ions in 6 out of 23 guinea pigs and necrotic lesions in 16. is method is considered specific for producing lesions of the lon in guinea pigs since 61% of the animals treated with antilon serum developed colonic lesions and only 20% of those eated with heterologous anti-small intestine serum did. Since e course of the disease was acute, these lesions cannot be mpared with those found in chronic ulcerative colitis in mans.

77 **EXPERIMENTAL IMMUNOLOGICAL ULCERATIVE COLITIS.** *(Fr.)* Zweibaum, A. (Broussais osp., Paris, France). *Actualites Hepatogastroent Hotel Dieu* 3):A208-A216, 1968.

hen incorporated into Freund's adjuvant and injected a single ne into the footpads of rats, 15 out of 40 strains of live *cherichia coli* isolated from rat stools consistently produced litis with very characteristic symptoms and anatomic lesions. arrhea and occult or frank bleeding developed 5-8 days after ection and persisted for as long as 15 months. Autopsy of imals which died spontaneously very often showed generalized ritonitis. Lesions produced in the cecum and colon were ry similar to those of ulcerative colitis in humans. A single ection of *E. coli* killed by heat did not produce chronic litis; colitis persisted as long as injections of bacteria were peated. This indicates that the colitis is produced by automunization rather than by the bacteria themselves. Titers antibacterial antibodies were high in animals which developed cerative colitis and very low or nil in those which did not. stematic immunofluorescence studies gave no evidence that s with immunologically-induced ulcerative colitis produced ticolon antibodies. The disease could be prevented by oral ministration of heterologous *E. coli* for 6 days or homologous *coli* daily. Animals with ulcerative colitis were cured by al administration of heterologous bacteria. Those treated with mologous bacteria developed colitis again after interruption treatment. These findings indicate that immunization with mologous *E. coli* produces a permanent disturbance in the cterial equilibrium of the colon and that the abnormal development of some of this flora is directly responsible for the production of ulcerative colitis.

78 **CORRELATION BETWEEN THE RADIOLOGICAL CHANGES IN THE COLON AND CLINICAL STATUS IN PATIENTS WITH ULCERATIVE COLITIS.** *(Dan.)* Katznelson, M. (Copenhagen County Hosp., Gentofte, Denmark), B. Korsgaard and M. Holm. *Ugeskr Laeg* 130(46):1954-1959, 1968.

In 183 patients with clinically and radiologically confirmed ulcerative colitis (70.9% "mild," 20.6% "moderate" and 8.5% "severe"), the clinical status was correlated with the results of barium-enema (113 procedures) and double-contrast (134 procedures) X-ray examination; of these patients, 24 were examined by both techniques and 52 were examined more than once. Radiological assessment was done without knowledge of the clinical status and was based on both the extent and the severity of the changes. In the clinically "mild" cases (4 or less stools/day, little or no visible blood, no fever, tachycardia or anemia, and a sedimentation rate below 30 mm/hr), there was a close correlation with the radiological findings (usually little or no ulceration and involvement of 0-1 colonic sections), although 3 mild cases had visible ulcers and 18 had involvement of 3 or more colonic sections; only 10 mild cases had 5 points or more on a scale which combined both criteria. In the moderate and severe (6 or more stools/day, with visible blood, fever, tachycardia, anemia and a sedimentation rate of 30 mm/day or higher) cases, the radiological changes tended to be less than anticipated; thus, several "severe" cases showed only small ulcers and involvement of less than 10 colonic sections, and the point count in severe cases varied uniformly from 0 to 55. In the 52 patients examined repeatedly, the results of barium-enema studies showed a significantly more consistent correlation with clinical status than those of double-contrast studies. However, there was no difference between the two methods in the diagnostic threshold. It is concluded that the clinical picture is more decisive than the radiological findings in the overall evaluation of a case of ulcerative colitis.

6379 **PLASMA RADIOACTIVITY AFTER RECTAL INSTILLATION OF RADIOIODINE-LABELLED HUMAN ALBUMIN IN NORMAL SUBJECTS AND IN PATIENTS WITH ULCERATIVE COLITIS.** *(E.)* Dalmark, M. (Copenhagen County Hosp., Gentofte, Denmark). *Scand J Gastroent* 3(5):490-496, 1968.

The permeability of the rectosigmoid mucosa to 50 μC of ^{131}I-albumin or ^{125}I-albumin was investigated in 7 normal subjects and 7 patients with ulcerative colitis by determining the plasma radioactivity 0.5-24 hr after rectal instillation. Plasma radioactivity was demonstrable 30-60 min after instillation in both patients and controls, with a maximum of about 1.3% per liter in about 4 hr; the difference between the 2 groups was not statistically significant. The total radioactivity precipitable with 10% trichloroacetic acid (TCA) was also essentially the same in controls and patients. Total urinary excretion of radioactivity in 24 hr was 31% in 5 cases, out of which 8% was precipitable with TCA. The renal clearance of the TCA-precipitable and non-TCA-precipitable fractions was 2-9 ml/min and 25-68 ml/min, resp., in the last 18 hr of the study. It is assumed that the nondialyzable fraction of the plasma radioactivity (about 33% at 4 hr) was bound to peptides liberated from the instilled albumin in the rectosigmoid. These results provide no support for the assumption that abnormal permeability of the colonic mucosa to macromolecules plays a role in the pathogenesis of ulcerative colitis.

6380 IMMUNOCHEMICAL STUDIES ON THE SERUM PROTEINS IN ULCERATIVE COLITIS. *(E.)* Stoica, G. ("N. Gh. Lupu" Inst. Int. Med., Bucharest, Romania) and N. Marcus. *Rev Roum Med Intern* 5(4):259-264, 1968.

In 14 patients with severe and/or recurrent forms of ulcerative colitis, serum immunoglobulins (γG, γA, γM) were analyzed by means of zone electrophoresis on agar gel and by immunoelectrophoresis. The most common abnormality was a diffuse (polyclonal type) increase in 1, 2, and occasionally all 3 of the serum immunoglobulin fractions. Quantitative changes observed in the α_1 and α_2 serum glycoproteins showed good correlation with the severity of the clinical signs of inflammation in individual patients. Though frequent and notable, these changes were not specific for ulcerative colitis.

6381 THE ULCERATIVE COLITIS LYMPHOCYTE: REACTION TO *E. COLI* 0 14 AND COLON ANTIGENS. *(E.)* Stefani, S. (VA Hosp., Hines, Ill.) and S. Fink. *Scand J Gastroent* 2(4):333-336, 1967.

Peripheral blood lymphocytes of 9 male patients with active (grade 2 or 3) ulcerative colitis and 8 normal subjects were cultured in the presence of *Escherichia coli* 0 14 and in the presence of autologous rectal mucosa homogenates. After 6 days of incubation, the degree of blastoid transformation and mitosis was determined by phase contrast microscopy. Lymphocytes from both groups of patients reacted well in the presence of *E. coli* 0 14 but did not react in the presence of rectal mucosa. This suggests that both the ulcerative colitis and normal lymphocytes were sensitized to *E. coli* 0 14 but not to colonic mucosa, despite the reported antigenic similarity between their extracts.

6382 TREATMENT OF ULCERATIVE COLITIS WITH AZATHIOPRINE. *(E.)* Theodor, E. (Tel-Hashomer Govt. Hosp., Tel-Aviv, Israel), E. Gilon and U. Waks. *Brit Med J* 4(5633):741-743, 1968.

Case histories are presented for 7 ulcerative colitis patients (16-54 yr old) in whom azathioprine was administered following relapses which could not be controlled by adrenocortical steroids. Although corticosteroid administration was continued concurrently with azothioprine, the dosage could be reduced, and in 1 case they were withdrawn. Remission began 1-2 weeks after initiation of the antimetabolite in 4 patients. Except for transient leukopenia in 2 patients and vomiting in 2 patients, no complications occurred. It is noted, however, that further experience in the treatment of severe ulcerative colitis with antimetabolites is needed.

6383 AN EPIDEMIOLOGICAL STUDY OF ULCERATIVE COLITIS IN COPENHAGEN COUNTY. *(Dan.)* Bonnevie, O. (Gentofte Hosp., Hellerup, Denmark), P. Riis and P. Anthonisen. *Nord Med* 80(34):1115-1119, 1968.

6384 THE ETIOLOGY, CLINICAL CHARACTERISTICS AND THERAPY OF ULCERATIVE COLITIS. *(Ger.)* Arend, P. (Med. Clin., U. Marburg/Lahn, Germany) and G. A. Martini. *Internist* 9(8):329-335, 1968.

6385 INDICATIONS AND RESULTS OF THE SURGICAL TREATMENT OF ULCERATIVE PROCTOCOLITIS. *(Ger.)* Stelzner, F. (Surg. Clin., U. Hamburg-Eppendorf, Germany). *Internist* 9(8):353-357, 1968.

6386 ULCERATIVE COLITIS DEVELOPING IN A PATIENT WITH BUERGER'S DISEASE. *(Fr.)* Vachon, A., G. Guillemin, P. Paliard, J. Cuilleret and Gilly. *J Med Lyon* 49(1146):1203-1208, 1968.

6387 PERICHOLANGITIS IN PATIENTS WITH ULCERATIVE COLITIS. *(Ger.)* Piesbergen, H. (Hamburg Gen. Hosp., Hamburg, Germany). *Deutsch Med Wschr* 93(40):1910-1911, 1968.

6388 MANAGEMENT OF ULCERATIVE COLITIS IN CHILDREN. *(E.)* Densler, J. F. (Babies' Hosp., Newark, N. J.) and C. Clemente. *J Natl Med Ass* 60(6):491-496, 1968.

6389 HISTOCHEMICAL STUDIES OF THE EPITHELI EPITHELIAL MUCINS IN IDIOPHATIC RECTOCOLITIS. *(It.)* Greco, V. (2nd Med. Clin., U. Rome, Italy), G. Lauro and C. C. Cassano. *Arch Ital Mal Appar Dig* 34(6):602-605, 1967.

6390 THE ETIOLOGY AND PATHOGENESIS OF ULCERATIVE COLITIS. *(Sp.)* Molina Alonso, *Medicina (Madrid)* 36(5):807-820, 1968.

6391 CUTANEOMUCOSAL ULCERATION AS A COMPLICATION OF ULCERATIVE COLITIS. *(Sp.)* Badosa-Gallart, J. (Nuestra Señora del Sagrado Corazón de Jesus Hosp., Barcelona, Spain). *Rev Esp Enferm Apar Dig* 27(5):785-792, 1968.

6392 ULCERO-HEMORRHAGIC RECTOCOLITIS IN PREGNANCY. *(It.)* Spensieri, S. (Inst. Clin. Obstet. Gynec., U. Genoa, Italy), E. Carnevale-Arella and A. Barbara. *Ann Ostet Ginec* 90(3):187-199, 1968.

6393 INDICATIONS FOR COLECTOMY IN ULCERATIVE COLITIS. *(Dut.)* Hellemans, N. (Int. Med. Clin., State U. Groningen, Netherlands). *T Gastroent* 11(1):25-35, 1968.

6394 RESULTS OF IMMUNOELECTROPHORESIS IN ULCERATIVE COLITIS. *(Ger.)* Scheiffarth, F. (U. Hosp., Erlangen-Nuremberg, Germany), H. Götz and F. Qaqish. *Med Klin* 63(37):1459-1462, 1968.

6395 ULCERATIVE COLITIS. *(Ger.)* Halter, F. (Med. Clin., U. Bern, Switzerland). *Schweiz Med Wschr* 98(41):1592-1598, 1968.

6396 THE PATHOLOGICAL ANATOMY OF ULCERATIVE HEMORRHAGIC RECTOCOLITIS. *(Fr.)* Martin, E. D. (Bichat Hosp., Paris, France), F. Potet, C. Marche and A. Alaoui. *Actual Hepatogastroent Hotel Dieu* 4(3):A165-A188, 1968.

7 **PATHOGENETIC FINDINGS IN ULCERATIVE HEMORRHAGIC RECTOCOLITIS.** *(Fr.)* Bonfils, S. (Bichat Hosp., Paris, France). *Actual Hepatogastroent Hotel Dieu* 4(3):A191-A201, 1968.

See also: 6314

6398 **CARCINOMA OF THE PANCREAS: RESULTS OF TREATMENT.** *(E.)* Gallitano, A. (U. Texas M.D. Anderson Hosp., Houston), H. Fransen and R. G. Martin. *Cancer* 22(5):939-944, 1968.

Among 134 cases of biopsy-proven carcinoma of the pancreas seen at the University of Texas M.D. Anderson Hospital and Tumor Institute at Houston between 1944 and 1965, only 20 had no metastases at the time of diagnosis, 66 were jaundiced (30 with pain), and 73 had pain. The average survival ranged from 4.0-5.7 months for 102 patients who were either untreated or treated by radio- or chemotherapy alone, gastrointestinal bypass or biliary drainage, to 8.7-9.3 months for 18 patients treated by pancreatoduodenectomy or biliary drainage plus radiotherapy, 15 months for 4 patients in whom the tumor was excised, 49.5 months for 4 patients given chemotherapy plus radiation, and 5.5 yr for 6 patients with islet cell carcinoma. None of the nontreated group survived more than 18 months, and in 6 of the 10 groups the mortality was 83.9-100% in the first year. In the other 4 groups, 25-75% survived for more than 2 yr; these were patients treated by chemotherapy plus radiation, pancreatoduodenectomy, or excision of the tail and body, and patients with islet cell carcinoma regardless of treatment. The longest survivals (13 yr and 15 yr) were in 2 patients with islet cell carcinoma, and in the entire group of 134 patients, there was only one other who survived more than 5 yr; this was a patient with adenocarcinoma of the head of the pancreas who was treated by a combination of chemotherapy and X-ray. However, 2 other patients treated by chemotherapy plus radiation survived over 4 yr. Pancreatoduodenectomy failed to relieve pain in 75% or jaundice in 67%, and 30% died within 1 month (maximum survival 3 yr). Biliary drainage was effective against jaundice in 21 of 34 cases and against pain in 14 of 21; when combined with radiotherapy, this procedure also provides some prolongation of life.

6399 **EXPERIMENTAL PERFUSION OF THE PANCREAS.** *(E.)* Draznin, N. (U. Clin., Bonn, Germany), A. Blomer, R. Scholz and H. Seboldt. *Minnesota Med* 51(11):1549-1551, 1968.

Perfusion of the excised pancreas with an amino acid solution containing Trasylol (400,000 U/liter), dextran (50 g/liter). Complamin (10 ml/liter) and sodium bicarbonate to pH 7.35-7.40 was found to prevent pancreatitis in 16 experimental transplants. The test animals, dogs weighing 15-20 kg, were heparinized with 5000 IU before excision of the organ. A specialized gravity perfusion device was used to control flow at an optimum of 75 ml/min and maintain temperature at 2-6C. Intracellular sodium and potassium content and oxygen consumption did not change significantly during perfusion. Successful grafts functioned for 6-19 days (without immunosuppressive therapy), and one animal survived for 2.5 months.

6400 **EFFECT OF STARVATION ON THE FUNCTION AND MORPHOLOGY OF RAT PANCREAS.** *(E.)* Bučko, A. (Res. Inst. Human Nutrit., Bratislava, Czechoslovakia), Z. Kopec and J. Babala. *Nutr Dieta* 10(4): 266-275, 1968.

Male albino rats (108) weighing 190-200 g were used to study the effects of starvation for 24-144 hr on the pancreas and on protein fractions of the plasma. Enzyme activity in pancreas homogenates showed a decrease after 48 hr of starvation and enzyme deficiency became acute after 96 hr. By 144 hr, amylase decreased to 3.6%, lipase to 14.9% and protease to 42.1% of control values. Protein nitrogen in wet tissue decreased steadily from 48 to 120 hr, then remained stable at 67.3% of initial values. Histologic degeneration and decrease in cellular RNA began after 72 hr of starvation. Total plasma proteins remained unchanged, as did albumin, α_1- and β-globulins, but α_2-globulins decreased and γ-globulins increased significantly.

6401 **COMPARATIVE EFFECTS OF EXCESS DIETAR DL-METHIONINE ON THE PANCREATIC MORPHOLOGY OF RATS FED DIETS BASED ON CASEIN OR SOYBEAN MEAL.** *(Fr.)* Bourdel, G. (Ctr. Nutrit. Res., Bellevue, France) and A. Girard. *Arch Anat Micr Morph Exp* 57(2):167-176, 1968.

In a study of the mechanism of methionine toxicity, 0.4 or 2% DL-methionine was added to the diets of weaned male Wistar CF rats. All animals were fed diets containing 15% protein as casein or soybean meal. Histological studies of the pancreas were made after 4, 8, and 12 weeks. Vacuolated cells were found in larger numbers and for a longer time among animals receiving 2% methionine and casein than among those receiving 2% methionine and soybean meal. Intravacuolar inclusions gave an intense positive reaction for DNA and probably originate from the nucleus. The number of mitoses increased earlier and this increase persisted longer among animals fed 2% methionine and casein than among other groups. Between the 4th and 8th week abnormal mitoses were seen in which the distribution of chromosomes suggests that chromosome remna from intravacuolar inclusions are involved.

6402 **ACID MUCOPOLYSACCHARIDES IN CULTU FIBROBLASTS OF CYSTIC FIBROSIS OF TH PANCREAS.** *(E.)* Matalon, R. (J. P. Kennedy Jr. Mental. Retard. Res. Ctr., Chicago, Ill.) and A. Dorfman. *Biochem Biophys Res Commun* 33(6):954-958, 1968.

The qualitative and quantitative distribution of acid mucopolysaccharides was determined in fibroblasts cultured from skin of 4 cystic fibrosis patients and compared with that in fibroblasts from normal patients and those with Hurler's and Marfan's syndromes. The cystic fibrosis cells contained large inclusions which stained metachromatically with toluidine blue and which were somewhat larger and less uniformly distributed than those found in Hurler's and Marfan's diseases. The total polysaccharide isolate (fractions of chondroitin sulfate, hyaluronic acid, and dermatan sulfate) was 8-10 times greater than normal in 3 patients and twice normal in 1 pati Although the amount of dermatan sulfate was increased in Hurler cells and the amount of hyaluronic acid was increase in Marfan cells, the distribution of each of the 3 fractions i cystic fibrosis cells closely paralled that of normal cells. Th impressive increase in mucopolysaccharides represents the fi clearcut chemical difference between cystic fibrosis and nor cells, and establishes the fact that the genetic defect, despit the uncertainty surrounding its nature, is expressed in the fibroblasts as well as in the exocrine glands.

403 THE CURRENT STATUS OF THE SURGICAL TREATMENT OF CANCER OF THE PAN-
REAS. *(Sp.)* Gallart-Esquerdo, A. (Nuestra Señora del
ıgrado Corazón de Jesús Hosp., Barcelona, Spain). *Medicina*
Iex) 48(1040):370-372, 1968.

404 TRAUMATIC LESIONS OF THE PANCREAS. *(Sp.)* Miyar González, A. (General Yagüe 3,
viedo, Spain). *Galicia Clin* 40(2):111-129, 1968.

405 A STUDY OF SWEATING IN TWO CASES OF ADULT MUCOVISCIDOSIS. *(Fr.)* Grandchamp,
. (Lab. Clin. Physiopath., Cantonal Hosp., Geneva, Switzer-
nd), A. Hany, L. Mérier and P. Press. *Schweiz Med Wschr*
3(36):1388-1392, 1968.

406 METABOLIC CHANGES FOLLOWING TOTAL PANCREATECTOMY IN ONE CASE. *(Ger.)*
unner, E. (1st Med. Clin., U. Vienna, Austria), H. Frischauf
d R. Kühlmayer. *Wien Klin Wschr* 80(44):805-808, 1968.

407 PRIMARY HYPERPARATHYROIDISM AND DISEASES OF THE PANCREAS: DISCUSSION
F ONE PERSONAL CASE AND 80 TAKEN FROM THE
TERATURE. *(Fr.)* Plauchu, M., J. Mallureet and J. F.
risse. *J Med Lyon* 49(1151):1503-1561, 1968.

408 BLIND DISTAL PANCREATECTOMY. *(It.)* Trivellini, A. *Arch Ital Chir* 94(1):155-156,
68.

409 VON HIPPEL-LANDAU'S DISEASE, CANCER OF THE HEAD OF THE PANCREAS, AND
NCREATIC ISLET-CELL TUMOR: A COMPARISON OF
HE RADIOLOGICAL ASPECTS OF THREE ANGIOMATOUS
UMORS REVEALED BY SELECTIVE ARTERIOGRAPHY.
r.) Legré, J. (Fac. Med., Marseille, France), C. Guien, J. P.
ément and H. Pietri. *Presse Med* 76(40):1911-1914, 1968.

10 RESULTS OF THE SURGICAL TREATMENT OF DUODENOPANCREATIC CARCINOMA. *(Ser.)* Štulhofer, M. (Dr. O. Novosel Gen. Hosp., Zagreb, Yugoslavia), B. Gjivan and Z. Brala. *Lijecn Vjesn* 90(5): 419-424, 1968.

6411 PSEUDOCYSTS OF THE PANCREAS: A REPORT OF 50 CASES. *(Fr.)* Cuilleret, J. (Hotel Dieu, Lyon, France), A. Michel, J. J. Carron, G. Spay, A. Campo-Paysaa, J. Cuche, P. Mikaeloff, A. Vachon and G. Guillemin. *Ann Chir* 22(15-16/17-18):1011-1028, 1968.

6412 SUPPRESSION OF THE POST-TETANIC POTENTIATION OF NEUROMUSCULAR TRANSMISSION FOLLOWING PANCREATECTOMY OR ADMINISTRATION OF ACETYLCHOLINESTERASE INHIBITORS. *(Rus.)* Sharapova, P. Kh. (Dept. Human & Anim. Physiol., V. I. Ul′ianov-Lenin U., Kazan, USSR). *Biull Eksp Biol Med* 66(10):12-14, 1968.

6413 EXPERIMENTAL STUDIES ON ALLOTRANSPLANTATION OF THE PANCREAS IN DOGS. *(Jap.)* Koike, M. (Osaka Munic. U. Grad. Sch. Med., Japan). *J Osaka City Med Cent* 17(3/4):205-229, 1968.

6414 THE MORPHOLOGY OF THE EXOCRINE PANCREAS IN THE AGED. *(It.)* Gasbarrini, G. (Inst. Spec. Med. Path., U. Bologna, Italy), F. Faggioli, F. Miglio and E. Ferrandino. *Arch Pat Clin Med* 44(6): 433-444, 1968.

6415 ANNULAR PANCREAS AS THE CAUSE OF HIGH INTESTINAL OBSTRUCTION IN NEWBORNS, ILLUSTRATED WITH FIVE CASES. *(Bul.)* Karaivano, M. (I. P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria), V. Tashev, L. Pachedzhiev and V. Kalcheva. *Khirurgiia (Sofiia)* 21(2):168-172, 1968.

6416 DIABETES SECONDARY TO PANCREATIC CHANGES. *(Sp.)* Lozano Castañeda, O. (Natl. Inst. Nutrit., Mexico City, Mexico). *Rev Invest Clin* 20(2):221-232, 1968.

See also: 5850, 5855, 5857, 5878, 5895, 5896, 5904, 5911, 5915, 5932, 5949, 5953, 5968, 5976, 6232

6417 THE PRESENCE OF A SPECIAL ANTIGENIC FACTOR IN THE DUODENAL FLUID DURING EXPERIMENTAL PANCREONECROSIS IN DOGS. *(Rus.)*

Cherkezova-Kinova, Ie. R. (Higher Med. Inst., Sofia, Bulgaria). *Biull Eksp Biol Med* 66(9):71-75, 1968.

The duodenal secretions collected via duodenal fistulas from dogs with experimental pancreatitis induced 26-28 hr earlier were found to yield an allergic reaction when they were injected s.c. into the same dogs. No such skin reactions were produced by s.c. injection of normal duodenal fluid. Serological studies with these pathological duodenal secretions revealed the presence of a factor which was related antigenically (as shown by complement-fixation tests in rabbits) to duodenal wall tissue but not to either normal or necrotic pancreatic tissue.

6418 COMPLICATIONS OF ACUTE NON-TRAUMATIC PANCREATITIS. *(E.)*

Elmslie, R. G. (Prince Henry Hosp., Sydney, Australia). *Med J Aust* 2(20):895-899, 1968.

Detailed case reports are presented for 4 patients (2 men and 2 women, 22-55 yr old) with acute pancreatitis who developed rare unpredictable complications which were either difficult to diagnose or to treat. These included: 1) persistent obstructive jaundice due to papillitis in a 31-yr-old alcoholic, 2) a retrogastric extrapancreatic cyst filled with concentrated pancreatic juice, 3) pancreatic ascites in association with an extrapancreatic cyst, and 4) intraabdominal abscesses occurring in other than the usual (upper-left) location. All 4 patients recovered after correct diagnosis and appropriate therapy, but it is pointed out that such complications may be fatal. They usually appear after the 2nd or 3rd week of hospitalization, and are considerably more common in alcoholics. When conservative treatment fails, the complications can generally be resolved by surgical intervention.

6419 THE INFLUENCE OF EXPERIMENTAL ACUTE PANCREATITIS ON THE ADRENALS IN DOGS.

(Pol.) Tyszkiewicz, S. (Acad. Med., Bialystok, Poland) and E. Bernacki. *Acta Physiol Pol* 19(5):657-669, 1968.

Thirteen untreated dogs died of acute pancreatitis within 24 hr after an injection of bile incubated with trypsin into the common bile duct. Among 18 other dogs kept alive after the same procedure by treatment with parathormone (6 U/kg/day), 13 were sacrificed 4 days later and 5 were sacrificed at the end of 1 month. Autopsy studies of all 31 dogs revealed significant pathological changes in the adrenal glands, confirming the possible value of adrenocortical hormones in the management of pancreatitis. In dogs dying or killed within 4 days after the onset of acute pancreatitis, focal degenerative lesions and necrosis were localized mainly to the medullary substance and the zonae reticularis and fasciculata of the adrenal cortex. These lesions were associated with severe damage to the adrenal vasculature. The adrenals of dogs surviving for 1 month after the onset of acute pancreatitis showed small cysts, surrounded by proliferating argentophil and collagen fibers, marked thickening and hyalinization of the vascular walls, and focal hypertrophy and proliferation of cortical cells, sometimes leading to the formation of adenoma-like structures.

6420 ACUTE EXPERIMENTAL PANCREATITIS INDUCED BY A CLOSED DUODENAL LOOP.

(It.) Galletti, G. (Inst. Gen. Clin. Surg., U. Bologna, Italy), A. Cavallari, C. Battisti and M. Pierangeli. *Riv Gastroent* 19(6, Suppl.):675-738, 1967.

Pfeffer's method of ligating the duodenal loop was used to induce acute pancreatitis in 42 dogs; 6 others served as controls. All of the animals were sacrificed 4-20 hr after surgery by injecting 3 mg/kg body weight of a mixture of neoprene, India ink, and water (1:1:2) into the femoral artery. Changes began to occur in the pancreatic blood vessels 4 hr after surge becoming more pronounced during the next 6 hr. The pancreatic parenchyma developed only a slight edema which appeared 8 hr after surgery. Many extravasations of plasma were found in the intercellular spaces and were accompanied by involutive changes in the cells of the adjacent acini 12 hr after surgery; the vascular changes remained similar to those seen earlier. Hemorrhagic necrosis involving the glandular parenchyma, particularly in the region around the blood vessels, appeared 14 hr after surgery. The structure of the excretory du remained intact until the 16th hr when their outside walls became involved. The epithelium lining the ducts remained inta until the more advanced stages of pancreatitis while their lume became filled with necrotic material and contrast medium. Th walls of some ducts had many ruptures through their entire thickness 18-20 hr after surgery. Vascular features were completely hidden after the 14th hr, but it could be shown that contrast medium had extravasated from the veins. To rule ou the possibility that duodenal reflux is an etiological factor in pancreatitis, the pancreatic duct as well as the duodenum was ligated in 4 dogs. These animals also developed necrotic hem rhagic pancreatitis, but their pancreatic excretory ducts remai intact and were filled with pancreatic secretion; no traces of contrast medium were found. It is concluded that vascular changes and not duodenopancreatic reflux play the major role in the etiology of acute experimental pancreatitis.

6421 HYPERTENSION AND LOCALIZED PORTAL STASIS IN CHRONIC PANCREATITIS: 126 CASES EXAMINED BY SPLENOPORTOGRAPHY AND SPLENOMANOMETRY. *(Fr.)*

Leger, L. (Cochin Hosp., Pari France), J. P. Lenriot and G. Lemaigre. *J Chir (Paris)* 95(5/6 599-608, 1968.

Splenoportal anomalies were found in 69 (54%) of 126 patien with chronic pancreatitis. The percentage of patients with pancreatic lithiasis was 56% for the entire group, but was eve higher (70%) among patients with splenoportal anomalies. In 14 patients these anomalies consisted of a smoothing out of the lower convexity of the splenic axis, resulting in blockage of free flow from the splenic vein. This was manifested by dilatation of the splenic vein upstream, elevated splenomanometric pressures despite the absence of collateral circulation, and initial symptoms of venous compression. In 25 cases (36.5%) there was incomplete stenosis of the splenic vein or splenomesenteric trunk which was sometimes accompanied by localized portal hypertension and evidence of collateral circula tion. Complete stenosis of the axis of the splenic vein was seen in 30 patients (43.5%). Pathological studies of surgical specimens from 9 patients in this last group showed that 5 had severe phlebosclerosis or proliferating localized endophlebitis, 2 had complete thromboses which had become slightly permeable to blood flow again, and 2 had splenic veins which were almost intact with only mild endophlebitis. Splenectomy combined with treatment of the pancreatic lesion is considered essential in solving the circulatory problems of these patients.

6422 ACUTE PANCREATITIS IN THE CHILD: STUDY OF 9 CASES. *(Por.)* Ferreira, E. A. B. (Fac. Med., U. Sao Paulo, Brazil), A. A. Ferreira Jr., M. R. de Oliveira, F. Queiroz, D. Birolini and P. Curti. *Rev Hosp Clin Fac Med S Paulo* 23(3):131-136, 1968.

Case reports are presented for 9 children, aged 4-12 yr (5 males, 4 females), with acute pancreatitis. Mumps was probably the cause in 1 case; no etiological factors could be definitely established in the remaining 8. In all but 2 cases the diagnosis was established on the basis of clinical symptoms, elevated serum amylase values, and radiological examination. A diagnosis of acute necrotic, hemorrhagic pancreatitis was established during surgery on 2 severely ill patients with normal serum amylase values and histories of ascariasis. The normal amylase levels are attributed to extension of the pancreatic lesions. In these 2 cases it is possible that parasites might have temporarily blocked the pancreatic ducts, but none were found there during surgery and neither of the patients were jaundiced, a common finding in such cases of acute pancreatitis. Blood cholesterol determinations ruled out the possibility of familial hyperlipemia in all 9 cases. Treatment was classical in 4 cases. Trypsin inhibitor (Trasylol, 4000 U/kg body weight/day for 5 days) was administered by i.v. infusion to the 2 operated patients and 3 others. Despite this treatment, one patient had persistently high serum amylase values.

6423 PHYSIOLOGICAL AND PATHOGENETIC ASPECTS OF THE RELATION BETWEEN ALCOHOLISM AND PANCREATITIS. *(Sp.)* Galindo, F. ("Dr. B. Udaondo" Natl. Hosp. Gastroent., Buenos Aires, Argentina). *Prensa Med Argent* 55(25):1196-1200, 1968.

In 10 normal subjects with Diamond-Dreiling catheters in the duodenum, intraduodenal administration of 10 cc ethanol increased both the volume of pancreatic secretion (from an average of 72.1 to 135.6 cc) and the bicarbonate output (from 2489 to 5099 mEq) during the next 80 min, while the lipase output increased in 8 cases (from an average of 32.5 to 38.7 Bondi U). Since the alcohol was instilled directly into the duodenum and the gastric secretions were continuously aspirated, this effect was independent of the known effect of alcohol on gastric secretion. In 3 patients with normal common bile ducts, previously operated on for cholelithiasis, intraduodenal administration of 5 cc ethanol increased both the secreting and the resting pressure in the common bile duct as measured by a Caroli manometer. These increases are ascribed to the hypertensive effect of alcohol on the sphincter of Oddi. In view of these results and the fact that a history of alcoholism was found in 15.5% of 77 cases of chronic pancreatitis and in 90% of those with calcifying pancreatitis lithiasis), it is concluded that alcoholism favors the development of pancreatitis by increasing both pancreatic secretion and sphincter tone, thus yielding canalicular hypertension, and at the same time increasing the pH of the pancreatic juice, which favors precipitation of calcium.

6424 LOCAL COOLING OF THE STOMACH IN SEVERE FORMS OF ACUTE PANCREATITIS. *(Rus.)* Petrov, B. A. (N. V. Sklifosovskii Sci.-Res. Inst. Emergency Med. Treat., Moscow, USSR), A. P. Shindiaikin, E. I. Gal'perin, V. M. Makhov and I. S. Volkova. *Khirurgiia (Moskva)* 44(8):3-8, 1968.

Local cooling of the stomach was used in the treatment of 28 patients with severe forms of acute pancreatitis because preliminary tests on dogs and patients with bleeding stomach ulcers had shown that it reduced blood levels of pancreatic enzymes. A rubber balloon was connected to a double rubber tube through which 600-900 ml of a 1:1 mixture of water and 96% alcohol, cooled to -4C, was pumped at a rate of 500 ml/min. The balloon was inserted after premedication and local anesthesia of the pharynx and esophagus. The patients were wrapped in electrical and wool blankets so that their rectal temperature did not drop below 35C. These 28 patients, aged 29-74 yr, had failed to respond to conservative treatment and Trasylol; 9 were more than 60 yr old. Initial blood levels of trypsin and lipase were elevated in 20 and 22 patients, resp., and trypsin inhibitor was reduced in all 28. The rapid clinical improvement occurring in 26 of the 28 was accompanied by decreases in blood trypsin and lipase and urinary amylase levels and increases in blood trypsin inhibitor. Shortly after cooling was discontinued surgery was performed on 2 patients with pancreatic necrosis who did not respond to cooling treatment; the bursa omentalis was opened and drained and then Trasylol was administered locally and i.v. Both recovered satisfactorily. Several days after cooling treatment 2 other patients were operated on for cholelithiasis and, in 1 case, acute cholangitis. Two illustrative case reports are presented.

6425 POSTGANGLIONIC NEUROTOMY FOR PAIN IN CHRONIC PANCREATITIS. *(Rus.)* Smirnov, A. V. (Leningrad Med. Inst. Sanitation Hyg., USSR) and Iu. N. Gol'man. *Khirurgiia (Moskva)* 44(8):66-68, 1968.

Serum pancreatic enzyme activities were determined in 30 normal controls and 126 patients with chronic recurrent pancreatitis. Follow-up studies were made on 37 patients at various intervals after postganglionic neurotomy performed by the method of Ioshioka and Wakabayashi. Before surgery, serum trypsin activity was greatly elevated and trypsin inhibitor activity greatly reduced; significant, but smaller, elevations were found in serum amylase and lipase activities. Surgery gave good clinical results in 23 patients with early pancreatitis and satisfactory results in 9. Unsatisfactory results were obtained in 5 patients with long histories of pancreatitis, severe disturbances in pancreatic endocrine and exocrine function, and associated diseases of the biliary tract. After surgery, serum trypsin activity decreased to 25-33% of its original value and trypsin inhibitor activity increased in all cases. Serum lipase and amylase activities tended to increase during the first 3-6 months after surgery but decreased to normal values later.

6426 DRAINAGE AND BYPASS OF WIRSUNG'S DUCT IN THE TREATMENT OF CHRONIC PANCREATITIS: REPORT OF 115 CASES. *(Fr.)* Mercadier, M. (La Pitié Hosp., Paris, France), J. P. Clot and C. Regensberg. *Ann Chir* 22(21/22):1279-1284, 1968.

Of 115 patients with chronic pancreatitis, 64 had calcareous forms and 51 had noncalcareous forms. The ratio of males to females was 10:1, the average age was 30-40 yr, 39% had histories of alcoholism and 21.7% histories of acute pancreatitis, but only 12.5% had had cholelithiasis. Wirsung's duct was drained externally in 28 cases, drained internally in 23, and anastomosed to the jejunum in 55; 9 cases were treated, with generally poor results, by anastomosis to a pseudocyst communicating with the pancreatic duct. These procedures were combined with cholecystectomies in 20 cases, various types of anastomosis between the biliary and gastrointestinal tracts in 58 cases, and gastroenterostomies in 12 cases. Only 2 patients (1.8%) died after surgery. Long-term follow-up of 95 cases showed that 21 (24%) died of progressive pancreatic disease. Good results were obtained in 43 (58%) of the 74 survivors, fair results in 19 (25%), and poor results in 12 (17%). Side-to-side pancreaticojejunostomies gave the best results. Repeat operations had to be performed on 32 of the 95 patients followed.

6427 MANAGEMENT OF PANCREATITIS IN INFANTS AND CHILDREN. *(E.)* Fonkalsrud, E. W. (UCLA Sch. Med., Los Angeles, Calif.), R. P. Henney, T. A. Riemenschneider and W. F. Barker. *Amer J Surg* 116(2): 198-203.

The treatment of 17 children under 18 yr of age for acute and chronic pancreatitis is described. Most common was pancreatitis in patients being treated with glucocorticoids or immunosuppressive agents for nephrosis or lupus erythematosus; this resulted in 4 deaths out of 6 cases. Idiopathic pancreatitis caused death in 2 out of 4 cases. No cases of obstructive pancreatitis (1), traumatic pancreatitis with pseudocyst (2) and without pseudocyst (3), or hereditary pancreatitis (1) resulted in death. Therapy followed the same principles established for adults and included replenishment of body fluids and blood, calcium gluconate, abdominal aspiration, anticholinergic drugs, meperidine, antibiotics and Trasylol. Post-traumatic pancreatitis with pseudocyst is best treated by external tube drainage if the proximal pancreatic duct is unobstructed.

6428 PANCREATITIS-AN AUTOPSY STUDY. *(E.)* Deodhar, L. P. (Topiwala Natl. Med. Coll., Bombay, India) and C. K. Deshpande. *J Postgrad Med* 14(4):187-190, 1968.

Among 2,297 autopsies performed during the period 1964-67, there were 25 cases of pancreatitis (19 males and 6 females, 1-70 yr of age). Gross and microscopic examination revealed 3 distinct groups of cases: acute pancreatitis, where pancreatic lesions were the primary cause of death (9 cases); chronic pancreatitis, where pancreatic lesions were a contributory cause of death (10 cases); and subacute pancreatitis or pancreatitis with fat necrosis, where the lesions were incidental at the time of autopsy (6 cases). Hemorrhage and necrosis in the major portion of the pancreas were observed in a majority of acute pancreatitis cases; in 1 case, the lesion was situated in heterotopic pancreatic tissue in the jejunum. Neutrophil infiltration of pancreatic tissue was also observed. In 50% of chronic pancreatitis cases, gallbladder (3) or pancreatic duct (2) calculi were obsered; acute duodenal ulceration was seen in 1 case with pancreatic calculi. Microscopic examination revealed fibrosis between pancreas acini and areas of mononuclear infiltration; squamous metaplasia of the lining cells was observed in some cases. Yellowish white opaque areas were seen in the vicinity of the pancreas, in the omentumm and in the mesentery in subacute pancreatitis patients and those with pancreatitis wit fat necrosis; there was no extra-abdominal fat necrosis. Microscopic examination revealed a network of fatty tissue filled with granular material staining reddish-pink in the central portion and basophilic in the peripheral areas. In 15 cases, infection and obstruction were the chief etiological factors; metabolic, toxic, chemical, traumatic, vascular, and idiopathic factors were contributory in the remaining cases. There were 4 cases in which pancreatitis was associated with liver disease; 2 of these cases, with cirrhosis, also had peptic ulcers. No parathyroid lesions were found in the cases with calculi.

6429 RELATIONSHIP OF HYPERPARATHYROIDISM TO PANCREATITIS. *(E.)* Kelly, T. R. (Akron City Hosp., Ohio). *Arch Surg* 97(2):267-274, 1968.

The relation between hyperparathyroidism and pancreatitis was studied in 30 Fischer-344 male rats, 20 of which received i.m. transplants of 40 or 80 isologous parathyroid glands. The third group underwant sham surgery with no transplantatio of parathyroid tissue and served as the controls. Groups 1 and developed hypercalcemia, the degree depending upon the amou of parathyroid tissue transplanted. The average serum amylase, which was approximately 1000 U preoperatively, rose to a height of about 2500 U, while group 3 showed no significant rise in serum amylase. While the trypsin level in group 3 was essentially normal, groups 1 and 2 also showed a rise in trypsin beginning on the first post-transplantation day and lasting an average of 5 days. Thereafter, the trypsin returned to normal levels. The pancreatic juice calcium rose, secondary to a similar change in serum calcium, and reached a maximum of approximately 7 mg/100 cc in rats receiving 80 parathyroid glands. The control rats showed no change in pancreatic juice calcium levels after surgery. The degree and frequency of pancreatic inflammation were directly proportional to the elevatio of calcium and trypsin in the pancreatic juice. It is concluded that hyperparathyroidism and pancreatitis are related through the following sequence: hyperparathyroidism, elevated serum calcium, increased pancreatic juice calcium, accelerated calcium-dependent conversion of trypsinogen to trypsin, and pancreatitis.

6430 STUDIES ON THE PANCREATITIS IN A MOUSE HEPATITIS VIRUS (MHV-3) INFECTION. *(E.)* Hirano, T. (Nagoya U. Sch. Med., Japan). *Nagoya J Med Sci* 30(3):319-325, 1967.

6431 ACUTE PANCREATITIS DUE TO OBSTRUCTI OF PANCREATIC DUCT BY A ROUND WOR *(E.)* Pandit, J. C. (J. J. Hosp., Bombay, India) and R. A. Irani. *Indian J Surg* 30(3):257-260, 1968.

6432 PANCREATITIS AND DISEASE OF THE BILE DUCTS. *(Ger.)* Herfort, K. (Res. Inst. Gastroent., Charles U., Prague, Czechoslovakia). *Deutsch Me J* 19(18):629-631, 1968.

6433 IS PANCREATITIS A COMMON OR A RARE DISEASE? *(Ger.)* Müller-Wieland, K. (1st Med. Clin., U. Hamburg, Germany). *Deutsch Med J* 19(18):631-634, 1968.

34 EXPERIMENTAL PANCREATITIS. *(Ger.)* Adlung, J. (1st Med. Clin., Med. Acad. Lubeck, rmany). *Deutsch Med J* 19(18):636-639, 1968.

35 POSSIBILITIES IN THE BIOCHEMICAL DIAGNOSIS OF PANCREATITIS. *(Ger.)* rentz, K. (1st Med. Clin., Med. Acad. Lübeck, Germany). *utsch Med J* 19(18):639-642, 1968.

36 PRINCIPLES FOR RESUSCITATION IN ACUTE PANCREATITIS. *(It.)* Faraci, R. (Sch. Anesth., Pavia, Italy), L. Gatta and A. Poggi. *Riv Gastroent* 19(6, ppl.):966-979, 1967.

37 BEHAVIOR OF THE BILIARY TRACT IN ACUTE PANCREATITIS. *(It.)* Farruggia, F. st. Gen. Clin. Surg., U. Bologna, Italy) and L. Zanella. *v Gastroent* 19(6, suppl.):775-788, 1967.

438 GASTROINTESTINAL BLEEDING IN PANCREATITIS. *(Ger.)* Leubner, H. (Med. Clin., . Innsbruck, Austria) and F. Gschnitzer. *Chirurg* 39(7): 12-315, 1968.

439 SURGICAL PROCEDURES IN CHRONIC PANCREATITIS. *(Ger.)* Vossschulte, K. (Surg. lin., U. Giessen, Germany) and E. Wagner. *Chirurg* 39(7): 07-312, 1968.

440 THE SYMPTOMATOLOGY, LABORATORY DIAGNOSIS AND CONSERVATIVE TREATENT OF CHRONIC PANCREATITIS. *(Ger.)* Rick, W. st Med. Clin., U. Düsseldorf, Germany). *Chirurg* 39(7): 01-306, 1968.

6441 PANCREATITIS AND MAGNESIUM DEFICIENCY. *(Ger.)* Holtmeier, H. J. (Med. Clin., U. Freiburg i.Br., Germany). *Schweiz Med Wschr* 98(43):1712-1716, 1968.

6442 THE MORPHOLOGY AND PATHOGENESIS OF CHRONIC PANCREATITIS. *(Ger.)* Haferkamp, O. (Inst. Path., U. Bonn, Germany). *Chirurg* 39(7):297-301, 1968.

6443 BEHAVIOR OF IMMUNOLOGICAL PREGNANCY TESTS IN PATIENTS WITH ACUTE PANCREATITIS. *(It.)* Nichelatti, T. (Circ. Hosp., Saronno, Varese, Italy) and G. Pagani. *Minerva Med* 59(74):3828-3829, 1968.

6444 THE CLINICAL CHARACTERISTICS AND TREATMENT OF ACUTE PANCREATITIS. *(It.)* Crespi, C. (L. A. Galmarini Hosp., Tradate, Varese, Italy), L. Marchi and A. Zanaboni. *Minerva Med* 59(74): 3830-3836, 1968.

6445 SUCCESSFUL TREATMENT OF PROBABLE PANCREATIC ENCEPHALOPATHY USING TRYPSIN INHIBITORS. *(Fr.)* Brion, S. (Foch Med.-Surg. Ctr., Paris, France), J. Aillet, J. Graveleau and L. Léonardon. *Presse Med* 76(1):9-12, 1968.

6446 RESULTS OF A TRIAL OF THE ENZYME INHIBITOR TRASYLOL IN ACUTE PANCREATITIS. *(Ger.)* Grozinger, K. H. (Surg. Clin., U. Heidelberg, Germany). *Fortschr Med* 86(7):281-283, 1968.

See also: 5740, 5854, 5856, 5971, 5977

LIVER AND BILIARY TRACT
GENERAL

6447 **LIVER RESECTION WITH THE CONTINUOUS WAVE CARBON DIOXIDE LASER: SOME EXPERIMENTAL OBSERVATIONS.** *(E.)* Mullins, F. (U. Louisville Sch. Med., Ky.), B. Jennings and L. McClusky. *Amer Surg* 34(10):717-722, 1968.

The simplicity, usefulness and safety of the continuous-wave CO_2 laser in removing multiple segments of liver (28-35% hepatectomy) was compared with that of electrosurgery in experiments on 12 Rhesus monkeys. Liver biopsies and serum enzyme determinations were carried out up to 1 yr postoperatively. In contrast to most other lasers with potential applications in medicine, the carbon dioxide laser is characterized by continuous power capability, high efficiency, ease of construction and safe operation for those using it. Continuous-wave laser energy at 10.6 microns offers several possible advantages in liver surgery: rapid bloodless dissection, no distant or continuing organ injury, and uncomplicated healing.

6448 **IS HYPERBILIRUBINEMIA PATHOLOGICAL?** *(Ger.)* Steiger, R. P. (Cantonal Hosp., Chur, Switzerland) and A. Roth. *Schweiz Med Wschr* 98(42):1675-1677, 1968.

Over a 17-day period, blood serum from 768 19-yr-old Swiss Army recruits was examined visually, and 36 samples suspected of having elevated bilirubin levels were studied colorimetrically by Jendrassik-Grof's method for total and direct bilirubin. Of these samples, 34 (4.4% of the total) had total bilirubin values of 1.40-3.00 mg% (1.1 mg% was taken as the upper limit of normal). In all cases it was the indirect fraction which was elevated. Only 2 of these recruits had histories of jaundice, and their SGOT and SGPT values were normal. Slightly elevated transaminase values were found in 1 case, but the recruit's history and other liver function tests were negative. A follow-up after 4 months showed that this subject's bilirubin and transaminase values had returned to normal. A possible explanation for high indirect bilirubin levels among a normal population might be that these subjects have Gilbert's familial cholemia.

6449 **EXTRACORPOREAL PERFUSION OF THE LIVER IN EXPERIMENTAL OBSTRUCTIVE JAUNDICE AND INCIPIENT COMA.** *(Pol.)* Bozek, P. (Acad. Med., Krakow, Poland) and M. Barczynski. *Pol Przegl Chir* 40(10):1065-1067, 1968.

Extracorporeal perfusion of an isolated healthy liver taken from a donor animal was used to treat 10 dogs with incipient hepatic coma and obstructive jaundice due to ligation and section of the common bile duct 2-3 weeks before. During the 2-hr perfusion, the BSP retention was only 5-10% compared to pretreatment values of 40-91%, the blood urea nitrogen increased by an average of 4 mg%, the nonprotein nitrogen increased by 8.6 mg%, the serum proteins decreased by 1.94 g%, the blood ammonia level decreased rapidly, and the liver produced 8-12 ml of bile. The serum bilirubin decreased from 2.9-37.8 mg% (average 15.7) to 0.76-14.9 mg% (average 7.7) during the 1st hr, with a smaller decrease during the 2nd hr. The results indicate that the isolated perfused liver performs its normal physiological functions.

6450 **SURGICAL TREATMENT OF PORTAL HYPERTENSION IN CHILDHOOD AND ADOLESCENCE: STUDY OF 17 CASES.** *(Por.)* Couto, D. Jr. (St. Francis Assissi Hosp., Rio de Janeiro, Brazil), S. Mattos Gomes and J. Aldrovando. *Hospital (Rio)* 74(2):473-488, 1968.

Operations were performed on 15 out of 17 Brazilian patients, aged 9-18 yr, with portal hypertension. Portal hypertension was caused by *Schistosoma mansoni* in 14 cases and by malformations in the portal circulation in 3. The most common symptoms were hypersplenism (14 cases), physical retardation (13 cases), digestive hemorrhages (8 cases), and cardiovascular disturbances (3 cases). Surgery was contraindicated in 1 patien because of severe pulmonary hypertension and cardiac decompensation and in 1 who had never had digestive hemorrhages and whose physical development was reasonably good. Surgica treatment consisted of splenectomy and splenorenal anastomosi in 7 cases, splenectomy alone or combined with other procedures in 5, portacaval anastomosis in 2, and mesenterocaval anastomosis in 2. One patient with pulmonary hypertension died of acute fulminating pulmonary edema 4 days after surger Postoperative complications included 1 case each of subphrenic abscess, pulmonary atelectasis, and phlebitis in the right leg. I attempting to make a splenorenal anastomosis one patient's renal vein proved too small and the severe bleeding which occurred necessitated removal of the right kidney; a simple splenectomy was performed. Because of frequent recurrences of bleeding this patient was later given a portacaval shunt with excellent results. Before surgery, all but 3 patients with schistosomiasis were treated with vermifuges. In 1 of these 3 patients portal filtration with extracorporeal circulation was used to remove 888 flukes; a splenorenal anastomosis was then performed.

6451 **LIVER-COPPER LEVELS IN LIVER DISEASE: STUDIES USING NEUTRON ACTIVATION ANALYSIS.** *(E.)* Smallwood, R. A. (Royal Free Hosp., Lond England), H. A. Williams, V. M. Rosenoer and S. Sherlock. *Lancet* 2(7582):1310-1313, 1968.

Liver-copper concentrations were estimated in 95 patients with liver disease by neutron activation analysis of liver samples (Todd *et al.*, *J Clin Path* 20, 1967). This method is simple, sensitive and specific, and requires very small needle-biopsy samples weighing only 2-3 mg. The normal range was found t be 15-55 μg copper per g dry liver. Levels greater than 250 μg/g were found only in Wilson's disease and long-standing intr hepatic and extrahepatic biliary obstruction. There was no diagnostic confusion between these two groups of patients, as those with biliary obstruction and high liver-copper levels had definite clinical and biochemical features of cholestasis. In patients with biliary obstruction the liver-copper level was related to the duration of symptoms. In other liver diseases (cirrhosis, alcoholic or toxic hepatitis, metastatic cancer, steato sis, Gilbert's disease), liver-copper levels were normal or moderately increased.

6452 **ETIOLOGICAL CORRELATIONS BETWEEN CHRONIC ANOXIC COR PULMONALE AND**

RONIC PROGRESSIVE LIVER DISEASE. *(It.)* Vancini, B. Maria della Misericordia Hosp., Cento, Bologna, Italy), Govoni, C. Pacchioni and F. Prospero. *Riv Gastroent* 6, Suppl.):1029-1040, 1967.

tudy was made of 21 patients with respiratory disease and ical and functional symptoms of liver disease, before and er treatment. This group included 14 (11 men, 3 women) h severe respiratory insufficiency caused by chronic obstruc- pulmonary emphysema associated with cor pulmonale and 5 men, 2 women) with chronic bronchitis, asthma, and early physema. Of the patients with severe respiratory insufficiency cor pulmonale, 13 had abnormal serum flocculation and/or bidity tests, 14 had positive BSP tests, and 7 had elevated PT values. Liver biopsies performed on 5 showed that 3 cirrhosis and 1 had fatty degeneration of the liver. Scinti- ms run with ^{198}Au showed evidence of diffuse fibrosis and rosis in all 5 patients examined. Of the 7 patients with mild piratory disease, several had abnormal serum flocculation /or turbidity tests, 3 had abnormal BSP tests, and 4 had htly elevated transaminase values. Liver biopsies showed is of degeneration and scintigrams were slightly abnormal he 2 patients studied. Treatment with cardiotonics, oxygen, diuretics produced some temporary improvement in the ilts of liver function tests among patients with respiratory ifficiency and cor pulmonale. This is attributed to improved ulation and reduced hypoxia in the liver cells. Despite these provements, the underlying disease remained unchanged. inges in liver function tests were reversible among patients h mild respiratory disease. From these findings and from the ories of these patients it is concluded that liver disease is esult of severe anoxia produced in the liver parenchyma by sodes of acute respiratory insufficiency rather than of cardiac ompensation since symptoms of liver disease were found in ients without cor pulmonale.

3 CRITICAL STUDY OF THE CURRENT THERAPY FOR SEVERE HEPATIC INSUFFICIENCY.

) Benhamou, J. P. (Beaujon Hosp., Clichy, Paris, France), Rueff and C. Sicot. *Rev Franc Etud Clin Biol* 13(7):651- , 1968.

^{2}-test was used to evaluate differences between the centage of recoveries among untreated patients with hepatic ure and patients treated with corticosteroids, peritoneal ysis and hemodialysis, exchange transfusions, perfusion of solated liver, and hyperbaric oxygen. The material included of the authors' own patients with hepatic encephalopathy and 250 reported in the literature. The number of patients ted by crossed circulation and liver perfusion was too small ermit a valid conclusion. No significant differences between ted and untreated patients were found for other forms of apy. No conclusion could be reached about coenzyme apy since the percentage of recoveries in one treated group higher than that in controls and the percentage of recover- n another treated group was not significantly different from in controls.

4 CHANGES IN THE SMALL VESSELS OF THE LIVER AFTER SOME OPERATIONS ON THE TONOMIC NERVOUS SYSTEM.

(Rus.) Iefimishin, N. S. odno Med. Inst., USSR). *Arkh Anat* 55(9):40-43, 1968.

Experimental ascites was induced in 41 dogs by constriction of the posterior vena cava under the diaphragm. The common hepatic artery was denervated in 21 of these animals, while in the other 20 the right splanchnic nerves were cut and the right lumbar sympathetic trunk resected near the first 2 sympathetic ganglia. Of the 21 dogs in the first group, 8 died; the 13 who survived were biopsied monthly for 1-5 months. Ascites resorbed in 7 dogs and persisted in 6. In the first few days after denervation of the common hepatic artery portal circulation into the liver increased significantly as manifested by dilatation of the interlobular veins. In most cases there was also improvement of flow from capillaries in the sinusoids to the hepatic veins. Acute disturbances in hepatic circulation and hemorrhagic necrosis developed in 3 animals in which the vena cava had been more tightly constricted. Two months after surgery hepatic edema was reduced, indicating that lymph flow from the liver had improved. Of the second group of 20 animals, 10 died shortly after surgery. Ascites regressed in 9 of the 10 survivors. Liver biopsies showed considerable constriction of the perivascular lymph vessels and of Disse's spaces and a reduction in perivascular edema. Improvement in lymph flow in the liver can either be ascribed to an increase in the flow of lymph out of the liver or to reduced aldosterone synthesis and increased aldosterone metabolism in the liver due to cutting of the right splanchnic nerve.

6455 ELECTRON MICROSCOPIC FINDINGS IN THE LIVER IN EXPERIMENTAL LISTERIOSIS.

(Ger.) Cossel, L. (Inst. Path. Karl Marx U., Leipzig, Germany), and P. F. Mahnke. *Beitr Path Anat* 138(1):65-95, 1968.

Histological and bacteriological studies were run on the livers, spleens, and peritoneal exudates from male white mice infected i.p. with a suspension of *Listeria monocytogenes.* Pure cultures of *L. monocytogenes* obtained from this material did not differ morphologically or biochemically from the initial strain. Granulomas produced by *Listeria* consisted primarily of mesenchymal cells (histiocytes) originating from endothelial or Kupffer's cells in the walls of the sinusoids. Blood monocytes may also be involved since the cytoplasmic ultrastructure of granuloma cells resembles that of mononuclear histiocytes; the nuclear structures, however, are not often found in this type of cell but resemble those in granulocytes. In most cases granuloma cells did not develop into typical large histiocytes because of their high metabolic requirements or the presence of bacterial toxins. The vacuoles formed around the bacteria may be the beginning of a defensive reaction by the cell in which membranes are formed around bacteria which have actively penetrated the cell. In contrast to *Listeria* grown on culture medium, these bacteria had no cell wall, inner cell wall, or intermediate layer. This may be due to their detachment and destruction by the cell. Epithelial cells in the granulomas were destroyed either by liquefactive degeneration or coagulative necrosis due to compression and isolation of the epithelium by proliferating granuloma cells and the resulting impairment of the blood supply. These findings indicate that primary damage to the hepatic parenchyma in listeriosis due to the hepatotropic effect of the bacteria is possible but very unlikely.

6456 BLOOD GROUP DISTRIBUTION IN 120 PATIENTS WITH PRIMARY LIVER CANCER AT DAKAR.

(Fr.) Sankalé, M. (Fac. Med. Pharm., Dakar,

Senegal), C. Divetain, M. Vessereau and B. Diop. *Path Biol (Paris)* 18(23/24):1071-1073, 1968.

A study of the A, B, O, and Rh blood groups in 120 African patients with primary liver cancer showed no association between this disease and any particular blood group. The blood group ratios were not significantly different from those of the general population in Senegal, and appeared to be about the same as those of the Ouolof group which constitutes the majority of the population in the Dakar region. There was also no relation between the blood group and the clinical findings, course of the disease, or appearance of metastases.

6457 CHANGES IN HEPATIC MICROCIRCULATION FOLLOWING SOME OPERATIONS ON THE SYMPATHETIC NERVOUS SYSTEM. *(Rus.)* Iefimishin, N. S. (Grodno Med. Inst., USSR). *Arkh Anat* 55(9):40-43, 1968.

In 21 dogs with experimental ascites produced by constriction of the posterior vena cava above the diaphragm, repeated biopsies and pressure measurements following denervation of the common hepatic artery revealed plethora of the interlobular veins and a drop in portal venous pressure. There was also constriction of the perivascular lymphatic vessels, indicating improvement in hepatic circulation, but dilatation of the Disse spaces persisted. Hemorrhagic necrosis due to marked dilatation of the sinusoidal capillaries was seen in 3 dogs, and a total of 8 died postoperatively. Among 13 survivors observed for 1-5 months, ascites disappeared in 7 and persisted in 6. In 20 other dogs with experimental ascites, section of the right splanchnic nerves and resection of the right lumbar sympathetic trunk in the area of the first two sympathetic ganglia was followed by constriction of both the perivascular lymphatic vessels and the Disse spaces. Initial plethora in the interlobular, central and sublobular veins decreased after a few days. Ten of the dogs died postoperatively, but ascites disappeared in 9 of the 10 survivors. Operations of this type have been suggested for the treatment of ascites and portal hypertension in man.

6458 THORACIC COMPLICATIONS OF AMEBIC LIVER ABSCESSES: REPORT OF 150 CASES. *(Sp.)* Valdez O., S. (Natl. Med. Ctr., Mexican Inst. Soc. Security, Mexico City), C. Ibarra P., O. Corrales G. and R. Farias C. *Prensa Med Mex* 33(9/10):316-323, 1968.

The 150 patients (122 male and 28 female) treated for thoracic complications of amebic liver abscesses during 1961-66 represented 78.1% of the 192 patients with amebic liver abscess and 1.9% of all patients admitted during this period. Complications encountered included bronchohepatic (67) and pleurohepatic (36) fistulas, drainage into both the pleura and the bronchi (20), pleural effusion due to spread of the abscess (26), and pericardial effusion (1 case); the bronchi of the right lower lobe were the most commonly involved. The principal symptoms were chest pain, cough, dyspnea, chocolate-colored vomit, hemoptysis, fever and general malaise. Diagnostic procedures found to be valuable included pneumoperitoneum, bronchography, hepatopneumography, scintillation scanning, and injection of contrast media into the hepatic abscess. Diagnosis was uncertain in about 20%, who were misdiagnosed as having pneumonia, tuberculosis, pleurisy or lung cancer. Treatment with emetine (142 cases), chloroquine (132 cases) and/or broad-spectrum antibiotics (117 cases) was given before or in conjunction with surgery to 84 patients, while 66 were treated by drugs alone; some patients required multiple operations (total of 148 procedures). A final cure was obtained in 33 cases (20 medical and 13 medicosurgical), while 92 improved and 15 died (2 treated medically, 13 treated surgically, and including 13 with bronchohepatic or pleurohepatic fistulas). The most important long-term sequellae were pachypleuritis (25 cases) and bronchiectasis (9 cases).

6459 CHANGES IN THE COMPOSITION OF BLOOD GASES IN THE PORTAL AND HEPATIC VEINS IN TOXIC HEPATITIS. *(Rus.)* Karimov, Kh. Ia. (Tashkent Med. Inst., USSR) and V. A. Akovbian. *Pat Fiz Eksp Ter* 12(5):58-60, 1968.

Van Slyke's method was used to determine the gas composition of blood from the femoral artery and vein and the portal and hepatic veins in 10 dogs with toxic hepatitis induced by i.p. or i.v. injection of 100-150 ml/kg heliotrine. Within 24-48 hr after administration of heliotrine, there was an increase in the O_2 concentration in the femoral vein resulting in a decrease in the arteriovenous difference and in the percentage of O_2 utilized. Within 48 hr after injection of heliotrine the O_2 concentration in the portal and hepatic veins was about the same and there was a decrease in the O_2 differential between the hepatic artery and vein. The gas composition of blood from the femoral artery and vein gradually returned to normal on the 3rd-4th day, while changes in blood flowing in and out of the liver became more pronounced. Measurements of portal blood flow showed that heliotrine reduced the flow rate by 1.5-2 sec. Dogs died 10-15 days after administration of heliotrine, apparently of functional liver failure. These findings indicate that hypoxia, apparently histotoxic in nature, developed in the liver tissue as a result of heliotrine poisoning.

6460 LIVER DISEASE IN NARCOTIC ADDICTS. I. THE ROLE OF THE DRUG. *(E.)* Gorodetzky, C. W. (Addict. Res. Ctr., Natl. Inst. Mental Health, Lexington, Ky.), J. D. Sapira, D. R. Jasinski and W. R. Martin. *Clin Pharmacol Ther* 9(6):720-724, 1968.

The effect of long-term high doses of morphine on liver function in man was evaluated by performing liver function tests on 20 male volunteers (16 Negro and 4 Caucasian, 26-39 yr old) before, during, and after the long-term administration of morphine. Prior to the test, all volunteers had been withdrawn from opiates for at least 6 months. None showed significant evidence of hepatic, cardiovascular, gastrointestinal, or pancreaticobiliary abnormalities. Morphine was administered s.c. at a daily stabilization dose of 240 mg for 6-8 months, after which it was gradually withdrawn over several weeks or methadone was substituted. Observations were continued for at least 1-2 months after drug withdrawal. No significant differences in the SGOT, SGPT, thymol turbidity or cephalin flocculation were found during the entire test period. It is concluded that the high incidence of abnormal liver function tests observed in morphine and heroin addicts is not due to a direct hepatotoxic effect of the drug.

LIVER AND BILIARY TRACT

461 ICTEROGENIC HEPATITIS PROGRESSING TOWARDS CIRRHOSIS WITH ANTINUCLEAR ANTIBODIES (LUPOID HEPATITIS?). *(Fr.)* Vachon, A., R. Téte, J, J. Gauthier, H. Chapuy and M. Silie. *Arch Franc Mal Appar Dig* 57(8/9):657-676, 1968.

Reports are presented on 6 patients (5 females and 1 male, aged 14-80 yr). Four had confirmed viral hepatitis, 1 had cholestatic hepatitis which was probably viral in origin, and 1 had primary biliary cirrhosis. All 6 patients had antinuclear antibodies in titers ranging from 1:64-1:8000. All 4 patients with confirmed viral hepatitis had hyperproteinemia and hypergammaglobulinemia. The test for LE cells was negative in 5 cases and doubtful in 1. Most patients had arthralgia, skin symptoms (urticaria or purpura), and fever. One patinet had transitory albuminuria, pulmonary congestion, and adenopathies. Jaundice recurred in 3 of the 4 patients with confirmed viral hepatitis 1, 5, and 6 months after the initial episode. Another patient had jaundice for more than 1.5 months. In 5 cases the cirrhosis and inflammatory sclerogenic hepatitis progressed rapidly. Three patients died, the course was arrested in 2, and 1 improved after chloraminophene therapy. It is believed that an autoimmune mechanism is involved in the disease process seen in these patients.

6462 ETIOLOGICAL PROBLEMS OF SYMPLASMATIC HEPATITIS IN THE INFANT. *(Fr.)* Perrimond, H. (Conception Hosp., Marseille, France), G. Lebreuil, B. Granjon, C. Raybaud, J. Coignet and A. Orsini. *Pediatrie* 23(6):631-647, 1968.

Case reports are presented for 6 infants (5 females and 1 male) with plasmodial transformation of the hepatocytes (neonatal giant-cell hepatitis). Histological confirmation was made at autopsy (5 cases) or surgery (1 case) at the age of 3 weeks-11 months. The etiology, established in 5 cases, included syphilis, toxoplasmosis, cytomegalic inclusion disease (2 cases, including 1 in a patient with Down's syndrome), and complete atresia of the bile ducts. The clinical course varied greatly. The 2 patients with cytomegalic inclusion disease had severe neonatal jaundice, 2 had obstructive jaundice, and 1 developed jaundice several hours before death. The other 2 patients remained anicteric. Since there is such a variety of etiological factors it is suggested that symplasmatic hepatitis be considered as a histological entity only.

6463 CHOLELITHIASIS AND HYDATED CYSTS OF THE LIVER: EXPERIMENTAL RESEARCH AND CLINICAL STUDIES. *(Fr.)* Seror, J. (Mustapha Hosp., Algiers, Algeria), B. Mentouri, K. Daoud and C. Vedrenne. *Ann Chir* 22(21/22):1269-1277, 1968.

Cholelithiasis was induced in 3 dogs by placing noninfected hydatid cysts, removed from patients with echinococcosis, in their gallbladders. In 1 case, however, the cystic duct was ligated and suppurative cholecystitis due to *E. coli* was probably the major factor involved in the production of cholelithiasis. An unsuccessful attempt was made to produce cholelithiasis with fluid and gravel from a hydatid cyst. Cholelithiasis was associated with hydatid cysts of the liver in 28 operated patients. In 58.1% of these patients the cyst was located in the juxtavesicular region compared to 21% of 202 patients with hydatid cysts alone. The gallbladder was not infected in most cases. The most frequently found lesions in the biliary tract were dilatation of the common bile duct, angiocholitis, and sclerotic inflammation of Oddi's sphincter. Pancreatitis was present in 1 case. In 42% of the cases the cyst opened into the bile ducts. One patient who had been operated on for hydatid cysts later developed cholelithiasis. The postoperative mortality among patients with hydatid cysts and cholelithiasis was 14.2%, just a little higher than among those with hydatid cysts alone.

6464 HEMIHEPATECTOMIES: STATISTICS ON 28 PERSONAL CASES. *(Fr.)* Mercadier, M. (La Pitié Hosp., Paris, France), D. Melliére and J. P. Clot. *Ann Chir* 22(21/22):1247-1254, 1968.

Hemihepatectomies were performed on 18 patients with benigh lesions of the liver (1 with posttraumatic hemobilia, 8 with hydatid cysts, 6 with hemangiomas, 2 with adenomas, and 1 with congenital dilatation of the intrahepatic biliary tract) and 10 with malignant neoplasms (3 with primary cancer of the liver, 4 with metastatic cancers, and 3 with lymphosarcomas infiltrating from the stomach). Only 1 patient died in shock immediately after surgery. This death is attributed to accidental ligation of the main hepatic artery artery rather than its right branch. Long-term results appear to be good in all patients with benign lesions of the liver. None of the patients with secondary cancer survived longer than 18 months after surgery. Of the 3 patients with primary cancer of the liver, 1 died 3.5 yr after surgery and 2 others are still alive 2 and 2.5 yr after surgery (1 now has cerebral metastases). Thorough presurgical exploration, particularly selective angiography, is recommended so that possible arterial anomalies can be detected and surgical complications avoided. A large thoracolaparotomy on the right makes it possible to follow the vena cava above and below the liver.

6465 PSEUDOSURGICAL JAUNDICE IN THE ADULT: THE DIAGNOSTIC VALUE OF LIVER BIOPSY AND THE ROLE AND DANGER OF EXPLORATORY LAPAROTOMY. *(Fr.)* Loup, P. (Inst. Path. Anat., U. Lausanne, Switzerland). *Schweiz Med Wschr* 98(44):1751-1756, 1968.

Among 46 patients subjected to laparotomy because of apparently "surgical" jaundice (including 3 operated on for choledocholithiasis and 5 others with cholelithiasis), 80% were shown by liver biopsy to have hepatic disease (32 cases of viral hepatitis, 5 of drug-induced hepatitis, 3 of alcoholic hepatitis, 3 of Dubin-Johnson syndrome, and 3 of idiopathic cholestasis). Five of these patients died immediately postoperatively, 2 died 5-10 weeks later, and 1 went into precoma, all 8 having severe viral (6) or alcoholic (2) hepatitis. In a larger group of 107 patients with jaundice (66 intra- and 41 extrahepatic), the histological diagnosis was correct in 69.1%, doubtful in 29%, and incorrect in 1.9% (2 cases, one of chlorpromazine jaundice diagnosed as "probably extrahepatic" and one of choledocholithiasis diagnosed as "probably drug-induced"). On the basis of these results, it is urged that every case of jaundice in which the etiology cannot be determined by exhaustive clinical and biochemical studies should be subjected to liver biopsy before considering surgery.

6466 ENZYME STUDIES IN DOGS WITH EXTRAHEPATIC BILIARY OBSTRUCTION. *(E.)* Aronsen, K. F. (Malmö Gen. Hosp., Sweden), I. Hägerstrand and J. G. Nordén. *Scand J Gastroent* 3(4):355-368, 1968.

A histochemical study of 7 liver enzymes was undertaken during and, after release of, complete extrahepatic biliary obstruction in 8 mongrel dogs. The enzymes studied were succinic dehydrogenase, nonspecific esterase, monoamine oxidase, acid phosphatase, alkaline phosphatase, adenosine triphosphatase and gamma-glutamyl transpeptidase. The mitochondrial enzymes, succinic dehydrogenase and monoamine oxidase, showed no change except for a decrease in fatty degenerated cells centrilobularly after a fairly long period of biliary obstruction. The only change found in the microsomal enzyme nonspecific esterase was an increase in some hepatic cells near regions of bile pigment accumulation. The lysosomal enzyme, acid phosphatase, increased first in hepatic cells and Kupffer cells and later appeared in round bodies near areas of bile pigment accumulation. The alkaline phosphatase and adenosine triphosphatase showed obvious and partly similar changes in their sinusoidal and canalicular patterns, and staining for these enzymes resulted in the appearance of knotty, irregular canaliculi. The enzyme gamma-glutamyl transpeptidase, which is also localized to the canaliculi, decreased when biliary obstruction was prolonged. A comparison of the histochemical and serum enzyme levels showed that alkaline phosphatase increased in the hepatic tissue before it did in the serum. The histochemically demonstrated decrease of gamma-glutamyl transpeptidase in the hepatic tissue after about 3 weeks of biliary stasis occurred simultaneously with a decrease in serum glutamyl transpeptidase.

6467 PORTAL PRESSURE READINGS AS AN INDICATION FOR SURGERY IN BILHARZIAL PORTAL HYPERTENSION. *(E.)* Bekhit, F. (Fac. Med., Cairo U., Egypt) and L. El Sayed. *J Egypt Med Ass* 51(2/3): 130-154, 1968.

The portal pressure was studied preoperatively, during surgery, and postoperatively in 43 cases of portal hypertension due to bilharzial hepatospenomegaly. The results showed that among the physiological factors affecting portal pressure are the sympathetic nervous system, chemical agents and humoral factors, respiratory movements of the chest and diaphragm, variations in intraabdominal pressure, and intrinsic liver mechanisms. The pathological factors affecting portal pressure are the main changes leading to liver cirrhosis. There was a lack of correlation however, between the portal pressure and the severity of the pathological changes on the clinical picture. While information as to the portal pressure is of practical value, its clinical interpretation must be made in the light of these shortcomings. Another prerequisite to an exchange of ideas about protal pressure levels and their clinical significance is a uniform method of portal pressure determination. Methods of measuring portal pressure include the direct method and various indirect methods such as percutaneous intrasplenic pressure measurement, measurement of portal pressure by liver puncture, and occlusive hepatic vein catheterization. The pitfalls and inaccuracies in the recording of portal pressure, the effects of anesthesia and opening of the peritoneal cavity on portal pressure recordings, the relation between the size of the spleen and portal hypertension, and the relation between portal pressure readings and esophageal varices are discussed. It is emphasized that esophageal varices should be treated by some type of shunt procedure regardless of the portal pressure.

6468 PITUITARY FUNCTION IN HAEMOCHROMATOSIS. *(E.)* Stocks, A. E. (Royal Melbourne Hosp., Australia) and F. I. R. Martin. *Amer J Med* 45(6): 839-845, 1968.

Pituitary and gonadal function were evaluated in 14 men and one woman with hemochromatosis, ranging in age from 34 to 74 yr. Eleven had diabetes mellitus and 9 required insulin. Ten of the 14 men had a definite history of depressed sexual function. Upon clinical examination, 3 men showed severe testicular atrophy, 5 had moderate atrophy, and in 6 the testes appeared normal. Eight men showed a female distribution of pubic hair and 4 had reduced beard growth, but none had gynecomastia. In all 15 cases the liver biopsy was typical of hemochromatosis. Tests of the output of growth hormone, corticotrophin, urinary gonadotrophin, plasma luteinizing hormone and thyrotropic hormone showed that only 6 of the 15 patients had no evidence of pituitary depression. While the results obtained suggest that pituitary dysfunction is common in hemochromatosis and is probably due to iron deposition in the anterior pituitary, the degree of pituitary failure was not related to either the severity of liver disease or the degree of abnormality in either iron or estrogen metabolism.

6469 NATURAL HISTORY OF ACTIVE CHRONIC HEPATITIS. II. PATHOLOGY, PATHOGENESIS AND CLINICO-PATHOLOGICAL CORRELATION. *(E.)* Mistilis, S. P. (Royal Prince Alfred Hosp., Sydney, Australia). *Aust Ann Med* 17(4):277-288, 1968.

In order to clarify the natural history of active chronic hepatitis in terms of its pathology, 150 liver biopsy specimens from 82 patients with this disorder were studied. The diagnostic and prognostic importance of liver biopsy was assessed and the pathological changes were compared with findings in clinically related disorders such as persistent hepatitis, cryptogenic cirrhosis, primary biliary cirrhosis, acute infectious hepatitis, and pericholangitis. An attempt was made to assess the relation between the clinical manifestations and severity of active chronic hepatitis on the one hand, and the type and severity of the histological changes on the other. Biopsy changes in treated patients were also compared with changes in those who did not receive corticosteroids, azathioprine or 6-mercaptopurine. The findings indicated that active chronic hepatitis may be diagnosed from aspiration biopsy specimens, which show the changes of both acute and chronic perilobular hepatitis. The changes appear to be specific and biopsy often provides the only means of diagnosing the disorder. The course of the disease is highly variable, characterized by repeated episodes of necrosis. Necrosis of entire consecutive lobules may account for clinical episodes of spontaneous hepatic coma. Patients with evidence of lobular or submassive necrosis have a subsequent poor prognosis. All patients eventually develop cirrhosis, but over half have histologically active disease during the entire course. In no case could reversibility of the process be demonstrated. There is no justification for the previous terminology which divided the disease into sub-groups, since these appear to be merely clinical variants of the same pathological process.

470 INDUCTION OF CHRONIC ACTIVE HEPATITIS BY HETEROLOGOUS SOLUBLE LIVER PROTEINS. *(Ger.)* Kössling, F. K. (Inst. Path., Johannes Gutenberg J., Mainz, Germany) and K. H. Meyerzum Büschenfelde. *'irchow Arch Path Anat* 345(4):365-376, 1968.

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472 EFFECT OF INTRAINTESTINAL OXYGEN THERAPY ON THE FUNCTIONAL STATE OF HE LIVER IN PATIENTS WITH ATHEROSCLEROSIS. *(Rus.)* Golovtsev, Iu. N. (Vinnitsa Med. Inst., USSR). *Vrach Delo* 0(7):12-15, 1968.

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474 THE PATHOGENESIS AND CLINICAL CHARACTERISTICS OF "ANTEROGRADE" PORTAL IYPERTENSION. *(Sp.)* Fernández-Nogués, F. (Fac. Med., Barcelona, Spain). *Med Clin (Barcelona)* 51(1):60-64, 1968.

475 EXCHANGE TRANSFUSION IN THE TREATMENT OF HEPATIC COMA. *(Sp.)* Bruguera, 1. (Fac. Med., Barcelona, Spain). *Med Clin (Barcelona)* 51(1):50-55, 1968.

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481 VARIATIONS IN THE ACTIVITY OF SOME ENZYMES IN THE HEPATIC LYMPH IN CUTE EXPERIMENTAL CHOLESTASIS. *(It.)* Rosa, G. (Inst. Gen. Clin. Surg., U. Padua, Italy), G. Segato, G. Mantovani-Orsetti and O. Fontanin. *Acta Chir Ital* 24(2): 177-192, 1968.

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6499 CYSTIC FIBROMATOSIS OF THE LIVER: DESCRIPTION OF A CASE AND REFLECTIONS ON ITS CLASSIFICATION AND CLINICAL CHARACTERISTICS. *(It.)* Villa, L. (Inst. Gen. Clin. Med., U. Milan, Italy), S. Eridani and R. Esposito. *Arch Ital Mal Appar Dig* 34(4):331-350, 1967.

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See also: 5693, 5814, 5836, 5837, 5842, 5843, 5866, 5869, 5870, 5874, 5875, 5880, 5881, 5882, 5889, 5890, 5892 5893, 5898, 5899, 5901, 5906, 5907, 5913, 5916, 5918, 5919, 5924, 5925, 5931, 5934, 5937, 5941, 5944, 5947, 5948, 5950, 5951, 5952, 5954, 5955, 5959, 5960, 5965, 5966, 5969, 5970, 5972, 5974, 5979, 6256, 6421

6531 **STUDIES ON THE MECHANISM OF FREEZING DAMAGE TO THE MOUSE LIVER USING A MITOCHONDRIAL ENZYME ASSAY. I. TEMPORAL LOCALIZATION OF THE INJURY PHASE DURING SLOW FREEZING.** *(E.)* Fishbein, W. N. (Armed Forces Inst. Path., Washington, D. C.) and R. E. Stowell. *Cryobiology* 4(6):283-289, 1968.

Experiments with fresh liver slices (10 x 5 x 1 mm) from CAF_1 mice showed that the succinate-cytochrome c reductase system of mouse liver is sensitive to freeze-thaw damage produced by cooling liver slices to -25C at a rate of 1 degree/min. About half the enzymatic activity was lost during one freeze-thaw cycle, and this was independent of the temperature at which freezing occurred down to -16C. The degree of damage was the same whether the liver slices were super-cooled with sudden freezing or were gradually frozen at 0C; it was also unaffected by the choice of rapid or gradual thawing (1 degree/min) and by variations in the suspending medium. Damage to this enzyme complex coincided with the phase of ice crystallization, particularly with the final stage of this process near the end of the heat of crystallization curve.

6532 **POSTOPERATIVE CHANGES IN TRANSAMINASES IN CHOLECYSTECTOMIZED PATIENTS.** *(It.)* Rossi, R. (Surg. Clin., U. Milan, Italy) and G. Boselli. *Arch Ital Chir* 94(1):140-145, 1968.

SGPT activity was determined before, during, and for 10 days after surgery in 20 patients with cholelithiasis who had simple cholecystectomies and in 20 patients with duodenal ulcers who had gastric resections. Before surgery all patients had normal SGPT values. Elevated SGPT values were found during or after surgery in 35 of the 40 cases. In 8 patients with duodenal ulcers and 1 with cholelithiasis, elevations were seen only during surgery and are attributed to the effect of the anesthetic on the liver. Increased SGPT values appearing only some time after surgery were found in 4 patients after gastric resection and 5 after cholecystectomy, while both early and late increases occurred in 5 patients subjected to gastric resection and 12 subjected to cholecystectomy. The late postoperative elevations are considered to be biochemical manifestations of hepatocellular trauma caused by surgery.

6533 **COMPARISON OF SOME INDICES OF PROTEIN METABOLISM WITH THE PATHOMORPHOLOGICAL CHANGES SEEN IN THE LIVER TISSUE DURING EXPERIMENTAL POISONING WITH DIPHTHERIA TOXIN.** *(Rus.)* Utiumova-Malova, A. V. (Astrakhan Med. Inst., USSR), A. V. Afanas'ieva and M. S. Brumshtein. *Biull Eksp Biol Med* 66(10):29-31, 1968.

The earliest detectable change in the plasma proteins following administration of diphtheria toxin (0.5 μl/kg i.v.) to dogs was a decrease in β-globulins and increase in γ-globulins, occurring 3-7 days after the injection. During this period, the histological appearance of the liver parenchyma gradually changed from a general picture of dystrophy and hepatocellular necrosis to a pattern characterized by increasing accumulation of leukocytes and glycogen-containing Kupffer cells. The first appearance of embryospecific α-globulins in the serum coincided with the development of proliferative changes in the liver, with increased numbers of leukocytes and glycogen-containing reticuloendothelial cells; these changes became increasingly pronounced 8-17 days after administration of diphtheria toxin. The decrease in the serum albumin level seen more than 2 weeks after injection was accompanied by periportal infiltration by lymphoid elements and fibroblasts, and can be looked upon as a reflection of early cirrhotic changes in the liver parenchyma.

6534 **AN ELECTRON MICROSCOPIC AND BIOCHEMICAL STUDY OF THE CHANGES IN ENDOPLASMIC RETICULUM INDUCED IN RAT LIVER BY 3'-METHYL-4-DIMETHYLAMINOAZOBENZENE AND PHENOBARBITAL.** *(Jap.)* Konno, Y. (Sapporo Med. Coll., Japan). *Sapporo Med J* 33(3):145-162, 1968.

Electron microscopy and histochemical studies (Epon 812, hematoxylin-eosin and PAS-toluidine blue) of liver tissue obtained from rats fed 3'-methyl-4-dimethylaminoazobenzene (MDAB; 0.06% in the diet for 14 days) or given phenobarbital (80 mg/kg/day for 6 days) confirmed that administration of carcinogenic dyes is followed by proliferation of the smooth endoplasmic reticulum in the hepatocytes accompanied by depletion of glycogen. Although the gross changes after phenobarbital administration were similar, the smooth endoplasmic reticulum in this case had a finely-branched tubular structure and a higher density than the compact, fine-vesicular, smooth endoplasmic reticulum seen after MDAB. Administration of MDAB was also followed by marked semicircular pattern formation, fragmentation and detachment of ribosomes in the rough endoplasmic reticulum, while after phenobarbital these changes were much less pronounced and after 6 days' starvation there was only a slight increase in morphologically normal rough endoplasmic reticulum. On the other hand, the activity of drug-metabolizing enzymes in the liver tissue was increased only by phenobarbital, not by MDAB.

6535 **INHIBITION OF DEOXYRIBONUCLEIC ACID SYNTHESIS IN REGENERATING RAT LIVER BY BERYLLIUM.** *(E.)* Witschi, H. (U. Cincinnati Coll. Med., Ohio). *Lab Invest* 19(1):67-70, 1968.

The effect of beryllium binding on the metabolic activity of rat liver nuclei was studied in male Cox rats (175-225 g) or Porton rats given 83 μmoles/kg of $BeSO_4 \cdot 4H_2O$ in distilled water via the tail vein. Control animals received an equal amount of saline. Studies in animals give 1 μC uniformly labeled L-leucine-^{14}C or 2 μC orotic acid-6-^{14}C i.p. 30 min before sacrifice showed that beryllium, given 5 or 28 hr before sacrifice, had no effect on either the incorporation of leucine into liver protein or the incorporation of orotic acid into liver RNA, neither in intact nor in partially hepatectomized rats. The incorporation of thymidine into DNA (1.5 μC thymidine-2-^{14}C i.p. 2 hr before sacrifice) was significantly inhibited, however. This concentration of beryllium (0.33 μmoles/g) in the liver neither interfered with the restoration of liver mass nor produced liver necrosis. When increasing doses of beryllium (7.5, 15, 30, 60, and 100 μmoles/kg) were given to hepatectomized rats 20 hr before sacrifice (28 hr after partial hepatectomy), the degree of inhibition of thymidine incorporated into DNA was found to be directly proportional to the beryllium concentration in the liver. Although it is difficult to relate the results of these experiments directly to the necrogenic action of beryllium, they may give some clue as to the mechanism of the sarcogenic and carcinogenic properties of beryllium in experimental animals.

536 THE EFFECTS OF CARBON TETRACHLORIDE, ALLYL ALCOHOL AND ETHANOL ON THE FATTY ACID CONTENT OF THE LIVER IN RATS. *(Ger.)* Berg, G. (Inst. Path. Anat., U. Erlangen-Nuremberg, Germany), U. Troll, H. Breining and O. Strubelt. *Med Exp* 18(2):113-120, 1968.

Variable changes in fatty acid composition, depending on timing and dosage, were observed in the livers of female Wistar rats given toxic doses of either CCl_4 (0. 5 ml/kg i.m.), allyl alcohol (2% aqueous solution, 2 ml/kg by stomach tube), or ethanol (either as a 20% solution, 20-60 ml/kg by stomach tube, or 6-7 ml/kg/day in the drinking water for 14 days); the animals were sacrificed 24 hr after administration of the solvent. These biochemical changes appeared before the development of morphological alterations in the liver parenchyma and became more pronounced with the development of hepatic steatosis. The alterations in phospholipid composition (slight increase in monounsaturated fatty acids in the presence of nearly constant proportions of saturated and polyunsaturated fatty acids) were slight compared to those in the neutral fats and the cholesterol esters (relative increase in saturated and decrease in unsaturated fatty acids). In all cases, however, the most striking change was the decrease in polyolefins; their proportion decreased from 23.3% to 7.6-10.5% in the cholesterol esters after all solvents except allyl alcohol (which produced only a small decrease), and from 24.7% to 3.9-11.7% in the neutral fats after ethanol (with smaller decreases after the other solvents).

6537 NOVOBIOCIN-INDUCED HYPERBILIRUBINEMIA AND ITS REDUCTION BY PHENOBARBITAL PRETREATMENT. *(E.)* Harbison, R. D. (Coll. Med., U. Iowa, Iowa City) and J. L. Spratt. *Toxic Appl Pharmacol* 11(2):257-263, 1967.

A single dose of novobiocin (500 mg/kg s. c.) produced both direct and indirect hyperbilirubinemia in both male Sprague-Dawley rats and Swiss-Webster mice. The highest observed plasma concentrations of both direct and indirect bilirubin occurred 5 hr after novobiocin treatment in the mouse (0.39 and 0.57 mg%, resp.) and 18 hr after novobiocin treatment in the rat 0.29 and 1.82 mg%, resp.). The direct bilirubin returned to normal in 96 hr in the mouse and 24 hr in the rat. The indirect bilirubin returned to normal in 24 hr for both mouse and rat. In animals pretreated with phenobarbital (80 mg/kg s. c. every 24 hr for 3 days) prior to novobiocin treatment, both direct and indirect bilirubin concentrations were significantly lower in both animals, and the highest observed concentrations occurred earlier than in the animals treated with novobiocin alone.

6538 MODIFICATION OF CHRONIC CARBON TETRACHLORIDE HEPATIC INJURY BY N,N'-DIPHENYL-p-PHENYLENEDIAMINE. *(E.)* Hartman, A. D. (U. Tennessee Med. Units, Memphis), N. R. Di Luzio and M. L. Trumbull. *Exp Molec Path* 9(3):349-362, 1968.

The ability of the lipid antioxidant N,N'-diphenyl-p-phenylenediamine (DPPD) to prevent or modify the hepatic structural and functional alterations induced by chronic administration of CCl_4 was evaluated in male Sprague-Dawley rats. DPPD was administered i.p. at a dose of 60 mg/100 g body weight prior to and at biweekly intervals during the experiment. CCl_4 was administered s. c. in corn oil as a 50% solution at a dose of 0.3 ml/100 g 3 times a week. Control rats received corn oil alone. DPPD inhibited but did not prevent the marked elevation in hepatic triglyceride levels observed in CCl_4-treated rats at the 3-week period. At 2 and 3 months, DPPD had no effect on the CCl_4-induced increment in hepatic triglycerides. The plasma BSP concentration was significantly elevated in CCl_4-treated rats at the 3-week and 3-month periods. In contrast, the DPPD-treated rats which received CCl_4 manifested normal plasma BSP concentrations. Treatment with DPPD significantly delayed the progressive cirrhotic alterations observed in untreated rats receiving CCl_4. These findings reveal the potential therapeutic importance of antioxidants such as DPPD in the modification of chemical, structural, and functional cellular changes induced by certain hepatotoxic agents.

6539 A CONTRIBUTORY STUDY ON RELATIONSHIP BETWEEN LIVER AND ERYTHROPOIETIN. *(E.)* Yasuda, H. (Tokyo Med. Coll., Japan). *J Tokyo Med Coll* 26(3/4):439-451, 1968.

6540 EFFECT OF THIO-TEPA ON THE CONTENT OF NICOTINAMIDE COENZYMES IN RAT LIVER. *(Uk.)* Bidenko, Ie. D. (M. I. Pirogov Med. Inst., Odessa, USSR). *Ukr Biokhim Zh* 40(5):436-439, 1968.

6541 THE ADRENOCORTICOLYTIC ACTIVITY OF 7,12-DIMETHYLBENZ[a]ANTHRACENE IN THE RAT. II. EFFECTS OF ACUTE CCl_4 POISONING, PARTIAL HEPATECTOMY, AND A STEATOGENIC DIET ON THE SENSITIVITY OF THE ADRENAL CORTEX TO THE CARCINOGEN. *(It.)* Colafranceschi, M. (Inst. Anat. Path. Histol., U. Florence, Italy) and P. Tosi. *Arch De Vecchi Anat Pat* 50(3):681-695, 1967.

6542 AN EXPERIMENTAL STUDY ON THE HEPATOTOXICITY OF HALOTHAN. *(Hun.)* Jakab, T. (Budapest U. Med. Sci., Hungary), G. Somogyi, J. Gulyás, T. Tóth, E. Kisida and L. Szabó. *Kiserl Orvostud* 20(4):405-410, 1968.

6543 A HISTOCHEMICAL STUDY OF THE CHANGES IN LIVER GLYCOGEN DURING PHALLOIDIN POISONING. *(It.)* Vallini, R. (Inst. Pharmacol., U. Ferrara, Italy), G. Sarto and P. Faveri. *Arcisped S Anna Ferrara* 21(2/3):213-221, 1968.

6544 JAUNDICE DUE TO ORAL CONTRACEPTIVES: REPORT OF TWO PERSONAL CASES AND REVIEW OF THE LITERATURE. *(It.)* De Lalla, F. (Inst. Infect. Dis., U. Milan, Italy) and M. Moroni. *Policlinico [Prat]* 75(40):1289-1297, 1968.

6545 STUDIES ON LIVER DAMAGE DURING CARDIAC SURGERY, WITH PARTICULAR ATTENTION TO THE POSTTRANSFUSION HEPATITIS FOLLOWING EXTRACORPOREAL CIRCULATION. *(Jap.)* Tamiya, T. (Chiba U. Sch. Med., Japan), H. Koriyama, T.

Ito, H. Horibe, K. Ito, N. Sasaki, H. Kawakami, T. Nishizawa and A. Yamaura. *Jap J Thorac Surg* 21(10):742-747, 1968.

6546 ANALYSIS OF THE CONDITION OF THE LIVER PARENCHYMA IN CHRONIC ALCOHOLICS. *(Ser.)* Čandrlić, I. (Gen. Hosp., Osijek, Yugoslavia), A. Maglajlić and D. Kozmar. *Med Pregl* 21(1/2): 75-83, 1968.

6547 ALTERATIONS IN LIVER FUNCTION FOLLOWING SURGERY ON THE LUNGS. *(Bul.)* Klimentov, V. (Sci.-Res. Inst. Tuberc., Bulgaria), V. Nichev and M. Kosturkova. *Khirurgiia (Sofiia)* 21(4):348-356, 1968.

6548 CHANGES IN THE LIVER FOLLOWING INTRAVENOUS INJECTION OF TAUROLITHOCHOLIC ACID. *(It.)* Gasbarrini, G. (Inst. Spec. Med. Path., U. Bologna, Italy), E. Roda, A. De Vecchis, F. B. Bianchi, G. Peta and A. Venerato. *Arch Pat Clin Med* 44(3):241-252, 1968.

6549 EXPERIMENTAL CELLULAR INSUFFICIENCY OF THE LIVER. *(Fr.)* Sterin, D. *Gaz Med France* 75(27):5355-5362, 1968.

6550 THE BIOCHEMICAL MECHANISM OF ACTION OF ARSENIC. *(Ger.)* Minden, H. (Cent. Inst. Labor Med., Berlin-Lichtenberg, Germany) and R. Rothe. *Int Arch Gewerbepath* 24(4):359-366, 1968.

6551 EFFECT OF ENVIRONMENTAL CONDITIONS ON THE DEVELOPMENT OF THE CHICK EMBRYO: MODIFICATIONS IN THE INFRASTRUCTURE OF THE HEPATOCYTE OF THE CHICK EMBRYO INCUBATED AT UNUSUAL TEMPERATURES. *(Fr.)* Preda, V. (Ctr. Biol. Res., Acad. Sci., Cluj, Romania), M. Rusu, A. Petrescu and S. M. Dumitrescu. *Rev Roum Embryol Cytol* 5(1):3-10, 1968.

6552 CLINICAL AND BIOPSY STUDIES ON LIVER DAMAGE DUE TO Rh-INCOMPATIBILITY. *(Ger.)* Gmyrek, D. (Charité Pediat. Clin., Humboldt U., Berlin, Germany), B. Wohlgemuth and R. Weiland. *Z Kinderheilk* 103(4): 307-324, 1968.

See also: 5807, 5813, 5822, 5829, 5849, 5858, 5859, 6171, 6247

;53 **MICROFILARIAE IN MARMOSET MONKEYS
OF THE SPECIES *SANGUINUS (OEDIPOMIDAS)*
EDIPUS AND THEIR IMPORTANCE FOR EXPERIMENT-
LLY INDUCED HEPATITIS.** *(Ger.)* Lange, W. (Robert
och Inst., Berlin, Germany), J. Apodaca and H. Köhler.
bl Bakt [Orig] 208(1/2):192-201, 1968.

eripheral blood tests run on 149 marmosets, recently im-
orted from Colombia, showed that 101 (67.7%) were infested
ith microfilariae. These microfilariae were also found in
ood vessels of the liver, kidney, adrenals and lungs and in
mph nodes. Liver biopsies taken from 95 marmosets, 45 of
hich were infested with microfilariae, showed that 46 had
nall infiltrates of histiocytes, lymphocytes, neutrophilic gran-
ocytes, and a few plasma cells. These were found chiefly
ound blood vessels in the periportal fields. Only 29 of these
5 animals had positive blood tests for microfilariae, while 34
ıt of 47 marmosets without liver changes also had positive
ood tests. This indicates that microfilariae are probably not
sponsible for the liver infiltrations. The possible role of these
icrofilariae in the etiology of hepatitis was ruled out by in-
ction of 6 marmosets with pooled sera from 5 infested ani-
als. Over a 6-week observation period no significant changes
curred in the livers of injected animals and their SGOT and
GPT values remained within normal limits.

554 **HEPATITIS IN MONKEYS FOLLOWING
INOCULATION WITH ADENOVIRUSES ISO-
ATED FROM PATIENTS WITH VIRAL HEPATITIS. III.
EROLOGICAL INVESTIGATIONS.** *(Ger.)* Köhler, H.
obert Koch Inst., Berlin, Germany), W. Lange, J. Apodaca
d E. Eggert. *Zbl Bakt [Orig]* 208(1/2):201-218, 1968.

evated serum transaminase levels and abnormalities in liver
stology were found in a number of marmosets *(Sanguinus
dipus)* during quarantine. These tests were usually run after
tibiotic and sulfonamide prophylaxis and were associated
ith high drug doses. However, elevated transaminase values
ere also found in untreated animals. These findings eventually
rmalized in both groups of animals. Small periportal liver
filtrates present in many of the quarantined animals were
parently due to previous infections involving the liver. Other
armosets showed signs of toxic liver damage which rapidly
solved. Infections were induced in some of the animals by
n. injection of 1.0 ml of a 1:500 dilution of Deinhardt's
fectious hepatitis serum from monkeys. Some of these ani-
als had previously developed very high serum transaminase
lues after injections of normal monkey serum. Complement
xation tests for human adenovirus serotypes were positive
only 5 out of 56 sera from 28 noninfected animals, while all
ose infected by monkey passages and tested consistently
veloped complement fixing antibodies a rather long time after
fection. These antibodies were found even if the animals
d not have elevated transaminase values or morphological
er abnormalities. Positive complement fixation tests for
enoviruses were obtained in 6 of 8 marmosets injected with
patitis virus isolated from humans and grown in cell cultures,
en though none of the animals developed signs of hepatitis.
einhardt's infectious hepatitis serum from monkeys produced
fections in 3 of 4 animals. Two of the infected animals and
e noninfected animal developed complement fixing antibodies.
It is concluded that adenoviruses probably play a role in the etiology of human viral hepatitis.

6555 **ATTEMPTS TO ISOLATE VIRAL AGENTS FROM PATIENTS WITH INFECTIOUS HEPATITIS.** *(It.)* Albano, A. (Inst. Hyg., U. Milan, Italy), S. De Donato and A. Criscuolo. *Ann Sclavo* 9(6/II):920-926, 1967.

Samples of blood and stools were collected periodically from 14 patients, aged 6-19 yr, with a clinical and laboratory diagnosis of infectious hepatitis; 20% stool extracts were made in Hank's medium with lactalbumin for inoculation into tissue cultures and in 0.01 M sodium citrate (pH 8.5) for inoculation into embryonated chicken eggs. Samples were inoculated into embryonated chicken eggs; kidney cell cultures from hamsters, marmosets (*Sanguinus nigricollis*), and Vietnamese swine; rat myeloma cells; Serum Institute rabbit cornea cells; and baby hamster kidney cells by conventional techniques, after treatment with Freon 113, and with increasing dilution of the samples. All attempts at passaging the virus were unsuccessful. Signs of degeneration appeared in the 3rd-5th passage in about half of the cultures. Some sera in dilutions as high as 1:100 completely destroyed the cell layer during the first passage. In the next 3-5 passages diffuse granulations appeared in round fibroblast-like cells which then became detached from the glass. No plaques were ever found, but sometimes hemorrhagic lesions developed on the chorioallantoic membrane of eggs which had been inoculated with samples taken when the patients had enlarged livers and were jaundiced. However, these lesions could not be propagated indefinitely and were sometimes seen, but less frequently, on the membranes of eggs inoculated with material from healthy subjects.

6556 **VIRAL HEPATITIS IN SYDNEY: A REVIEW OF FATAL ILLNESSES IN A HOSPITAL SERIES.** *(E.)* Boughton, C. R. (Prince Henry Hosp., Sydney, Australia). *Med J Aust* 2(8):343-351, 1968.

During the period 1950-63, there were 55 fatalities among the 4341 patients admitted for viral hepatitis; the case-fatality rate was thus 12.7/1000, as compared to 1-4/1000 in western communities. Adding 13 fatal cases which occurred during 1964-66, 66 of the 68 fatalities were due to liver failure. Although the same number of males and females were admitted with viral hepatitis, 2.5 times as many females died from the disease. In the age group over 15 yr, 4 times as many females died from the disease, while among children under 15 yr, males predominated. Serum hepatitis accounted for a greater percentage of cases in the fatal group than in the nonfatal group. The fatal illness tended to be of longer duration in females over 30 yr than in the younger age groups. The most common complications during the fatal illness were hemorrhage, renal dysfunction, hypotension and sepsis. Severe acute liver atrophy occurred within 2-3 weeks of the onset of the clinical disease. Regeneration of liver parenchyma was seen in only 1 case. The combination of coma and a small liver during the course of an attack of acute viral hepatitis was of gave prognostic significance. The 2 recoveries in this series were probably due to spontaneous recovery aided by protein restriction and framycetin. Therapy was of no avail when the patient's state of consciousness did not respond to protein restriction and framycetin. Adrenal steroids, exchange transfusions and peritoneal dialysis were ineffective in altering the prognosis. The factors responsible for the fatal outcome in some cases of viral hepatitis are still not known.

6557 **A STUDY OF 15 PARAMETERS OF SERUM PROTEINS IN VIRAL HEPATITIS.** *(Fr.)* Sandor, G., B. Sureau, L. Martin, J. Berrod and R. Martin. *Bull Acad Nat Med (Paris)* 152(24/25):396-401, 1968.

Electrophoretic and quantitative immunochemical determinations of serum protein components were made on serum from 25 children and 7 adults with viral hepatitis, 90 subjects with a variety of severe and mild infections which did not involve the liver, 33 patients with hepatic cirrhosis, and 50 controls. In contrast to the findings in other infections, viral hepatitis was characterized by little or no change in the concentrations of serum albumin, α-globulins, and orosomucoid; an increase in the euglobulin/globulin ratio; a very significant increase in the γ-globulin/α-globulin ratio and R_3 fraction; and a tendency for IgM concentrations to be high. Much the same pattern was found for serum protein fractions in viral hepatitis and cirrhosis. However, in cirrhosis the albumin concentration was significantly lower and the γ-globulin and IgG concentrations and the γ-globulin/-α-globulin ratio were significantly higher. The distribution of R3 values was about the same in viral hepatitis and cirrhosis. These findings indicate that the reticuloendothelial system of the liver, rather than the hepatocyte, plays the major role in the synthesis of serum albumin.

6558 **PROSPECTIVE STUDY OF A RANDOMLY SELECTED GROUP OF PATIENTS CONVALESCING FROM VIRAL HEPATITIS TO ESTABLISH FACTORS FAVORING THE DEVELOPMENT OF CHRONIC HEPATITIS.** *(Fr.)* Lăzăresco, V. (N. G. Lupu Inst. Int. Med., Bucharest, Romania), C. Miron, M. Zamfiresco-Gheorghiu, V. Runcan, M. Voiculesco, M. Golăesco, V. Căruntu and L. Pereder. *Rev Roum Med Intern* 5(4):221-229, 1968.

A 2-3 yr prospective study was made of 310 patients recovering from viral hepatitis. Of these, 38 (12.3%) were lost to follow-up. Of the remaining 272 patients (136 men and 136 women), 32 (11.8%) developed chronic hepatitis and 5 (1.9%) developed confirmed cirrhosis. Significantly more men than women developed chronic hepatitis and cirrhosis. A small peak occurred in males and females aged 20-29 yr and a much larger peak in males at 50-59 yr. The chance of hepatitis becoming chronic was directly related to the duration of the disease. Hepatitis virus had been transmitted parenterally in 62.5% and p.o. in 37.5% of those later developing chronic hepatitis. Significantly more patients, particularly women, with chronic hepatitis had histories of syphilis, pulmonary tuberculosis, malaria, peptic ulcer, cholecystitis, appendicitis, alcoholism, diabetes mellitus, and allergies with gastrointestinal symptoms. Patients developing chronic hepatitis included more whose occupations involved physical labor and who had disregarded instructions about rest and diet. The B strain, identified by biochemical parameters, was implicated more often than the A strain as the etiological agent in patients developing chronic hepatitis.

6559 **OBSERVATIONS ON PROTHROMBIN-COMPLEX IN INFECTIVE HEPATITIS (PROTHROMBIN: FACTOR V AND FACTOR VII).** *(E.)* Tejwani, B. M., (M. G. M. Med. Coll., Indore, India), P. S. Vasavada and D. P. Mukherjee. *Antiseptic* 65(10):730-734, 1968.

In 50 patients with mild to moderately severe infectious hepatitis, a complete coagulogram both qualitative and quantitative, was constructed with special reference to the prothrombin complexes (prothrombin: Factor V and Factor VII). It was observed that 5 patients (10%) showed prothrombin deficiency, 4 (8%) had Factor V deficiency, 2 (4%) had Factor VII deficiency, 8 (16%) had both prothrombin and Factor V deficiency, 2 (4%) had both prothrombin and Factor VII deficiency, 3 (6%) had both Factor V and VII deficiency, and 16 (32%) had a deficiency of all 3 components.

6560 **VIRAL HEPATITIS IN CHILDHOOD WITH PARTICULAR ATTENTION TO ITS TREATMENT BY MEANS OF EMETINE.** *(Sp.)* Del Puerto, B. M. (Fac. Med. Sci., U. Buenos Aires, Argentina), J. C. Tato, A. Koltan, O. M. Bures, P. R. De Chieri, A. García, T. I. Escaray and B. Lorenzo. *Prensa Med Argent* 55(18):818-834, 1968.

6561 **ISOENZYMES OF SERUM ALDOLASE IN PATIENTS WITH INFECTIOUS HEPATITIS.** *(Ger.)* Dikov, A. (Oncol. Res. Int., Sofia, Bulgaria) and M. Romanov. *Z Ges Inn Med* 23(15):471-472, 1968.

6562 **AN EVALUATION OF SOME NEW DIAGNOSTIC TECHNIQUES AND MODERN METHODS OF TREATMENT FOR VIRAL HEPATITIS IN CHILDHOOD.** *(It.)* De Matteis, F. (S. Salvatore Munic. Hosp., L'Aquila, Italy). *Minerva Pediat* 20(30):1526-1542, 1968.

6563 **CHANGING CONCEPTS IN THE EPIDEMIOLOGY OF VIRAL HEPATITIS.** *(E.)* Koff, R. S. (Harvar Med. Sch., Boston, Mass.) and K. J. Isselbacher. *New Eng J Me* 278(25):1371-1380, 1968.

6564 **ELECTROCARDIOGRAPHIC CHANGES DURING CORTICOSTEROID (PREDNISOLONE) TREATMENT IN PATIENTS WITH INFECTIOUS HEPATITIS COMPLICATED BY CARDIOVASCULAR DISEASE WITH SIGNS OF CARDIAC AND CORONARY INSUFFICIENCY.** *(Rus.)* Argov, Iu. M. (S. P. Botkin Infect. Dis. Hosp., Leningrad USSR). *Ter Arkh* 40(7):25-30, 1968.

6565 **HEPATIC COMA CAUSED BY VIRAL HEPATITI AND TREATED SUCCESSFULLY WITH HYPERBARIC OXYGEN.** *(Fr.)* Dordain, M. (Charles-Nicolle Hosp., Rouen, France), G. Humbert, M. Robert, J. Leroy and P. Delaunay. *Sem Hop Paris* 44(43):2617-2622, 1968.

6566 **A CONTRIBUTION TO THE STUDY OF SEVER JAUNDICE OF PREGNANCY IN MOROCCO.** *(F* Delons, S. (St. Antoine Hosp., Paris, France), A. Berbich, R. Reynaud and P. Lebon. *Rev Medicochir Mal Foie* 43(3):117-130, 1968.

6567 **THE TREATMENT OF ACUTE HEPATITIS.** *(Ge* Siede, W. (1st Med. Clin., U. Frankfurt a.M., Germany). *Munchen Med Wschr* 110(36):2022-2026, 1968.

568 SERUM ISOAMYLASES IN ACUTE ICTEROGENIC HEPATITIS. *(It.)* Pisi, E. (Inst. Gen. lin. Med., U. Bologna, Italy), R. Di Feliciantonio, S. Ferri, . B. Bianchi and G. Bacci. *Arch Ital Mal Appar Dig* 4(5):487-491, 1967.

569 DIURESIS, NATRIURESIS AND ALDOSTERONURIA DUE TO ANGIOTENSIN II IN ATIENTS WITH ICTEROGENIC HEPATITIS. *(It.)* Zuin, . (Gen. Med. Clin., U. Padua, Italy), P. Spandri, G. Mazzei, . Cadrobbi, F. Binda and C. Dal Palu. *Arch Ital Mal Appar ig* 34(5):491-497, 1967.

570 A PLAN FOR STUDYING THE LONG-TERM SEQUELLAE OF VIRAL HEPATITIS. *(Pol.)* lagdzik, W. (State Inst. Hyg., Warsaw, Poland). *Przegl pidem* 22(3):395-401, 1968.

571 CHANGES IN HEPATIC RHEOGRAMS IN PATIENTS WITH CHRONIC, SLOWLY PRORESSING HEPATITIS FOLLOWING AN ATTACK OF OTKIN'S DISEASE. *(Rus.)* Ivanova-Tikhvinskaia, Ie. L. Yaroslavl Med. Inst., USSR), A. S. Loginov and M. E. asilevskii. *Ter Arkh* 40(7):45-48, 1968.

572 THE BEHAVIOR OF THE PLASMA KININOGEN LEVEL IN ACUTE HEPATITIS. *(Ger.)* Blumel, . (1st Surg. Clin., U. Vienna, Austria), A. Neumayr, L. eschl and H. Rettenbacher-Teubner. *Wien Klin Wschr* 0(42):778-781, 1968.

6573 DISEASES APPEARING IN ASSOCIATION WITH VIRAL HEPATITIS. *(It.)* Di Nola, F. (Amedeo di Savoia Infect. Dis. Hosp., Turin, Italy), M. Rapellini, L. Cerutti, A. Pasquale, M. L. Soranzo and W. Grillone. *Minerva Med* 59(83):4405-4417, 1968.

6574 PROBLEMS OF HEPATITIS WITH PARTICULAR ATTENTION TO VIRAL HEPATITIS. *(Ger.)* Gros, H. (Munic. Hosp., Saarbrucken, Germany). *Oeff Gesundh* 30(9):335-345, 1968.

6575 ACUTE APPENDICITIS COMPLICATING INFECTIOUS HEPATITIS. *(Ger.)* Geschke, H. (Distr. Hosp., Stassfurt, Germany). *Med Klin* 63(37):1463-1465, 1968.

6576 VIRAL HEPATITIS IN CHILDREN, WITH PARTICULAR ATTENTION TO ITS TREATMENT BY MEANS OF EMETINE. *(Sp.)* Del Puerto, B. M. (Prof. Dr. Pedrodro de Elizalde Pediat. Hosp., Buenos Aires, Argentina), J. C. Tato, A. Koltan, O. M. Bures, P. R. de Cheri, A. Garcia, T. L. Escaray and B. Lorenzo. *Galicia Clin* 40(10):829-838, 1968.

6577 PROTRACTED VIRAL HEPATITIS IN INFANCY: CASES OBSERVED AT THE INFECTIOUS DISEASE CLINIC IN GENOA DURING THE THREE-YEAR PERIOD 1965-67. *(It.)* Bassetti, D. (Infect. Dis. Clin., U. Genoa, Italy). *Minerva Pediat* 20(40):2081-2088, 1968.

See also: 5926

6578 EXPERIMENTAL STUDY OF INTRATHORACIC TRANSPOSITION OF THE SPLEEN IN THE TREATMENT OF PORTAL HYPERTENSION.

(Fr.) Gautier Benoit, C. (Surg. Clin. West, Lille, France), J. Caron, L. Corsin, J. Delhaye, Devulder and M. Ribet. *Lille Chir* 23(1/2):5-12, 1968.

In 13 adult dogs and 4 puppies, the spleen was transposed into the thoracic cavity and portal hypertension was produced by dissecting the trunk of the portal vein and stenosing the vein with a cellophane collar. A thoracicophrenic laparotomy was used in adult dogs and an abdominal approach in the puppies. Of the adult dogs, 1 died of a malignant cholangioma, 2 died of complications of general anesthesia, 3 died of mesenteric infarcts, and 3 died of infection before radiography could be performed. Of the 4 remaining dogs, 1 was checked for collateral circulation by splenoportography and 3 others by ileoportography 2.5, 6, 10, and 12 months after surgery. Except for the doubtful case checked by splenoportography, none of these adults showed evidence of developing collateral circulation from the hilum of the transposed spleen. Of the 4 puppies with portal hypertension and transposed spleens, 1 died immediately after surgery of a mesenteric infarct. Two of the 3 others, checked by ileoportography 12, 13, and 14 months after surgery, had developed collateral circulation from the hilum of the transposed spleen to rejoin the azygos vein either through intercostal veins or through the mediastinum. These findings suggest that Nylander's operation (*Ann Surg* 142(6):954-956, 1955) might be useful in treating children with hemorrhages caused by lesions in the trunk of the portal vein when the caliber of the blood vessels is too small to permit radicular anastomosis.

6579 A SIXTEEN-YEAR EXPERIENCE WITH END-TO-SIDE PORTACAVAL SHUNT FOR VARICAL HEMORRHAGE: ANALYSIS OF DATA AND COMPARISON WITH OTHER TYPES OF PORTASYSTEMIC ANASTOMOSES.

(E.) Panke, W. F. (New York U. Sch. Med., New York City), L. M. Rousselot and A. R. Burchell. *Ann Surg* 168(6):957-965, 1968.

Results of elective end-to-side portacaval shunts are reported for 210 patients (aged 15 to 76 yr, 122 males and 88 females) who had histologically confirmed hepatic cirrhosis. The cause of the cirrhosis was alcoholism in 150 patients and acute infectious hepatitis in 31. In every case a diagnosis of portal hypertension and esophagogastric varices was confirmed by manometric studies and portography. Surgical risk, as determined by liver function, tissue wasting, and ascites, was good in 130 cases, fair in 65, and poor in 15. The overall hospital mortality was 10% (21 patients), the most common cause of death being liver failure. Patients with ascites accounted for 18 of the 21 hospital mortalities. Among 129 cases of late mortality the most common causes of death were progressive liver disease, hepatoma, peptic ulcers and their complications, and pneumonia. There were no significant differences in the length of survival in patients with alcoholic and those with nonalcoholic cirrhosis. However, abstinence from alcohol increased survival significantly. There was no correlation between survival and preshunt portal blood flow as measured in 38 of 129 patients. A total of 26% of all patients developed hepatic encephalopathy, most within the first year after surgery. The incidence of encephalopathy was higher in older patients with nonalcoholic cirrhosis. Deaths due to recurrent bleeding from esophageal varices occurred in 3 patients (1.4%), all of whom had shunt closure at autopsy. The 10-yr survival rate was 13% and the 5-yr survival rate, 20%.

6580 COMPARATIVE STUDY OF DIURETIC AGENT IN PATIENTS WITH CIRRHOSIS AND ASCITE

(Sp.) Lennhoff, M. (Natl. Inst. Nutrit., Mexico City, Mexico), R. Duhalt and J. C. Peña. *Rev Invest Clin* 20(2):165-180, 196

Electrolyte metabolism was studied in 8 patients with alcoholi cirrhosis and ascites during treatment with various combination of ethacrynic acid, furosemide, hydrochlorothiazide, spironolactone, and triamterene. Two patients had normal Na excretion and 6 had excessive proximal Na absorption; none had gastrointestinal bleeding or symptoms of ammonia intoxication. The diuretic action of ethacrynic acid was significantly stronger tha that of an equal dose of fuorsemide. This was confirmed by dose-response curves. The effect obtained by combining these 2 diuretics was not significantly different from that obtained by equal doses of either drug alone. The use of spironolacton enhanced the action of all the diuretics studied and greatly reduced K excretion and increased urinary Na/K and serum K values. There were no significant differences between the urinary K excretion produced by ethacrynic acid, furosemide, and hydrochlorothiazide. During therapy, all patients had moderate elevations in uric acid but no joint pain. Significant increases also occurred in mean urea and creatinine values.

6581 ARTERIOVENOUS SHUNTS IN CIRRHOTIC PATIENTS STUDIED WITH HUMAN SERUM ALBUMIN MACROAGGREGATES TAGGED WITH ^{131}I (MAA^{131}I).

(E.) Dal Palù, C. (Inst. Med. Path., U. Trieste, Italy), I. Dal Zotto and A. C. Pessina. *Scand J Gastroent* 3(4):425-431, 1968.

The extent of arteriovenous anastomosis in the visceral and peripheral circulations was studied by the selective injection of radioiodinated human serum albumin macroaggregates in 41 cirrhotic patients and 20 controls convalescing from diseases which do not affect the cardiovascular system. The validity of the technique was demonstrated by the marked difference in the radioactivity curves obtained in cirrhotic patients and controls. When 15 μC of the labeled serum albumin was injected into the subclavian vein and radioactivity was recorded over the 3rd-4th intercostal space at the left sternal border and over the left temporal area, pulmonary arteriovenous shunts were demonstrated in 15 of 22 cirrhotics and none of 15 controls. There was no significant correlation between the extent of shunting and clinical features such as cutaneous spider angiomatas, jaundice and ascites, but the most striking abnormality in the radioactivity curve was seen in the one patient with cyanosis and clubbing of the fingers. In other groups of cirrhotic and control patients, the tracer was injected into the spleen with counting over the hepatic and precordial areas, into the left branch of the portal vein with counting over the precordial area, into the splenic artery with counting over the spleen, liver and precordium, into the superior mesenteric artery with counting over the right lower abdomen liver and precordium, into the brachial artery with counting over the palm and upper arm, and into the femoral artery with counting over the sole and inguinal area. The results of these studies showed that the shunt phenomenon is not limited to the pulmonary circulation but also involves the peripheral and splanchnic circulations.

6582 SEQUELAE OF PREOPERATIVE HORMONE THERAPY IN PATIENTS WITH PORTAL HYPERTENSION. *(Rus.)* Uglov, F. G. (I. P. Pavlov 1st Leningrad Med. Inst., USSR) and A. Ia. Kharakhash. *Vestn Khir Grekov* 101(9):6-10, 1968.

Corticosteroids (prednisone, prednisolone, triamcinolone, and others) were given to 11 out of 41 patients with cirrhosis of the liver and portal hypertension. Total doses of 1500-2000 mg were taken over periods ranging from 6 months to 2 yr. Surgery was performed on 7 of these patients: splenectomy in 1 and splenectomies with end-to-side splenorenal anastomoses in 6. After surgery, 5 developed acute liver failure and 4 of these 5 died; one recovered only after long-term conservative therapy. Sequelae in patients who had been given long-term corticosteroid therapy included susceptibility to infections, nephritis, erosive gastritis, acute stomach ulcers with perforation (3 patients), and profuse and fatal gastric bleeding (2 cases). Two other patients recovered smoothly because of specific therapy which included hydrocortisone i.v. before surgery and i.m. for the first 3 days after. ACTH and antibiotics were given 5-10 days after the hydrocortisone had been discontinued. Among the 30 other patients who died not receive corticosteroid therapy there were 7 postoperative deaths from acute liver failure. No other sequelae were seen in this group. It is concluded that corticosteroids are unnecessary and contraindicated in patients with portal hypertension.

6583 ACUTE HYALINE NECROSIS OF THE LIVER: A SURGICAL TRAP. *(E.)* Mikkelsen, W. P. (U. South. California Sch. Med., Los Angeles), F. L. Turrill and W. H. Kern. *Amer J Surg* 116(2):266-272, 1968.

Among 23 patients with acute hyaline necrosis of the liver who did not receive portacaval shunts, 16 presented with a clinical picture mimicking a "surgical" hepatobiliary lesion. Surgery was performed on 10 of these patients and contributed significantly to the death of 4. In another group of 54 patients given portacaval shunts for cirrhosis and bleeding varices, 23 had acute hyaline necrosis. There were 15 immediate postoperative deaths and 4 late deaths in this group, leaving only 4 survivors 1-7 yr later. In contrast, among the 31 patients with cirrhosis but without hyaline necrosis, there were only 2 postoperative and 5 late deaths, leaving 24 survivors. Although the disease may be suspected clinically, especially in the presence of liver tenderness, conclusive diagnosis requires histologic examination of a liver biopsy specimen. The usual criteria for evaluating surgical risk among patients receiving a portacaval shunt were of no value in identifying those with acute hyaline necrosis. It is recommended that needle biopsy of the liver precede consideration for emergency or urgent operative control of bleeding varices in patients with cirrhosis.

6584 CIRRHOSIS OF THE LIVER IN ADULTS. A PATHOLOGICAL STUDY OF 68 POSTMORTEM CASES. *(E.)* Maheshwari, H. B. (K. G. Med. Coll., Lucknow, India) and R. M. L. Mehrotra. *J Indian Med Ass* 51(4):155-162, 1968.

6585 CIRRHOSIS IN ALCOHOLICS. *(E.)* Leevy, C. M. (New Jersey Coll. Med., Jersey City). *Med Clin N Amer* 52(6):1445-1455, 1968.

6586 PROBLEMS AND OPERATIVE TECHNIQUE IN PORTACAVAL ANASTOMOSES. *(E.)* Esser, G. (Dept. Surg., U. Bonn, Germany). *Minn Med* 51(11):1527-1530, 1968.

6587 PORTAL HYPERTENSION VARICOSE VEIN BLEEDING AND SHUNT OPERATION. *(E.)* Gütgemann, A. (Dept. Surg., U. Bonn, Germany) and G. Esser. *Minn Med* 51(11):1517-1525, 1968.

6588 PORTAL HYPERTENSION, LIVER DAMAGE, AND REFRACTORY DIABETES MELLITUS IN A 40-YEAR-OLD MAN. *(Jap.)* Nomura, M. (Kanto-Chuo Hosp., Tokyo, Japan). *Nichidai Ig Z* 27(9):1060-1073, 1968.

6589 THE CIRRHOTOGENIC COURSE OF SCLEROTIC HYALINE NECROSIS OF THE LIVER, A CYTOLYTIC HEPATOSIS CAUSED BY CHRONIC ALCOHOLISM. *(Fr.)* Bertrand, L., A. Pages and H. Michel. *Rev Int Hepat* 18(2):339-353, 1968.

6590 DELAYED HYPERSENSITIVITY TO HEPATIC ANTIGEN IN PRIMARY BILIARY CIRRHOSIS. *(It.)* Pipitone, V. (Inst. Spec. Med. Path., U. Bari, Italy), M. Carrozzo and O. Albano. *Arch Ital Mal Appar Dig* 34(4):418-424, 1967.

6591 THE DEVELOPMENT OF CIRRHOSIS AS REFLECTED IN THE PLASMA PROTEINS. *(Fr.)* Rousselet, F. and M. L. Girard. *Rev Int Hepat* 18(2):323-337, 1968.

6592 AGE-RELATED CHANGES IN HEPATIC BLOOD FLOW IN NORMAL AND CIRRHOTIC SUBJECTS. *(It.)* Dal Zotto, I. (Gen. Med. Clin., U. Padua, Italy), G. C. Gazzola, G. Maddalena and A. Ruol. *Arch Ital Mal Appar Dig* 34(5):497-504, 1967.

6593 HEPATIC CIRRHOSIS AND BETA-THALASSEMIA–A RELATIVELY COMMON COMBINATION OF DISEASES. *(It.)* De Sandre, G. (Gen. Med. Clin., U. Padua, Italy), L. Vettore, G. Perona and S. Cortesi. *Arch Ital Mal Appar Dig* 34(5):504-508, 1967.

6594 THE ROLE OF END-TO-END SPLENORENAL ANASTOMOSIS IN THE TREATMENT OF PORTAL HYPERTENSION: ANATOMICAL AND HEMODYNAMIC FINDINGS. *(It.)* Galmarini, D. (Gen. Surg. Clin., U. Milan, Italy), L. R. Fassati, P. G. Zanoli and S. B. Doldi. *Arch Ital Chir* 94(2):163-189, 1968.

6595 STUDIES ON THE MAGNESIUM CONTENT OF THE LIVER IN CIRRHOSIS. *(Ger.)*

Wilke, H. (Inst. Path., Free U. Berlin, Germany) and H. Spielmann. *Klin Wschr* 46(21):1162-1164, 1968.

6596 LATE RESULTS IN THE TREATMENT OF PORTAL HYPERTENSION. *(Rus.)* Panakhiian, A. G. (Kalinin Med. Inst., USSR). *Khirurgiia (Moskva)* 44(3):31-35, 1968.

6597 THE PROBLEM OF PORTAL HYPERTENSION COMPLICATED BY BLEEDING ESOPHAGEAL VARICES. *(Ger.)* Denck, H. (Munic. Hosp., Vienna, Austria). *Wien Klin Wschr* 80(42):776-778, 1968.

6598 GASTRIC ACIDITY IN CIRRHOSIS OF THE LIVER. *(Pol.)* Machalski, M. (1st Clin. Int. Dis., Silesian Acad. Med., Katowice, Poland) and Z. Machowski *Pol Tyg Lek* 23(40):1511-1513, 1968.

6599 A TECHNIQUE FOR END-TO-SIDE PORTACAVAL ANASTOMOSIS: EXPERIENCE WITH 254 CASES. *(Fr.)* Hivet, M. (St. Antoine Hosp., Paris, France) and J. P. Chevrel. *Ann Chir* 22(15-16/17-18): 1029-1035, 1968.

6600 INTRAHEPATIC PORTAL ACRO-ANGIOMATOSIS IN CASE OF CIRRHOSIS: AN ANATOMICAL AND RADIOLOGICAL ENTITY. *(Fr.)* Leger, L. (Cochin Hosp., Paris, France), R. Brou, G. Lemaigre and J. P. Lenriot. *Presse Med* 76(35):1687-1690, 1968.

6601 SOME CURRENT PROBLEMS IN THE DIAGNOSIS AND SURGICAL TREATMENT OF PORTAL HYPERTENSION. *(Rus.)* Uglov, F. G. (I. P. Pavlov 1st Leningrad Med. Inst., USSR) and N. U. Usmanov. *Sovet Med* 31(9):63-69, 1968.

6602 THE IMMUNOLOGICAL REACTIVITY OF THE BODY FOLLOWING SURGICAL TREATMENT OF HEPATIC CIRRHOSIS. *(Rus.)* Levin, G. S. (Uzbek Sci.-Res. Inst. Hematol., Tashkent, USSR), Iu. Iu. Parlagoshvili and M. Z. Imas. *Sovet Med* 31(9):69-75, 1968.

6603 THE IMPORTANCE OF SYSTEMATIC TESTS FOR THE PRESENCE OF WILSON'S DISEASE IN ANY YOUNG PATIENT WITH CIRRHOSIS. *(Fr.)* Trad, J. (Franco-Moslem Hosp., Bobigny, Paris, France), A. Paraf and M. Bordry. *Entret Bichat [Med]* pp. 595-600, 1968.

6604 THE INCREASED INCIDENCE OF PRIMARY LIVER CANCER IN CIRRHOSIS: PROSPECTIVE STUDIES ON KNOWN CIRRHOTIC PATIENTS. *(Fr.)* Péquignot, H. (Broussais Hosp., Paris, France), J. P. Etienne, P. Delavierre and J. P. Petite. *Entret Bichat [Med]* pp. 71-76, 1968.

6605 FUNDAMENTALS AND INDICATIONS FOR COLECTOMY IN THE TREATMENT OF ENCEPHALOPATHY FOLLOWING PORTOSYSTEMIC ANASTOMOSES. *(Por.)* Pontes, J. T. (São Paulo U. Med. Sch., Brazil). *Arq Gastroent* 5(2):43-46, 1968.

6606 URINARY IRON EXCRETION CAUSED BY THE TREATMENT OF HEPATIC CIRRHOSIS. *(Ger.)* Quina, M. G. (Fac. Med., U. Lisbon, Portugal) and C. Rocha. *Z Gastroent* 6(4):287-289, 1968.

6607 ENCEPHALOMYELOPATHY IN HEPATIC CIRRHOSIS. *(Sp.)* Rodés, J. (Fac. Med., Barcelona, Spain), M. Bruguera and J. M. Bordas. *Med Clin (Barcelona)* 51(1):160-163, 1968.

6608 CIRRHOSIS AND DIABETES. *(Sp.)* Reitg de Llobet, L. (Fac. Med., Barcelona, Spain). *Med Clin (Barcelona)* 51(1):144-149, 1968.

6609 JAUNDICE IN PATIENTS WITH CIRRHOSIS. *(Sp.)* Bordas Alsina, J. M. (Fac. Med., Barcelona, Spain). *Med Clin (Barcelona)* 51(1):38-49, 1968.

6610 PERSONAL EXPERIENCE WITH GASTRO-ESOPHAGEAL TAMPONADE IN THE TREATMENT OF SEVERE DIGESTIVE TRACT HEMORRHAGE DUE TO PORTAL HYPERTENSION. *(Sp.)* Pavlovsky, H. J. (Ramos Mejía Hosp., Buenos Aires, Argentina). *Medicina (Mex)* 48(1041):391-397, 1968.

6611 RENAL CIRCULATORY INSUFFICIENCY AS A COMPLICATION OF HEPATIC CIRRHOSIS. *(Dan.)* Thomsen, A. C. (Copenhagen, Denmark). *Nord Med* 80(33):1091-1092, 1968.

6612 COAGULATION DISORDERS IN PATIENTS WITH CIRRHOSIS OF THE LIVER. *(Ger.)* Frick, P. G. (Med. Clin., U. Zurich, Switzerland). *Deutsch Med. Wschr* 93(41):1966-1967, 1968.

6613 THE TREATMENT OF HEPATIC CIRRHOSIS. *(Sp.)* Meeroff, M. (Buenos Aires, Argentina). *Galicia Clin* 40(5):355-375, 1968.

6614 KIDNEY FUNCTION IN CIRRHOSIS OF THE LIVER (A REVIEW OF THE LITERATURE). *(Rus.)* Geller, L. I. (Khabarovsk Med. Inst., USSR). *Ter Arkh* 40(7):15-21, 1968.

6615 VARIATIONS IN BLOOD ACCELERIN LEVELS FOLLOWING SPLENECTOMY IN PATIENTS WITH CIRRHOSIS. *(Fr.)* Lévy, V. G. (St. Antoine Hosp., Paris, France), A. Najman, D. Vergoz and J. Caroli. *Rev Medicochir Mal Foie* 43(3):105-116, 1968.

6616 SPIROMETRIC STUDIES FOLLOWING INTRATHORACIC DISPLACEMENT OF THE SPLEEN DUE TO PORTAL HYPERTENSION IN CHILDREN. *(Ger.)* Sztaba, R. (Pediat. Surg. Clin., Med. Acad. Gdansk, Poland) and R. Rogón. *Zbl Chir* 93(36):1256-1260, 1968.

See also: 5848, 5891, 5927, 6261

6617 AUTOPSY STUDY ON THE MODE OF DISSEMINATION OF GALLBLADDER CANCER.

(It.) Botticelli, A. (Inst. Anat. Path. Histol., U. Modena, Italy). *Arch De Vecchi Anat Pat* 50(1/2):463-467, 1967.

An autopsy study of 54 patients with gallbladder neoplasms (36 adenocarcinomas and 18 carcinomas) showed that 19 had not metastasized and 35 had. The sites of metastasis were the liver (26 cases), lymph glands in the lesser omentum (6 cases), common bile duct (3 cases), pancreas (3 cases), and cystic duct, hepatic duct, stomach, ovaries, duodenum, peripancreatic lymph glands, and lesser omentum (1 case each). Abdominal carcinomatosis was present in 2 cases. In the 49 cases in which it could be determined, metastasis occurred to adjacent sites in 21 cases, by direct spread in 14 cases, through the blood in 6 cases, through the blood and lymph simultaneously in 3 cases, to adjacent sites and through the lymph simultaneously in 2 cases, and by direct spread and to adjacent sites simultaneously in 1 case. Case reports are presented for simple gallbladder adenocarcinomas which 1) infiltrated the walls of the gallbladder, cystic duct, common bile duct, and into the duodenum from the serosa to the submucosa; 2) metastasized into the liver and peripancreatic lymph glands; and 3) metastasized into the lymph glands of the lesser omentum.

6618 SIMPLE METHOD FOR THE CREATION OF A BILIARY FISTULA IN THE DOG. *(Pol.)*

Uszynski, H. (Acad. Med., Warsaw, Poland) and J. L. Potocki. *Acta Physiol Pol* 19(5):797-803, 1968.

Following a brief review of surgical techniques for the construction of biliary fistulas in experimental animals, a simple method is described which is based on that of Rous and McMaster (*J Exp Med* 37:11, 1923) and involves the use of two polyvinyl tubes of special construction (2.8-3.0 mm external diameter, 1.7-2.0 mm internal diameter, 27-29 cm long) to drain the common bile duct. Each tube passes through a transverse plastic disk (10 cm from the end) equipped with holes which are used to fasten it to the tissues, thus decreasing inflammatory reactions due to movement of the tube and preventing its removal by the animal. Repeated serum alkaline phosphatase, bilirubin and cholesterol determinations should be carried out in order to verify the patency of the bile duct and the absence of cholestasis. Favorable results are reported for 20 male dogs maintained with biliary fistulas for periods of 6-8 weeks.

6619 FISTULA FORMATION AND THE EFFECT OF ECHINOCOCCAL LIVER CYSTS ON THE BILIARY TRACT.

(Bul.) Daskalov, M. (Higher Med. Inst., Sofia, Bulgaria). *Khirurgiia (Sofiia)* 21(2):143-147, 1968.

Detailed case reports are presented for 5 patients (1 man and 4 women, 49-59 yr old) with echinococcal cysts of the liver communicating with the biliary tract. The symptoms were those of cholelithiasis, choledocholithiasis, cholecystitis and biliary dyskinesia; in 2 cases diagnosed preoperatively, who had concomitant cholelithiasis, the cysts had displaced the gallbladder. Fever, jaundice and pain responded poorly to conservative treatment, but surgery was successful in 3 cases. Based on experience with these and other cases, it is concluded that the biliary tract is affected most often when large purulent cysts are located in the middle lobe of the liver. Primary localization in the extrahepatic bile ducts and simultaneous communication with both the biliary tract and the bronchi are rare. Surgery should be individualized and directed towards eliminating the septic focus, closing any fistulae into the bile ducts, and assuring the flow of bile into the duodenum.

6620 HYMECROMONE: A NEW CHOLERETIC AND ANTISPASMODIC FOR ODDI'S SPHINCTER. CLINICAL PHARMACOLOGY AND STATISTICAL CLINICAL STUDY.

(It.) Cremoncini, C. (Hosp. Inst., Milan, Italy) and L. Tominz. *Minerva Med* 59(82):4359-4368, 1968.

A single large dose (1450 mg) of hymecromone (6-hydroxy-4-methyl-2-keto-chromene-3), administered as its sodium salt through a catheter into the duodenum, produced a bile flow 2-3 times greater than that under basal conditions after a latent period of 15-30 min. This effect lasted 30-70 min or more. A single dose of 1000-1600 mg prevented spasms produced in Oddi's sphincter by injection of 5 mg morphine and, if the patient was under long-term hymecromone therapy, those produced by 10 mg morphine. Spontaneous spasms were also relieved in 2 patients. In a clinical test, 34 patients (23 males and 11 females, aged 21-78 yr) were given 800-1800 mg/day divided into 2-3 doses. These patients included 11 with chronic hepatitis, 6 with chronic obstructive jaundice, 6 with biliary dyskinesia, 5 with angiocholitis, 3 with liver cirrhosis, 2 with cholelithiasis, and 1 with the sequelae of acute hepatitis. Treatment lasted 11-51 days. In addition to subjective improvement, follow-ups showed significant reduction in the size of the liver and improvements in blood protein and serum bilirubin values and in the serum colloid lability and BSP tests. Bile pigment in the urine decreased or disappeared and urinary urobilin values decreased in many cases. No significant changes occurred in SGOT and SGPT values or in prothrombin activity. No side effects were seen. In a double-blind study on 12 patients with chronic liver disease and hepatomegaly, no differences were found between the effects of hymecromone and those of sodium dehydrocholate.

6621 SOME LESS COMMON FEATURES OF BILIARY PATHOLOGY. III. EARLY DIAGNOSIS OF PRIMARY GALLBLADDER NEOPLASMS.

(It.) Martinotti, A. (Polyclinic, U. Milan, Italy), V. di Carlo, G. Bevilacqua and A. Randazzo. *Arch Ital Chir* 93(6):857-875, 1967.

A series of 27 case reports of patients with primary gallbladder neoplasms is analyzed. Of these, 24 were diagnosed at surgery and 3 at autopsy. Preoperative diagnoses were acute cholecystitis in 14 cases, biliary peritonitis in 2, and gallbladder neoplasms in 7. The 4 remaining cases were diagnosed as acute appendicitis, ascitogenic hepatic cirrhosis, cholecystoduodenal fistula, and obstructive posthepatitic jaundice. Of these patients, 81.5% were females and the mean age was 63 yr. Only 8 patients had a history of previous biliary tract disease. Clinical and laboratory findings considered to be significant in the differential diagnosis of primary gallbladder neoplasms are a recent history of pain lasting for more than a week; a low fever or absence of fever in patients with symptoms of acute abdomen; lack of response to treatment consisting of bedrest, cold applications, antibiotics, and antispasmodics; obstructive jaundice which begins with characteristic biliary pain but is not very intense and does not resolve rapidly; elevated serum alkaline phosphatase values with normal serum bilirubin values; high erythrocyte sedimentation rates in apyretic patients; and elevations in α_1- and α_2-globulins sometimes associated with an elevation in β- and γ-globulins or, less frequently, an elevation of γ-globulins alone. Cholangiography gave negative results in 13 out of the 14 cases in which it was employed, showing only a perforated gallbladder neoplasm. Cholecystectomies were performed in 6 of 24 patients and exploratory laparotomies in 18. Histological examination showed the tumor to be an adenocarcinoma in 18 cases, a solid carcinoma in 6, an undifferentiated carcinoma in 2, and an epidermoid carcinoma in 1. The tumors had metastasized in all but 3 cases.

6622 GLANDULAR HYPERPLASIA AND DYSPLASIA IN THE GALLBLADDER MUCOSA, PARTICULARLY IN CHOLELITHIASIS AND GALLBLADDER CARCINOMA.

(It.) Properzi, A. (Santo Salvatore Munic. Hosp., L'Aquila, Italy) and T. Ventura. *Chir Gastroent* 2(1):35-46, 1968.

Histological examination of 214 gallbladders removed from 166 women and 48 men revealed abnormalities in 95 (44.4%). In 72 cases the findings included hyperplasia of normal cells in the mucosa and an increase in the number and intraparietal lengthening of pseudoglandular diverticula which had an epithelium identical to that found in the gallbladder mucosa. Gallstones were found in the diverticula no matter whether inflammation was mild or severe. Glandular metaplasia or dysplasia was present in 21 cases. It was apparently not related to preexisting hyperplasia, but to the appearance of analogous glandular structures in the duodenum. Lymph follicles were found in the body of the gallbladder in 5 of these 21 cases and a pea-sized nodule was found in 1. Adenocarcinomatous changes were found in dysplastic glandular structures in 2 cases. Lymph follicles which had been invaded by the neoplasm were present in both of these cases.

6623 BILIARY BILE ACIDS AND HEPATIC ULTRA-STRUCTURE IN HAMSTERS FED GALLSTONE-INDUCING AND -DISSOLVING DIETS.

(E.) Bergman, F. (Dept. Surg., U. Umeå, Sweden.), W. van der Linden and J. Sjövall. *Acta Physiol Scand* 74(3):480-491, 1968.

Cholelithogenesis and the composition of gallbladder and fistula bile were studied in hamsters fed gallstone-inducing diets with and without addition of 3% cholestyramine or 5 mg% thyroxine. In all cases, the predominant bile acids were cholic, chenodeoxycholic, deoxycholic, 7-ketodeoxycholic, and $7\alpha,12\alpha$-dihydroxy-3-keto-5β-cholanoic acids. Small amounts of 7-ketolithocholic, 12-ketolithocholic, 12α-hydroxy-3-keto-5β-cholanoic, allocholic and ursodeoxycholic acids were also found. In hamsters fed a diet favoring the formation of cholesterol gallstones the mean of the chenodeoxycholic/cholic acid ratios was 0.88. When 3% cholestyramine was added to the diet this ratio fell to about 0.04. Thyroxine caused a similar but less striking decrease in the ratio. The bile acid/cholesterol ratio was lower in animals on the cholesterol stone-inducing diet. In contrast to previous findings in guinea pigs, this ratio was increased by cholestyramine, which prevented rather than inducing the formation of cholesterol stones. Morphological and histochemical changes were found in the hepatic parenchymal cells of only the animals treated with the thyroxine-containing diet, which induces predominantly pigmented stones. The cells contained numerous lipid droplets and clusters of autophagic vacuoles, believed to represent advanced focal cytoplasmic degeneration.

6624 ANATOMICAL AND SURGICAL FINDINGS ON THE CONFLUENCE OF THE COMMON BILE DUCT AND THE PANCREATIC DUCT AT THE AMPULLA OF VATER WITH SPECIAL REFERENCE TO ODDI SPHINCTEROTOMY.

(It.) Peri, G. (Gen. Surg. Clin., U. Milan, Italy), S. Fiaccavento and V. Blini. *Arch Ital Chir* 94(2):240-252, 1968.

The anatomy of the ampulla of Vater and the component ducts is described in detail on the basis of 30 specimens removed shortly after death from patients without biliopancreatic disease. In 66.6% of the specimens (20 cases), the ampulla was located in the middle of the descending duodenal segment, at an average of 7.5 cm from the pylorus, but in 10 cases the ampulla was near the upper (13.3%) or lower (20%) bend of the duodenum, anywhere from 4 to 12 cm from the pylorus. A joint hepatico-pancreatic duct, varying in length from 3-6 mm (average 4.1 mm), was found in 16 cases (53.1%); in 12 cases, however, the hepatic and pancreatic ducts entered the duodenum separately, although close together, and in 2 other cases the 2 ducts remained widely separated. In 23 cases (76.6%), the terminal pancreatic duct entered the medial wall of the distal choledochus at a typical acute angle, but in the other 7 cases the terminal segmen of the pancreatic duct ran in front of (10%) or behind (13.3%) the common bile duct. In 14 cases (46.6%), the pancreatic duct terminated in the lower-medial or lower-anterior quadrant of the ampulla with respect to the common bile duct, but this "normal" configuration was not found in the other 16 cases; in 9, the pancreati duct terminated in the upper-medial or upper-anterior quadrant, in 3 it terminated in the upper-lateral or upper posterior quadrant, and in 4 it terminated in the lower-lateral or lower-posterior quadrant. The significant anomali discovered in the reciprocal relations between the pancreatic duct and the common bile duct may play a role in the pancreatic duct damage which sometimes occurs during sphincterotomy, as well as in the frequent development of pancreatitis complicating biliary tract surgery or disease.

6625 GALLSTONE ILEUS: A REVIEW OF THE ROENTGENOGRAPHIC FINDINGS AND REPORT OF A NEW ROENTGEN SIGN.

(E.) Eisenman, J. I. (California Coll. Med., Los Angeles), E. J. Finck and B. J. O'Loughlin. *Amer J Roentgen* 101(2):361-366, 1967.

In addition to reviewing the clinical and supine roentgenograph signs on which a diagnosis of gallstone ileus is usually based (gas in the gallbladder or biliary tract, ectopic localization of a gallstone, and partial or complete obstruction of the small bowel), the authors describe a new sign, called the loop obtura tion pattern. This sign refers to the characteristic appearance the loops of small bowel in the supine roentgenogram of the lomen in the presence of gallstone ileus. There is minimal- moderate gaseous distension of the fluid-filled loops; the vulae conniventes are prominent and show no evidence of treme effacement; loops of bowel containing only fluid are ll delineated, and the gas-containing loops often take on a ial configuration. This sign was present in 30 of 35 confirmed es of gallstone ileus with the obturating stone in the mesen- ic small bowel. Gas was present in the gallbladder or biliary e in 21 of these cases, and 9 showed an ectopic calculus, but 11 cases the loop obturation pattern was the only roentgeno- phic sign. The underlying mechanism of this sign is the rel- vely small amount of gas and moderate amount of fluid in the tended small bowel. The increased incidence of gallstone us in the elderly is also pointed out.

26 CLINICAL STUDIES ON THE ACTIVITY OF REC 14-0127, A NEW SYNTHETIC CHOLERETIC ;ENT. *(It.)* Trecca, T. F. (2nd Surg. Clin., U. Rome, Italy). *zz Int Med Chir* 73(19):1733-1741, 1968.

e increases in biliary secretion following p.o. or s.c. adminis- tion of the choleretic agent REC 14-0127 were studied by her duodenal catheterization or drainage through an external iary fistula in 25 patients (6 men and 19 women, 24-71 yr) with surgical diseases of the liver and biliary tract (chole- iasis, pancreatitis, papillitis, jaundice, biliary dyskinesia, bil- y fistulas, and biliary tract obstruction due to stenosis or icer). Increases in bile flow ranging from 16% to 400% were tained in 24 of the 25 subjects. The average increase was 1% in 13 patients with normal common bile ducts, 106% in patients with involvement of the common bile duct, 94% in atients with normal liver function, 108% in 18 patients with atic dysfunction, and 179% in the 7 of these having severe function. Although the duration of the choleretic effect was y variable, the latent period was only 5-15 minutes. There re no side effects. It is concluded that REC 14-0127 has a mpt and energetic choleretic effect which is independent the anatomical or functional condition of the liver or biliary ct.

27 THE ETIOLOGICAL RELATION BETWEEN CHOLELITHIASIS AND CANCER OF THE LLBLADDER (208 PERSONAL OBSERVATIONS). *(It.)* rtolozzi, M. (Munic. Hosp., Treviso, Italy). *Chir Gastroent* 3):279-282, 1968.

llbladder neoplasms were found in 208 of 24,379 autopsies d 94,680 biopsies performed over a period of more than 30 The incidence of gallbladder neoplasms was about 6.50% both the autopsy and biopsy series. The ratio of females to les among those with cholelithiasis was 3:1 and the ratio ong those with gallbladder cancer was 5:1. Squamous cell rcinomas and adenocarcinomas occurred most frequently, t 1 case of a spindle cell sarcoma originating from the uscular layer of the venous walls and 3 cases of papilliferous rcinomas without cholelithiasis were also seem. It is concluded at cholelithiasis can produce gallbladder neoplasms but that oleliths cannot form in a neoplastic gallbladder.

28 CHOLECYSTOSTOMY IN ACUTE CHOLECYSTITIS: FACTORS INFLUENCING MOR- DITY AND MORTALITY. *(E.)* Gingrich, R. A. (U. Oregon d. Sch., Portland), W. C. Awe, A. M. Boyden and C. G. terson. *Amer J Surg* 116(2):310-315, 1968.

Of 323 patients treated surgically for acute cholecystitis during a 15-yr period, 114 (35%) had cholecystostomy as the initial form of treatment. There were 65 females and 49 males, with an average age of 70 yr. The operative mortality was 24.6% and nonfatal complications occurred in 30%. Death in the majority of cases was due to complications related to the use of general anesthesia or large exploratory incisions. Postoperative tube cholangiography revealed residual stones in 54% of those studied, most of whom experienced further difficulty. In contrast, 61% of patients in whom postoperative tube cholangiography was unrevealing remained asymptomatic after cholecystostomy alone. These results reemphasize the importance of "planned cholecystostomy" using local anesthesia and an orderly plan of postcholecystostomy care.

6629 EFFECTS OF GALLBLADDER AND COMMON BILE DUCT MANIPULATION ON FIRING OF SENSORY VAGAL FIBERS WITH CARDIAC RHYTHM. *(E.)* Bagabaldo, Z. G., (Coll. Med., U. Philippines, Manila). *Acta Med Philipp* 4(4):181-188, 1968.

The effects of manipulation and distension of the gallbladder and common bile duct on the rate and pattern of firing of afferent cardiovascular vagal fibers were studied in cats. In 18 unselected cats, after exposure of the vagi in the neck and isolation of fibers with a cardiac rhythm, the abdominal cavity was opened and the common bile duct was cannulated. Experimental procedures consisted of (1) manipulation of the gallbladder, (2) distension of the gallbladder with normal saline, (3) manual drainage of the gallbladder, and (4) manipulation of the common bile duct. Based on the relation between the burst of potentials and the different waves of the electrocardiogram, 4 types of cardiovascular afferent fibers were detected: (1) 4 type A atrial, (2) 5 type B atrial, (3) 5 ventricular, and (4) 4 arterial baroreceptors. In the majority of instances, the atrial B fibers showed an increase in rate, while most of the responses of the ventricular fibers were towards inhibition. The arterial baroreceptors gave positive increases in rate in response to manipulation and distension of the gallbladder, while the atrial A fibers gave variable results. The results of this study are of importance to the surgeon, since it reveals that manipulation of the gallbladder and common bile duct can alter the flow of impulses passing through the cardiovascular afferent nerve fibers, even though there were no significant changes in the electrocardiogram.

6630 THE HISTOLOGY OF THE AMPULLA OF VATER FOLLOWING TRANSDUODENAL SPHINCTEROTOMY. *(E.)* Schiodt, T., (U. Hosp., Copenhagen, Denmark), B. C. Christensen and M. Andreassen. *Scand J Gastroent* 3(5):553-560, 1968.

The ampullar region in 38 patients subjected to transduodenal sphincterotomy was examined macro- and microscopically. The series was divided into 2 groups: 21 patients with choledocholithiasis (16) or pancreatitis (5) and 17 with a more doubtful diagnosis of organic biliary tract disease. Histologic examination of the ampullar region yielded no evidence to suggest an organic basis for the sphincteric disorders. Neither did a comparison between sphincter tissues from patients of the clinical series and from autopsies of 38 patients with and 38 patients without biliary tract diseases reveal any significant differences in histologic structure. There was no significant preponderance of sphincteric fibrosis in the clinical series.

6631 CHOLELITHIASIS IN SICKLE CELL ANEMIA. *(E.)* Barrett-Connor, E. (U. Miami Sch. Med., Florida). *Amer J Med* 45(6):889-898, 1968.

The incidence, diagnostic problems, clinical course and management of sickle cell anemia complicated by cholelithiasis are discussed in detail. In 25 of 47 patients, aged 12 to 58 yr, with homozygous sickle cell disease, the presence or absence of cholelithiasis was unknown because of inadequate study or the absence of biliary tract investigation. Of the remaining 22 patients, 16 had cholelithiasis and 15 of these were diagnosed by oral cholecystography. Case reports are presented for 10 of the 11 patients subjected to cholecystectomy. Based on this experience, it is concluded that at least one third of patients over the age of 10 with homozygous sickle cell disease have gall stones and pathologic findings of chronic cholecystitis. However, less than 10% have symptoms or signs which can be attributed to the biliary tract or relieved by cholecystectomy. Since it is difficult to diagnose the complications of cholelithiasis by clinical and laboratory studies in the presence of sickle cell anemia, except in cases with radiopaque stones, it is suggested that elective cholecystectomy be considered in all patients with sickle cell anemia and cholelithiasis.

6632 CONGENITAL ATRESIA OF THE BILE DUCTS. *(It.)* Von Elzenbaum, H. (Munic. Hosp., Silandro-Val Venosta, Bolzano, Italy). *Osped Ital Chir* 18(6):525-538, 1968.

6633 PERSONAL EXPERIENCE IN SURGERY OF THE COMMON BILE DUCT. *(It.)* Prior, C. *Rass Arch Chir* 1(3):148-151, 1967.

6634 FLORANTYRON IN BILIARY TRACT DYSKINESIA. *(Sw.)* Aronsen, K. F. (Sch. Med.. U. Lund, Sweden), J. Gundersen and K. Haeger. *Lakartidningen* 65(Supp. 3):74-78, 1968.

6635 A TECHNIQUE FOR BILIOJEJUNAL ANASTOMOSIS. *(Sp.)* Haddad, R. E. (Gen. Hosp., Mexico City). *Cir Cir* 36(3):237-246, 1968.

6636 THE CLINICAL CHARACTERISTICS AND MORPHOLOGY OF TUBERCULOSIS OF THE GALLBLADDER. *(Ger.)* Mlynek, H. J. (Surg. Clin., Karl-Marx U., Leipzig, Germany) and C. Engel. *Zbl Chir* 93(38):1332-1336, 1968.

6637 FATAL HEMORRHAGE FROM THE GALLBLADDER INTO THE ABDOMINAL CAVITY. *(Ger.)* Haupt, R. (St. Georg Hosp., Leipzig, Germany). *Zbl Chir* 93(32):1121-1123, 1968.

6638 THE ROLE OF LOOSELY-BOUND CHOLESTEROL AND BILE BILIRUBIN IN THE MECHANISM OF FORMATION OF GALL STONES. *(Rus.)* Fin'ko, D. I. and V. S. Davidik. *Vestn Khir Grekov* 101(9):62-66, 1968.

6639 CLINICAL AND AUTOPSY OBSERVATIONS IN A CASE OF PRIMARY CARCINOMA OF THE GALLBLADDER ASSOCIATED WITH CHOLELITHIASIS. *(It.)* Vancini, B. (Santa Maria della Misericordia di Pieve Hosp., Cento, Italy), P. Govoni and C. Pacchioni. *Riv Gastroent* 19(6, suppl.):1020-1028, 1967.

6640 STATISTICAL AND CLINICAL CONSIDERATION ON GALLBLADDER DISEASE. *(It.)* Berera, F. (Reg. Hosp., Lecco, Italy). *Riv Gastroent* 19(6, suppl.):1041-1052, 1967.

6641 DIET FOLLOWING CHOLECYSTECTOMY. *(Por* Corrêa, S. M. (Bonsucesso Hosp., Rio de Janeiro, Brazil) and L. V. de Fontoura Carneiro. *Folha Med* 57(2): 207-212, 1968.

6642 SYSTEMATIC EVALUATION OF THE AMYLAS ACTIVITY AFTER OPERATIONS ON THE BILIARY TRACT. *(Sp.)* Gramatica, L. (Santa Cruz y San Pablo Hosp., Barcelona, Spáin), M. T. Ribera, R. Salvador, J. López Gibert and J. Huguet. *An Med [Cir]* 54(3):165-172, 1968.

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6644 COMMENTS ON THE SURGICAL TECHNIQUE FOR REOPERATIONS ON THE BILIARY TRACT MADE NECESSARY BY STENOSIS OF THE AMPULLA AND MID PORTION OF THE COMMON BILE DUCT. *(It.)* Caluzzi, F. (Inst. Gen. Clin. Surg., U. Turin, Italy), E. De Giorgi, I. Sassi, A. Calvi and A. Mussa. *Minerva Chir* 23(17):877-886, 1968.

6645 INFLAMMATORY STENOSIS OF THE EXTRAHEPATIC BILE DUCTS. *(Fr.)* Leger, L. (Cochin Hosp., Paris, France), P. Détrie, Y. Chapuis, G. Woell and G. Lemaigre. *J Chir (Paris)* 95(1):43-56, 1968.

6646 REPORT OF A CASE OF OSSIFICATION OF THE GALLBLADDER: A CONTRIBUTION TO THE STUDY OF HETEROTOPIC OSSIFICATION. *(It.)* Duchini, L. (Inst. Anat. Path. Histol., U. Florence, Italy). *Arch De Vecchi Anat Pat* 50(1/2):537-552, 1967.

6647 THE INCIDENCE, DIAGNOSIS AND TREATMENT OF THE SO-CALLED POSTCHOLECYSTECTOMY SYNDROME. *(Bul.)* Chervenkov, P. (Inst. Postgrad. Med., Varna, Bulgaria) and L. Ganchev. *Khirurgiia (Sofiia)* 21(4):323-329, 1968.

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6649 STATE OF THE BILE DUCTS IN PATIENTS WITH EPILEPSY AT VARIOUS STAGES OF THE DISEASE. *(Ukr.)* Sologub, N. M. (O. O. Bogomolets Inst. Physiol., Acad. Sci. Ukr. SSR, Kiev, USSR), N. Ia. Sofienko and A. G. Sarkisova. *Fiziol Zh (Kiev)* 14(5): 638-645, 1968.

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52 INVOLVEMENT OF THE COMMON BILE DUCT IN GALLBLADDER DISEASE. *(Ger.)* dmaier, F. (Prov. Hosp., Klagenfurt, Austria). *Wien Klin schr* 80(39):717-718, 1968.

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54 HEPATIC ARTERY LIGATION IN THE MANAGEMENT OF MASSIVE HEMOBILIA CAUSED A T-TUBE AFTER RECONSTRUCTION OF THE COM-ON BILE DUCT: REPORT OF A CASE. *(Por.)* stelfranchi, P. L. (Fac. Med., U. São Paulo, Brazil). *Rev ul Med* 73(1):7-12, 1968.

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6660 JAUNDICE DUE TO NON-NEOPLASTIC ADENOPATHY NEAR THE COMMON BILE DUCT. *(It.)* Sartirana, A. (Maggiore Hosp., Milan, Italy) and A. Kuntner. *Arch Ital Chir* 94(2):226-239, 1968.

6661 SURGERY ON THE SPHINCTER OF ODDI IN CHOLELITHIASIS AFFECTING THE COMMON BILE DUCT. *(It.)* Mattioli, F. (Inst. Gen. Clin. Surg., U. Genoa, Italy). *Rass Arch Chir* 1(3):195-212, 1967.

6662 INDICATIONS AND RESULTS IN THE SURGICAL TREATMENT OF CHOLELITHIASIS. *(It.)* Vernetti, L. (Galliera Hosp., Genoa, Italy). *Rass Arch Chir* 1(3):120-147, 1967.

6663 CHOLELITHIASIS AND THE DIAMETER OF THE COMMON BILE DUCT. *(Pol.)* Platek, H. (MSW Milit. Hosp., Olsztyn, Poland). *Wiad Lek* 21(19): 1761-1763, 1968.

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6665 CHRONIC POSTOPERATIVE BILE PERITONITIS. *(Fr.)* Moreaux, J. (Bichat Hosp., Paris, France) and M. Huguier. *Ann Chir* 22(15-16/17-18):997-1002, 1968.

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05870* 05874* 05875* 05882* 05889 05891 05892 05893
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05951 05952 05954 05960 05965 05969 05972 05974
05979 06387 06448* 06452* 06458* 06465* 06469* 06485
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05966 06421*
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05873* 05876* 05914 05920 05964
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05872* 05880* 05901 05909 05914 05917 05941 05955
06625* 06647 06652 06658
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05990* 06112 06119 06121 06130 06131 06133 06136
06140 06141 06253*
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05907 05913 05918 05924 05927 05937 05944 05948
05959 05966 05969 05974 06482 06493 06600
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05953 05976 05977 06409
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06178 06185 06192 06195 06203 06207 06212
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06336 06370 06398*
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05688*
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05865* 06330* 06350 06358 06359 06361 06367 06370
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05892 05893 05907 05913 05915 05918 05921 05924
05927 05937 05944 05949 05965 05974 05976 05977
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06598
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05895 05896 05915 06058 06324 06405 06406 06407
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GASTROENTEROLOGY

ABSTRACTS
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NATIONAL INSTITUTE OF ARTHRITIS AND METABOLIC DISEASES

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
Public Health Service
National Institutes of Health

AUGUST 1968
VOLUME 3, NO. 8
6666–7591

Volume 3 , No. 8 Abstract and Citation Nos. 6666-7591

GASTROENTEROLOGY ABSTRACTS AND CITATIONS

A monthly publication of the
National Institute of Arthritis and Metabolic Diseases

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis and Metabolic Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication of GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available, free to qualified interested investigators and practitioners, citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology has accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis and Metabolic Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

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Use of Funds for printing this publication
approved by the Director of the Bureau of
the Budget on October 7, 1968.

For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. Subscription price per year in the United States, Canada, and Mexico, $8.50; foreign $10.75; and $1.00 per single copy. Payment is required in advance, and check or money order should be made payable to the Superintendent of Documents.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus.* For journals not covered by *Index Medicus,* the abbreviations (with some modifications) found in *World Medical Periodicals,* 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mC, μC	milli-, microcurie(s)
ADP	adenosine diphosphate	mg	milligram(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
BSP	sulfobromophthalein	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
μg	microgram(s)		
hr	hour(s)	resp.	respectively
i.m.	intramuscular	Rev.	review (only in citations)
i.p.	intraperitoneal	RNA	ribonucleic acid
IU	international unit(s)	s.c.	subcutaneous
i.v.	intravenous	sec	second(s)
kg	kilogram(s)	SGOT	serum glutamic-oxalacetic transaminase
LD_{50}	median lethal dose(s)	SGPT	serum glutamic-pyruvic transaminase
LDH	lactic acid dehydrogenase	U	unit(s)
m	meter(s)	UV	ultraviolet
M	molar	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mM	millimolar	yr	year(s)
μM	micromolar		

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

6666 ROUTES OF COLLATERAL CIRCULATION OF THE GASTROINTESTINAL TRACT AS ASCERTAINED IN A DISSECTION OF 500 BODIES. *(E.)*

Michels, N. A. (Jefferson Med. Coll., Philadelphia, Pa.), P. Siddharth, P. L. Kornblith and W. W. Parke. *Int Surg* 49(1): 8-28, 1968.

Descriptions of previously unmentioned collateral pathways to the gastrointestinal tract are presented on the basis of the findings in dissections of 500 bodies (430 adult and 64 newborn and infant). The esophago-gastro-duodenal collateral pathways include those within and those outside of the gastric vasculature proper. Inside routes include (1) the supragastric (right and left gastric), (2) the infragastric (right and left gastroepiploic), and (3) the infracolonic (right and left epiploic). Outside routes include (1) the anterior and posterior pancreaticoduodenal arcades, (2) the long transpancreatic route made by the transverse pancreatic artery, and (3 and 4) the paraesophageal and retroesophageal routes. The latter are commonly made by cardioesophageal branches from the left gastric artery, or from an aberrant left hepatic artery arising from the left gastric, which anastomose with similar branches from the short gastrics stemming from the splenic system or from the largely unknown recurrent branch of the left inferior phrenic artery. Collateral pathways to the small and large intestine are numerous and include: (1) those between the celiac and superior mesenteric arteries (celiacocolic, hepatocolic, splenocolic, pancreaticocolic, gastrocolic, and omentocolic), (2) routes between the superior and inferior mesenteric arteries, (3) routes between the inferior mesenteric and internal iliac arteries, and connecting via the latter to the external iliac and femoral systems, and (4) those between the visceral and parietal branches of the abdominal aorta. By means of this rarely mentioned retroperitoneal plexus, a life-sustaining collateral circulation may be established after complete occlusion of the celiac and superior mesenteric arteries. Knowledge of these collateral pathways is important for the interpretation of gastrointestinal arteriograms and for the successful performance of gastrointestinal transplant operations.

6667 A PHYSIOLOGIC DEMONSTRATION OF THE ANATOMIC DISTRIBUTION OF THE VAGAL SYSTEM TO THE STOMACH. *(E.)*

Pritchard, G. R. (U. Washington Sch. Med., Seattle), C. A. Griffith and H. N. Harkins. *Surg Gynec Obstet* 126(4):791-798, 1968.

In anesthetized, healthy, adult mongrel dogs (18-25 kg) the vagus nerves were stimulated at 4 specific anatomic levels; the left and right vagi in the neck; the branches of the esophageal plexus in the thorax; the anterior and posterior vagal trunks in the abdomen; and the terminal branches from the anterior and posterior gastric truncal divisions to the stomach. Neutral red was given i.v. at the time of vagal stimulation to demonstrate gastric secretion (appearance of red dye on the innervated areas of the gastric mucosa exposed at gastrostomy). After the injection of neutral red and before vagotomy, the dye appeared on the mucosa of the gastric fundus and corpus within 2-20 min. After vagotomy, the secretion ceased within 10-30 min. After vagal stimulation the dye was secreted by the fundus and corpus within 10-80 min; maximal secretion occurred in the distal part of the gastric corpus (maximal parietal cell concentration). Secretion by the proximal gastric fundus was relatively scant and sometimes absent, and dye was never secreted by the antrum. After initial localized responses to vagal stimulation, continued stimulation produced generalized secretion by the entire fundus and corpus within 2-20 min in 3 of 12 cases in which stimulation was applied to branches of the esophageal plexus and in 6 of 7 stimulations of the vagal trunks; generalized secretion did not appear with stimulation of the terminal branches innervating the fundus and proximal corpus, but did appear with stimulation of the branches innervating the distal corpus. Stimulation of distal branches leading to the antrum produced a weak but definite generalized secretion. This delayed response is ascribed to the vagal release of gastrin. These findings indicate that the fibers of each cervical nerve are distributed to the entire stomach through the esophageal plexus, where segmental innervation occurs. Overlapping progressively decreases from the esophageal plexus to the stomach, where innervation is highly segmental. These findings are pertinent to the occurrence of various anatomic types of incomplete vagotomy and the adequacy of each type as determined by the specific area of stomach which remains innervated.

6668 THE WIDTH OF THE COMMON BILE DUCT. *(E.)*

Leslie, D. (Royal Melbourne Hosp., Australia). *Surg Gynec Obstet* 126(4):761-764, 1968.

The width of the common bile duct was measured in 202 patients undergoing operations on the biliary tract in order to correlate the width of the duct with the presence or absence of duct disease. Measurements were taken with the duct flattened against the side of a ruler, 1 cm below the junction of the cystic and common ducts; this gives the flat-width of the duct (one half the circumference), from which the true external diameter can readily be calculated. The average width of 49 diseased ducts was 21 mm (maximum 58 mm and minimum 9 mm); the average width of 153 nondiseased ducts was 12 mm (maximum 17 mm and minimum 5 mm). These results indicate that ducts which are less than 9 mm contain no disease in the distal portion, those greater than 17 mm always contain disease, and those in between may or may not contain disease with the probability of disease increasing rapidly with widths greater than 14 mm.

6669 DISACCHARIDASE ACTIVITY OF THE INTESTINAL MUCOSA IN INFANTS. *(Sp.)*

Sampayo, R. R. L. (Clin. Hosp., Buenos Aires, Argentina), C. Sosa Miatello, J. Higa and I. De Larrechea. *Prensa Med Argent* 55(19):891-897, 1968.

Disaccharidase activities were determined in samples of the jejunal mucosa, obtained with a Crosby capsule, from 15 normal infants and 22 infants with malnutrition, aged 2-25 months. The following mean values and ranges were obtained for normal infants: lactase 21.17 (1.37-70.70) U/min/g protein; invertase 47.85 (11.50-104) U/min/g protein; and maltase 164.58 (36.3-340) U/min/g protein. The corresponding values for 15 infants with grade-2 malnutrition were 13.0 (0.00-33) U/min/g protein; 30.30 (7.4-76.3) U/min/g protein; and 121.1 (29.6-344) U/min/g protein, resp. Values for children with grade-1 malnutrition tended to be intermediate, suggesting the possibility that malabsorption played a role in the poor development of these children.

6670 **EFFECT OF VAGAL STIMULATION ON ENTEROCHROMAFFIN CELL GRANULATION IN THE GUINEA PIG SMALL INTESTINE.** *(E.)* Tansy, M. F. (Temple U. Sch. Dent. Pharm., Philadelphia, Pa.), A. S. Miller and A. Stein. *J Pharm Sci* 57(11):1906-1909, 1968.

The effects of vagal stimulation on the granulation of the duodenal enterochromaffin cell system were studied in 76 healthy male guinea pigs, weighing 350-450 g, in order to elucidate the mechanism of serotonin release in the bowel. Peripheral vagal stimulation at either 10 volts, 25 cycles/sec or 1 volt, 250 cycles/sec for 1 msec produced a significant reduction in argentaffin-positive enterochromaffin cell granularity. The mean decrease in granularity ranged from 73% with low-intensity current to 60% with the higher current strength, while a 50% reduction was seen in animals subjected to direct electrical stimulation of the duodenal musculature. These results indicate that vagal stimulation *per se* and not hydrochloric acid secretion, increased intraluminal pressure, or hyperperistalsis affects the granulation of the enterochromaffin cells. Since the administration of atropine sulfate had no effect on the degranulation, it is suggested that the vagal effect may occur via a noncholinergic mechanism.

6671 **SOME ASPECTS OF THE FINE STRUCTURE OF THE GALL BLADDER EPITHELIUM OF THE MOUSE.** *(E.)* Yamada, K. (Nagoya U. Sch. Med., Japan). *Okajima Folia Anat Jap* 45(1):11-19, 1968.

Gallbladders from 10 adult inbred mice of both sexes were examined by electron microscopy and the findings were correlated with the cytophysiology of the gallbladder epithelium. Epithelial cells with characteristic spherical or oval, pale, cytoplasmic droplets (0.5-1.2 μ in diameter), enclosed by a thin circular membrane containing fine granular material with occasional dark vesicular elements, were seen. The pale cytoplasmic droplets were often observed close to the Golgi apparatus. Granular, vesicular, and amorphous materials contained in the dilated Golgi cisternae and resembling these pale cytoplasmic droplets suggested a Golgian origin. In some cells, droplets were clustered near the cytoplasmic apex and their limiting membranes were seen to coalesce with the apical plasma membrane, with discharge of their contents into the lumen of the gallbladder. In some epithelial cells, projections at the apical end of the cytoplasm were observed. These findings suggest a possible excretory or secretory function in the mouse gallbladder epithelium.

6672 **THE EFFECTS OF HYDROCORTISONE AND ACTH ON THE GASTRIC MUCOSA OF THE RAT.** *(E.)* Crean, G. P. (Clin. Endocrin. Res. Unit, U. Edinburgh, Scotland). *Scand J Gastroent* 2(4):305-310, 1967.

The effects of administering hydrocortisone (4 or 8 mg daily s.c. for 22 days) and ACTH (4 or 8 U daily s.c. for 36 days) on the gastric mucosa of male Wistar rats were assessed from the changes noted in the weight of the whole stomach, the surface area of the fundus, the height and volume of the fundic mucosa, and the total parietal cell mass. Both treatments resulted in reduced stomach weights and reduced height and volume of the fundic mucosa, but the surface area of the fundus and the parietal cell mass were unaffected; no gastric ulcers were noted. Although the larger doses of the drugs generally exerted a greater effect than the smaller doses, no statistically significant differences between the 2 doses were noted except that the 8 mg dose of hydrocortisone exerted a greater effect on stomach weight than the 4 mg dose. These results dispute the view that the parietal cell population of the stomach can be increased by the administration of corticosteroids or ACTH. In fact, large amounts of adrenocortical hormones may inhibit the growth of the gastric mucosa.

6673 **ULTRASTRUCTURE OF THE GASTRIC MUCOSA IN HEALTHY HUMAN SUBJECTS AND IN PATIENTS WITH PERNICIOUS ANEMIA.** *(E.)* Tichy, J. (Med. Fac., Charles U., Hradec Kralove, Czechoslovakia) and M. Hradsky. *Gastroenterologia* 107(6):379-388, 1967.

The ultrastructure of the gastric mucosa was compared in 7 patients (4 males and 3 females) with pernicious anemia and 5 healthy controls (3 males and 2 females). All 12 subjects were 60-65 yr old. The chief cells, mucoid cells, parietal cells, and argyrophil cells of the normal gastric mucosa are described in detail. The most prominent abnormalities seen in the patients with pernicious anemia were a chronic inflammatory infiltration, metaplasia of the surface epithelial cells to an intestinal type, a generalized cellular atrophy and disintegration, and absence of the parietal cells. Gross alterations were encountered in the chief cells: focal areas of high density in variable-sized secretory granules which showed an irregularly tortuous surface membrane, vacuolization of the cytoplasm, and accumulations of nuclear chromatin near the nuclear membrane.

6674 **ANATOMICAL VARIATIONS IN THE CONNECTIONS OF THE HEPATIC DUCTS AS REVEALED BY CHOLANGIOGRAPHY.** *(Pol.)* Kocon, T. (Inst. Norm. Anat., Acad. Med., Warsaw, Poland). *Pol Przegl Chir* 40(6):654-660, 1968.

Based on 597 operative cholangiograms recorded during the period 1959-66, the anatomical configuration of the human hepatobiliary tree is classified into 4 main types. In 376 completely normal cholangiograms and 31 others (total of 407 or 68.2%), the choledochus was formed by 2 hepatic ducts, one from the right and one from the left. In 22 of these 31 abnormal cases, the right hepatic duct was extremely short, and in 9 others the cystic duct terminated in the right hepatic duct or at the junction of the 2 hepatic ducts. In 86 other cases (14.4%), 3 hepatic ducts were found to join at the same level, 2 coming from the right lobe and one from the left, and in another 79 cases (13.2%) there were also 3 hepatic ducts (2 from the right and one from the left) but they did not meet at the same level. In 13 cases (2.2%), the choledochus was formed by 4 hepatic ducts. Some cases with accessory hepatic ducts were found in all of the above groups (total of 3.4%), and 12 final cholangiograms could not be classified into any of the 4 main types.

6675 **AGAR ELECTROPHORETIC AND IMMUNOLOGICAL STUDIES ON EXTRACTS OF NORMAL HUMAN COLONIC MUCOSA.** *(Ger.)* Scheiffarth, F. (U. Hosp., Erlangen, Germany), H. Götz, H. Warnatz and G. Autenrieth. *Z Gastroent* 5(5):291-298, 1967.

Mucosal extracts from the large intestine of 14 cadavers, 7 biopsied patients, and 2 patients with cancer of the colon were studied by immunoelectrophoresis and immunodiffusion. Extracts subjected to agar electrophoresis were separated into 8 different fractions, one of which contained 3 subfractions. High concentrations of mucoproteins and very small amounts of lipoproteins were found between the site of application and the albumin region. Oxidase activity occurred primarily in the α_2-globulin region, but lower concentrations were also present in the α_1- and β_1-globulin regions. Alkaline phosphatase activity was found in the α-globulin region, but in much higher concentrations than in serum. Two sharply defined fractions, one in the β_2- and the other in the γ-globulin region, had amylase activity. This might be either mucosal or bacterial in origin. Bright zones in the α_2-globulin region which stained with iodine were probably glycoproteins. Results of immunoelectrophoresis and Ouchterlony immunodiffusion showed that almost all proteins in these extracts were immunologically related to human serum proteins. Only 1-2 fractions belonging to the α_2/β_1-globulin system appeared to be organ-specific. Slices of mucosa incubated with labeled rabbit immune serum showed fluorescence in the cytoplasm, but not the nucleus, of goblet cells. This may be due either to the way in which the mucosa was treated or to differences in the antibody-inducing action of extract constituents.

6676 **A CONTRIBUTION TO THE PROBLEM OF HISTOGENESIS OF THE ESOPHAGEAL EPITHELIUM IN MAMMALS.** *(E.)* Seifertová, V. (Fac. Med., Charles U., Prague, Czechoslovakia). *Folia Morph* 16(4):364-372, 1968.

The development of the esophageal epithelium was studied by light microscopy in sections from human embryos (2-6 months of embryonic development) and from the pre- and postnatal stages of rat fetuses (from the 13th day to the end of gestation and up to 19 days after birth). The epithelial development was divided into 3 morphological stages: 1) the 1st stage, characterized by a high, 2-3-layered epithelium, preserved in the human up to the 35-mm embryo and in the rat up to the 15th day of gestation; 2) the 2nd stage, in which the superficial layers changed to columnar, cuboidal, or ovoid cells with cilia in the human, and squamous cells in the rat; in the human, the basal cell layer assumed its final cuboidal shape by the 70-mm stage, and the lower cells became vesicular; 3) the 3rd stage, characterized by a stratified squamous epithelium which differentiated at the base of the mucosal folds in 80-mm human fetuses and 15-day-old rat fetuses. These changes occurred simultaneously along the entire course of the esophagus with no evident morphological differences at any time between the upper and lower portions of the esophagus. This simultaneous formation of squamous epithelium along the entire course of the esophagus tends to disprove the ectodermal theory of the proliferation of squamous cells from the oral cavity caudally into the esophagus.

6677 **CELLULAR STORES OF GASTRIC HISTAMINE IN THE DEVELOPING RAT.** *(E.)* Håkanson, R. (Dept. Pharmacol., U. Lund, Sweden), C. Owman and N. O. Sjöberg. *Life Sci* 6(23):2535-2543, 1967.

The development and cellular localization of gastric histamine and the appearance of histidine decarboxylase in the stomach wall of the developing rat were studied in adult albino rats, rat fetuses of 17-22 days gestation, and young rats 0-40 days old. Histamine was determined histochemically in pieces of the glandular portion of the stomach which were freeze-dried and exposed to o-phthaldialdehyde vapor (converting histamine into a fluorophore emitting an intense blue light). Histamine was also determined by the conventional fluorimetric technique. Histidine decarboxylase was determined by a fluorimetric and radiometric technique. The concentration of gastric histamine in the fetal and neonatal rats was low (3 μg/g); at 2 weeks after birth, the concentration increased markedly, reaching 50% of the normal adult concentration by day 30 (adult concentration = 20 μg/g), and 100% at day 40. Significant histidine decarboxylase activity was found in the stomach of the fetal rat, which fell to immeasurable levels immediately after birth; adult levels were established, however, at 1-2 months of age. Fluorescent mast cells in the stomach wall were not very evident for the 1st few days after birth, but increased in number to adult levels by the 3rd week, becoming located in the superficial region of the mucosa as well as in the submucosa and the muscular layers. Low-intensity fluorescent enterochromaffin cells were evident in the epithelium at the bottom of the developing gastric glands at 5-6 days of age, which likewise increased in number to adult levels at the 3rd week, although the intensity of fluorescence remained lower than that in the adult animal. Good correlation was noted, therefore, between the histochemical and chemical determinations of histamine and histidine decarboxylase in the developing rat stomach.

6678 **ENTEROCHROMAFFIN CELLS AND 5-HYDROXYTRYPTAMINE CONTENT OF THE COLON OF MICE.** *(E.)* Schofield, G. C. (Dept. Anat., Monash U., Clayton, Victoria, Australia), A. K. S. Ho and J. M. Southwell. *J Anat* 101(4):711-721, 1967.

Enterochromaffin cells in the mucosa of proximal, intermediate and distal segments of mouse colon were demonstrated in formalin-fixed tissues irradiated by ultraviolet light or stained by the hexamine-silver and diazonium techniques. In fluorescent and hexamine-silver impregnated preparations, 2 varieties of enterochromaffin cells were seen: 1) intraepithelial cells with finely granular cytoplasm, often with a basal process of variable length directed toward the fundus of the intestinal glands; and 2) circular or ovoid cells with coarsely granular, usually acidophilic, cytoplasm, occurring in the epithelium or lamina propria; some of the latter showed staining properties typical of mast cells. Type 1 cells were predominant in the distal colon while type 2 were more numerous in the proximal colon. In most mice, estimates of the number of fluorescent cells and of the serotonin content showed a gradient along the length of the colon with highest values in the proximal colon. However, there was no consistent correlation between numbers of fluorescent cells and the amount of serotonin in the areas of colon studied, and marked differences were noted between individual animals. Injection of 5.0 mg/kg reserpine i.p. 4, 12 or 24 hr prior to sacrifice significantly reduced the serotonin content of colon tissue to 1/4 to 1/3 of the control level. At 12 and 24 hr after drug administration, a low cell count was observed throughout the colon, but the reduction was not statistically significant. The chromogenic and fluorescent material of the enterochromaffin cells is unlikely to be serotonin exclusively and may be due to the presence of other related substances. Studies on the staining and fluorescent properties of several biological materials, including monoamines, in formalin-fixed gelatin models indicate that the staining techniques used are not specific for serotonin.

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6679 EXPERIMENTAL STUDIES ON LYMPH CIRCULATION IN THE LIVER. XI. ON WHAT PART OF THE EFFERENT HEPATIC LYMPH PATHWAYS CAN AN EFFECTIVE SHUNT BE MADE FOR ASCITES PRODUCED BY POST-HEPATIC VENOUS STENOSIS. *(Fr.)* Mallet-Guy, P. (Surg. Clin., Med. Fac., Lyon, France) and Y. Mandairon. *Lyon Chir* 63(5):652-666, 1967.

Manometric measurements made on normal dogs showed that pressure in the hepatic lymph ducts decreased progressively, from 12.1 cm H_2O in the preganglionic lymph ducts to 6.6 cm in the postganglionic ducts, 2.7 cm at the convergence of the abdominal lymph ducts, and 0 or negative at the end of the thoracic duct. Lymph normally flowed at a rate of 1.2 ml/min in hepatic lymph ducts. Increases in these pressures and a tripling of the flow rate in the lymph ducts were produced by either clamping or ligating the thoracic part of the vena cava. Ascites, induced in dogs by ligating the thoracic part of the inferior vena cava, either disappeared or the volume of fluid decreased after the cisterna chyli was opened and drained. At the same time, portal pressures decreased from 10.15-16.4 cm H_2O to a mean of 12.5 cm. This would seem to be more effective than drainage of the cervical thoracic duct in the treatment of intractable ascites and portal hypertension.

6680 PRE- AND POSTNATAL DEVELOPMENT OF THE GANGLION CELLS OF THE RECTUM AND ITS SURGICAL IMPLICATIONS. *(E.)* Smith, B. (Ohio State U. Coll. Med., Columbus). *J Pediat Surg* 3(3):386-391, 1968.

The chronological maturation of the ganglion cells of the sigmoid colon and rectum during pre- and postnatal development was studied in tissue from 30 fetuses as well as from premature and full-term infants who came to autopsy. Any patient with an anomaly of the nervous system or a history suggestive of achalasia, pyloric stenosis, or Hirschsprung's disease was excluded. Speciemens were fixed and stained with Kluver-Barrera stain and Bodian silver impregnation. In the 1st trimester of gestation, no neuroblasts were seen in the 10 mm embryo; at 12 mm, neuroblasts could be seen in Auerbach's plexus and at 50 mm, clusters of neuroblasts were noted in Meissner's plexus. Cells stained darkly with the characteristics of early neuroblasts and no evidence of prolongation. In the 2nd trimester, in the pyloric area, the nuclei became paler and the amount of cytoplasm increased while darkly stained nuclei were still numerous in the rectosigmoid area. Auerbach's plexus was better defined than Meissner's plexus. In the 3rd trimester, although Auerbach's plexus still contained early neuroblasts, most of the neurons of the cardia, pylorus, and sigmoid colon had aggregated into ganglia with thin capsules, and Schwann cells were seen in the more proximal sections. Occasional multipolar cells were seen at the periphery of the ganglia. From birth to 1 month of age, maturation of cells continued, being more apparent in pyloric sections than in the colon or lower rectum. Up to 2 yr of age, large stellate neurons were abundant, and differences in sections from the pylorus and lower rectum were not as evident. In childhood (2-5 yr), large, fully-mature cells were abundant with profuse branching of dendrites, although single branching was still observed. The diagnosis of Hirschsprung's disease is therefore justified only when ganglion cells normal for the age of the child are absent from the ganglia of both enteric plexi, as viewed on an adequate rectal biopsy.

6681 THE ARTERIAL ANATOMY OF THE PANCREAS AND ITS VARIATIONS. *(Fr.)* Mellière, D. (Bichat Hosp., Paris, France, pp. 207-209 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

6682 DISTRIBUTION OF THE LYMPHATICS IN THE LIVER. *(Fr.)* Szabo, G. (Inst. Traumatol., Budapest, Hungary). pp. 12-16 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

6683 THE PERICAPILLARY SPACE OF DISSE IN THE LIVER. *(Fr.)* Rouiller, C. (Sch. Med., Geneva, Switzerland) and P. Nicolescu. pp. 3-11 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

6684 THE LYMPHATIC CIRCULATION OF THE PANCREAS. *(Fr.)* Murat, J. (Ed. Herriot Hosp., Lyon, France) and M. Barbier. pp. 81-85 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

6685 CYTOPLASMIC ANTIGENS IN THE LIVER. *(Rus.)* Kashkin, K. P. (Inst. Med. Radiol., Acad. Med. Sci. USSR, Obninsk). *Biull Eksp Biol Med* 64(12):66-70, 1967.

6686 THE CHARACTERISTIC ULTRASTRUCTURE OF THE CUTICLE OF THE EPITHELIAL CELLS OF THE SMALL INTESTINAL MUCOSA IN DOGS, RATS AND MAN. *(Rus.)* Skliarov, Ia. P. (Lvov Med. Inst., USSR) and Ie. Ie. Iaremko. *Biull Eksp Biol Med* 65(1):112-115, 1968.

6687 A MORPHOLOGICAL AND FUNCTIONAL STUDY OF THE DUODENOJEJUNAL WALL BEFORE AND AFTER ELECTRICAL STIMULATION. *(It.)* Labò, G. (Inst. Gen. Med. Clin., U. Bologna, Italy), T. Posteli, G. Gasbarrini, F. Faggioli and N. Melchionda. *Arch Ital Mal Appar Dig* 34(3):263-267, 1967.

6688 THE ANATOMY OF THE HAUSTRA OF THE HUMAN COLON. *(E.)* Pace, J. L. (Royal U. Malta). *Proc Roy Soc Med* 61(9):934-935, 1968.

6689 THE "SPHINCTERS" OF THE DIGESTIVE SYSTEM. *(E.)* Didio, L. J. A. (Med. Coll. Ohio, Toledo) and M. C. Anderson. Baltimore, Williams and Wilkens, 1968. 255 pp.

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6690 GASTRIC HISTIDINE DECARBOXYLASE. *(E.)* Powell, P. E. (U. Toronto, Canada) and M. A. Kumar. *Anal Biochem* 22(3):485-492, 1968.

6691 THE INTESTINAL EPITHELIAL CELL. *(E.)* Smyth, D. H. *Ulster Med J* 37:107-125, 1968.

6692 ELECTRON MICROSCOPIC STUDIES ON THE RETICULOENDOTHELIAL SYSTEM OF THE RAT LIVER FOLLOWING MOBILIZATION HYPERLIPEMIA AND THE INFUSION OR FEEDING OF FAT. *(Ger.)* Lapp, H. (Inst. Path., U. Frankfurt/Main, Germany). *Verh Deutsch Ges Path* 51:394-401, 1967.

6693 ELECTRON MICROSCOPIC FINDINGS IN THE SMALL INTESTINAL AND ENDOMETRIAL EPITHELIUM OF THE MOUSE FOLLOWING ADMINISTRATION OF COLCHICINE. *(Ger.)* Stolpmann, H. J. (Inst. Path., Free U. Berlin, Germany) and H. J. Merker. *Verh Deutsch Ges Path* 51:401-406, 1967.

6694 KINETIC MODELS FOR CELL POPULATIONS IN THE INTESTINAL MUCOSA OF GERM-FREE ANIMALS. *(It.)* Paleani Vettori, G. (Inst. Radiobiol., U. Rome, Italy), M. Colosimo, G. Briganti, V. Monaco, O. Cimini and M. Trinci. *Nunt Radiol* 33(8):875-884, 1967.

6695 PHYSIOLOGIC CAPACITY OF THE STOMACH DURING THE FIRST THREE DAYS OF LIFE. *(E.)* Caldito, J. C. (Santo Tomas U. Hosp., Manila, Philippines), C. L. Buenaviaje and R. S. Songco. *Santo Tomas J Med* 22(6):367-371, 1967.

6696 STRUCTURAL ANALYSIS OF THE ESOPHAGUS WITH REGARD TO THE OBSERVATIONS DURING SURGERY. *(Ger.)* Stelzner, F. (Surg. Clin., U. Hamburg-Eppendorf, Germany) and W. Lierse. *Zbl Chir* 92(26a):1857-1860, 1967.

6697 HISTOCHEMICAL ANALYSIS OF THE ARGYROPHILIC SUBSTRATUM IN HUMAN HEPATIC RETICULIN. *(E.)* Velican, D. ("N. G. Lupu" Inst. Intern. Med., Acad. Sci., Bucharest, Romania). *Rev Roum Med Intern* 4(4):267-271, 1967.

See also: 7061

6698 ABSORPTION AND LYMPHATIC TRANSPORT OF CHOLESTEROL IN THE RAT. *(E.)* Sylvén, C. (Chem. Ctr., U. Lund, Sweden) and B. Borgström. *J Lipid Res* 9(5):596-601, 1968.

Fasted male albino rats with thoracic duct fistulae (for lymph) collection) and gastric cannulae (for administration of test meals) were fed triolein with or without varying amounts of cholesterol-4-^{14}C or cholesterol-^{3}H. Lymph sampling was done at hourly intervals during the 1st 8 hr after feeding, and 1 sample was collected 8-16 hr after feeding. In the fasting state the cholesterol transported via the thoracic duct was 0.87 μmole/hr. A test meal of 800 μmoles of triolein gave a maximum rate of transport of cholesterol of 1.65 μmoles/hr. The maximal cholesterol transport rate increased further to 5 μmoles/hr with the addition of 50 μmoles of cholesterol to the 800 μmole triolein meal. The exogenous fraction of the cholesterol transported increased linearly with increasing cholesterol load, constituting 90% of the total cholesterol transported at the highest dose (100 μmoles). An almost constant fraction (0.4) of the dietary cholesterol was recovered in the thoracic duct lymph in 24 hr irrespective of the dose fed, from a trace up to 100 μmoles of cholesterol in 800 μmoles of triolein. It is concluded that cholesterol absorption has the characteristics of a passive diffusion process.

6699 DISACCHARIDE ABSORPTION BY AMPHIBIAN SMALL INTESTINE *IN VITRO.* *(E.)* Parsons, D. S. (Dept. Biochem., U. Oxford, England) and J. S. Prichard. *J Physiol* 199(1):137-150, 1968.

In vitro studies were made of the digestion and absorption of disaccharides in the small intestine of some species of common amphibia (*Rana temporaria, Rana pipiens* and *Bufo vulgaris*) which were perfused through the vascular system. While maltase and trehalase activity were found in the intestine of all these species, small amounts of sucrase were present only in the intestine of *R. pipiens* and no lactase was found in any of the animals studied. Maltose absorption was accompanied by the appearance of glucose in both the vascular effluent and the intestinal lumen. A Michaelis–Menten type of relation was discovered between the rate of absorption of glucose and the concentration in the luminal fluid of either disaccharide or free glucose. When expressed in molar units, the apparent Km for glucose transport was about one fifth that of the disaccharidase and the maximum rate of glucose transport was less than the maximum rate of disaccharide hydrolysis. It is concluded that in the amphibian, intestinal disaccharide hydrolysis and glucose transport are either functions of separate subcellular systems which are closely related spatially, or different facets of the activity of a common system.

6700 FORMATION OF A COMPLEX BETWEEN VALINE AND INTESTINAL MUCOSAL LIPID; ITS POSSIBLE ROLE IN VALINE ABSORPTION. *(E.)* Reiser, S. (Indiana U. Sch. Med., Indianapolis) and P. A. Christiansen. *J Lipid Res* 9(5):606-612, 1968.

The ability of lipid extracted from the rat small intestine (heptane-soluble fraction) to bind valine was determined using L-valine-^{14}C, and the properties of this complex were compared with those of the intact, *in vitro* (everted intestinal sac) transport system for valine. After incubation in a valine-containing medium (3 mM), the extracted lipid contained an average of 3.63 μmoles of valine/100 mg lipid. Cyanide (0.002 M), 2,4-dinitrophenol (0.0002 M), and anaerobic conditions had little effect on this process. Valine uptake by the lipid fraction was complete after 2.5 min. Of a number of sugars and amino acids tested, isoleucine, methionine and leucine were the most potent inhibitors of valine uptake into lipid; the inhibition appeared to be competitive in nature. A similar uptake of glucose into the mucosal lipid was not inhibited by leucine, methionine, or isoleucine, but was inhibited by galactose. Various phosphoglycerides, used in place of mucosal lipid, were able to carry 20-150 times as much valine into the heptane-soluble fraction as were other lipid classes. The results indicate that intestinal mucosal lipid can bind valine under a variety of experimental conditions, suggesting a relation between valine-lipid complex formation and valine transport.

6701 THE DIFFERENTIAL EFFECTS OF SODIUM, LITHIUM AND POTASSIUM ON THE ABSORPTION OF GLUCOSE, XYLOSE AND WATER FROM RAT JEJUNUM *IN VIVO.* *(E.)* Rosensweig, N. S. (Johns Hopkins U. Sch. Med., Baltimore, Md.) and T. R. Hendrix. *Johns Hopkins Med J* 121(6):412-420, 1967.

In adult male Wistar rats (400-500 g), the effect of sodium replacement by lithium and potassium on the intestinal absorption of glucose and xylose *in vivo* was determined using a perfused, exteriorized loop of intact jejunum. Studies were carried out for 6 consecutive 15-min intervals. Test solutions were prepared by adding D-glucose or D-xylose to NaCl, LiCl, or KCl solutions (150 mM) to obtain sugar concentrations of 6 or 30 mM. In the 1st group of experiments, the absorption of glucose (30 mM) and water was compared to the absorption of xylose (30 mM) and water from NaCl, LiCl, and KCl solutions. With glucose, lithium and potassium had similar inhibitory effects (as compared to sodium) on sugar and water absorption and sugar concentration in the loop at the end of the 15-min absorption period. With xylose, potassium again inhibited both sugar and water absorption, but lithium inhibited only water absorption with no impairment of sugar absorption. Furthermore, the xylose concentration in the loop at the end of the observation period was significantly less than with sodium. Repeating the experiments with 6 mM sugar solutions gave similar results. When water absorption from sugar-electrolyte solution was compared to that from electrolyte solution alone, it was noted that at 6 mM, water absorption was unaffected by either sugar. At 30 mM, glucose in NaCl was found to enhance water absorption over that with isotonic NaCl alone, while xylose in NaCl decreased water absorption. With glucose (30 mM) in LiCl, the enhanced water absorption was abolished, while water absorption with xylose (30 mM) in NaCl and LiCl was less than from the respective electrolyte solutions alone. It is suggested that the effect of lithium may be related to its inability to substitute for sodium in the active transport mechanism which pumps sodium out of the cell.

6702 THE EFFECT OF DRUGS ON THE RATE OF PERSORPTION. *(E.)* Volkheimer, G. (1st Med.

U. Clin., Charité Hosp., Berlin, Germany), F. H. Schulz, I. Hofmann, J. Pieser, O. Rack, G. Reichelt, W. Rothenbaecher, G. Schmelich, B. Schurig, G. Teicher and B. Weiss. *Pharmacology* 1(1):8-14, 1968.

Persorption of solid, undissolved food particles was studied quantitatively in 34 male and female subjects (mean age 24 yr) given 200 g of corn starch p.o. and the effects of prostigmine (0.5 mg), caffeine (200 mg) and atropine (0.5 mg) were determined. A procedure for measuring the number of starch granules in 10-ml samples of venous blood is described. Simultaneous administration of either prostigmine or caffeine with the corn starch resulted in increased numbers of starch granules in venous blood analyzed within 30-240 min; the differences were statistically significant after 30, 60 and 120 min with prostigmine and after 60 and 150 min with caffeine. The number of starch granules in the venous blood was lower when atropine was injected 20 min after administration of the starch; the values after 60, 90 and 150 min differed significantly from control values for starch alone. These data indicate that the motor activity of the gastrointestinal tract is important for the process of persorption.

6703 **THE EFFECT OF ETHANOL ON GLUCOSE ABSORPTION IN ISOLATED SEGMENTS OF RAT SMALL INTESTINE.** *(Pol.)* Panowicz, H. (1st Clin. Int. Dis., Pomeranian Acad. Med., Szczecin, Poland). *Roczn Pom Akad Med Swierczewski* 13:385-401, 1967.

Glucose absorption by 20-cm segment of distal duodenum isolated from male and female albino rats (200-310 g body weight) was studied in the Wiseman apparatus as modified by Smyth and Whaler (*J. Physiol.* 121:2P, 1953). In a group of 44 experimental rats and 13 controls, addition of 0.2-20% ethanol to the medium bathing the intestinal segment (which contained 200 mg% glucose on both sides) inhibited active transport of glucose by 17.6-100%. In another group of 18 experimental rats and 6 controls, addition of 1-10% ethanol to the medium (which contained 300 mg% glucose within the lumen and 100 mg% glucose on the serosal side) inhibited both active transport and diffusion by 35.2-70.4%. Finally, 10 rats were given ethanol (20% aqueous solution, 1.5 ml/100 g body weight p.o. 6 days/week) for 13 weeks before preparation of the duodenal segments; this treatment inhibited active transport of glucose by 20% compared to controls. Alcohol is concluded to have a direct effect on the transport processes in the intestinal mucosa.

6704 **EFFECT OF PANCREATIN ON THE ABSORPTION OF FE^{59} IN RATS WITH FATTY LIVER.** *(E.)* Murray, M. J. (U. Minnesota Med. Sch., Minneapolis) and N. Stein. *Amer J Dig Dis* 13(6):527-529, 1968.

Fatty livers were induced in 53 female Sprague-Dawley rats by feeding the Zaki C-8 choline-deficient diet (containing 30 μg iron/g) for 56 days, at which time animals were divided into 4 groups for absorption studies. Group 1 rats were maintained on the C-8 diet only; group 2 rats received the C-8 diet with pancreatin (Viokase), 2 g/day; group 3 rats received the C-8 diet with added bromelin (Ananase), 2 g/day; and group 4 rats received the C-8 diet with 12 mg choline chloride and 50 μg ferrous citrate per gram of diet. The pancreatin given to group 2 rats also contained 12 mg/g choline and 50 μg/g Fe. The day following initiation of the special diets, rats were given a test dose of 0.25 μC of ^{59}Fe with 0.25 mg of ferrous citrate. Total body radioactivity was determined immediately and 10 days later. No significant depression of iron absorption was observed in any of these groups, indicating that pancreatic extracts are unable to depress the increased Fe absorption in rats with fatty liver induced by choline deficiency.

6705 **INTESTINAL ABSORPTION OF LONG-CHAIN FATTY ACIDS.** *(E.)* Miyazaki, I. (Kumamoto U. Med. Sch., Japan). *Kumamoto Med J* 20(4):168-177, 1967.

The intestinal absorption of long-chain fatty acids was investigated in dogs with cannulated or intact thoracic ducts using ^{131}I-triolein as a tracer and gas-liquid chromatography to analyze the fatty acid composition of the plasma and lymph. A silicone-rubber tube was employed to catheterize the portal vein and thoracic duct. Following ingestion of ^{131}I-triolein, radioactivity was consistently detected in the portal vein. After enteric administration of linseed oil, linolenic acid was also found in the portal vein. These findings support the view that the portal system contributes to the intestinal absorption of long-chain fatty acids.

6706 **TIME TO ACHIEVE STEADY-STATE LUMINAL GLUCOSE CONCENTRATION DURING INTESTINAL LAVAGE.** *(E.)* Zatzman, M. L. (U. Missouri Sch. Med., Columbia) and J. D. Moore. *J Appl Physiol* 25(1):95-97, 1968.

A system is described for estimating the time required to achieve a steady-state glucose concentration during lavage of the hamster small intestine *in vivo*. In conjunction with polyethylene glycol (PEG) analysis, the record can be converted to a continuous presentation of elimination and absorption rates. The glucose-analyzing system is composed of the usual modules of an AutoAnalyzer with minor changes in the manifold to produce full-scale deflections with glucose concentrations of either 150 or 1,500 mg/100 ml. Using an intestinal perfusion rate of 0.5-0. 6 ml/min, a steady-state concentration is achieved in 20 min. The system performed accurate, continuous determinations of glucose concentration "on line" with a delay of 8-9 min between the initiation of sampling and appearance of the record. Full-scale deflection requires only 45 sec of continuous sampling. This system is ideal for the study of drug effects on glucose absorption.

6707 **ANTIBODIES TO INTESTINAL MICROVILLOUS MEMBRANES. II. INHIBITION OF INTRINSIC FACTOR-MEDIATED ATTACHMENT OF VITAMIN B$_{12}$ TO HAMSTER BRUSH BORDERS.** *(E.)* Mackenzie, I. L. (Dept. Med., Boston U., Mass.), R. M. Donaldson Jr., W. L. Kopp and J. S. Trier. *J Exp Med* 128(2):375-386, 1968.

Incubation of brush borders isolated from distal hamster small intestine with rabbit antisera to distal (AbD), but not proximal, intestinal microvillous membranes inhibited the attachment of intrinsic factor (IF)-^{57}CoB$_{12}$ complex to the absorptive cell surface of the brush borders. Inhibition of IF-mediated uptake of vitamin B$_{12}$ varied inversely with the dilution of antiserum. Excess of IF-bound ^{57}CoB$_{12}$ could overcome the inhibitory effect of AbD. The affinity constant for the inhibitor, calculated from the Lineweaver-Burk plot, was of the same order of magnitude (1.69 x 10^{-9} M) as the equilibrium constants for other antigen-antibody reactions. The inhibitory factor in AbD was absorbed from antisera by brush borders isolated from the distal, but not proximal, hamster intestine. Gel filtration and DEAE-cellulose chromatography demonstrated that immunoglobulin G contained the inhibitory factor. Antisera in concentrations 4-10 times greater than that required to inhibit brush border uptake of IF-bound B_{12} failed to inhibit the binding of B_{12} by intrinsic factor, did not form a complex with IF-bound $^{57}CoB_{12}$ which could be precipitated by ammonium sulfate, and did not alter the anodal migration of IF-$^{57}CoB_{12}$ during vertical starch gel electrophoresis. Thus, hamster IF did not absorb the inhibitory factor in AbD. Sucrase and maltase activities of the brush border were not inhibited by glucose transport across everted sacs of hamster intestine was not impaired by antisera capable of completely blocking uptake of IF-bound B_{12}. These observations indicate that the inhibitory factor in AbD competes with the IF-B_{12} complex for a common binding site or receptor on the surface of the distal brush border of hamster intestine.

6708 INTRACELLULAR ACTIVITY OF L-PHENYLALANINE DURING INTESTINAL TRANSPORT. *(E.)* Faelli, A. (Inst. Gen. Physiol., U. Milan, Italy), G. Esposito and V. Capraro. *Med Pharmacol Exp (Basel)* 17(5): 483-488, 1967.

To determine whether a contemporary increase in osmotic activity occurs during accumulation of actively transported substances in the intestinal cell, everted jejunal sacs from semistarved Wistar rats were initially incubated for 1 hr at 28C in 40 ml of Krebs-Henseleit solution with the serosal fluid consisting of 2 ml of the same solution with trace amounts of ^{3}H-inulin (for determination of the extracellular space). In another experiment, L-phenylalanine (10 mM) was added to both the mucosal and serosal fluid. After the incubation period, the incubating fluids and the intracellular contents of the mucosal epithelial cells were analyzed for sodium, potassium, ^{3}H-inulin and L-phenylalanine. The intracellular sodium concentration remained constant under both experimental conditions, whereas the intracellular potassium concentration was lower in the presence of L-phenylalanine. However, the total intracellular sodium content (based on dry tissue weight) increased in the presence of L-phenylalanine while the total intracellular potassium content remained the same under both conditions. The entrance of L-phenylalanine into the intestinal epithelial cells is, therefore, accompanied by a net entrance of sodium and a net entrance of water, which dilutes the cellular potassium. The net entrance of water corresponded osmotically to the net gain in ions and L-phenylalanine. It is concluded that L-phenylalanine entering the intestinal cells is in an osmotically active form.

6709 EFFECT OF DIETARY FAT (SOYBEAN OIL) ON INTESTINAL IRON ABSORPTION IN RATS. *(E.)* Hirooka, M. (Akita Cent. Hosp., Japan), S. Ikeda and N. Kubota. *Tohoku J Exp Med* 95(3):243-252, 1968.

6710 CHARACTERISTICS OF WATER ABSORPTION IN AN ISOLATED LOOP OF SMALL INTESTINE. *(Rus.)* Bulatova, V. M. (Lvov Med. Inst., USSR). *Vop Pitan* 26(6):82-83, 1967.

6711 ABSORPTIVE CAPACITY OF THE DIGESTIVE TRACT AFTER RESECTION OF THE LOWER HALF OF THE SMALL INTESTINE. *(Rus.)* Kriukova, L. V. (Inst. Norm. Path. Physiol., Acad. Med. Sci. USSR, Moscow). *Biull Eksp Biol Med* 65(1):45-47, 1968.

6712 EFFECT OF THE SIMULTANEOUS ABSORPTION OF NUTRIENTS ON THE ABSORPTION OF IRON FROM THE DIGESTIVE TRACT. 1. EFFECT OF PROTEIN. *(Cz.)* Brodan, V. (Inst. Res. Human Nutrit., Prague Czechoslovakia), M. Brodanová, E. Kuhn, V. Kordač, J. Válek and J. Pechar. *Cesk Gastroent Vyz* 21(6):383-392, 1967.

6713 EFFECT OF THE SIMULTANEOUS ABSORPTION OF NUTRIENTS ON THE ABSORPTION OF IRON FROM THE DIGESTIVE TRACT. 2. EFFECT OF FAT. *(Cz.)* Brodan, V. (Inst. Res. Human Nutrit., Prague, Czechoslovakia), M. Brodanová, E. Kuhn, V. Kordač and J. Válek. *Cesk Gastroent Vyz* 21(6):393-400, 1967.

6714 EFFECT OF THE SIMULTANEOUS ABSORPTION OF NUTRIENTS ON THE ABSORPTION OF IRON FROM THE DIGESTIVE TRACT. 3. EFFECT OF FRUCTOSE. *(Cz.)* Brodan, V. (Inst. Res. Human Nutrit., Prague, Czechoslovakia), M. Brodanová, E. Kuhn, V. Kordac and J. Valek. *Cesk Gastroent Vyz* 21(8):521-525, 1967.

6715 THE PROBLEM OF "DELAYED TRANSPORT" IN IRON ABSORPTION CURVES. *(Cz.)* Brodan, V. (Inst. Res. Human Nutrit., Prague, Czechoslovakia), M. Brodanová, E. Kuhn, J. Válek and V. Kordač. *Cesk Gastroent Vyz* 22(1):19-23, 1968.

6716 KINETIC PROBLEMS IN INTESTINAL IRON ABSORPTION: THE KINETICS OF INTESTINAL IRON ABSORPTION IN ANIMALS WITH EXPERIMENTAL HYPERSIDEROSIS. *(It.)* Robustelli della Cuna, G. (Inst. Gen. Med. Clin., U. Pavia, Italy). *Haematologica* 52(8): 651-656, 1967.

6717 ABSORPTION AND UTILIZATION OF GLUCOS BY MEAL-FED AND NIBBLING RATS. *(E.)* Leveille, G. A. (Div. Nutr. Biochem., U. Illinois, Urbana) and K. Chakrabarty. *J Nutr* 96(1):69-75, 1968.

See also: 6723,6743,6881,7492

6718 EFFECT OF SELECTIVE GASTRIC AND TOTAL ABDOMINAL VAGOTOMY ON THE CANINE GALLBLADDER.

(E.) Scheinin, T. M. (Dept. Surg., U. Turku, Finland), M. V. Inberg and E. Lehtinen. *Ann Med Exp Fenn* 45(4):377-380, 1967.

The effects of selective gastric vagotomy and total vagotomy on the gallbladder were compared by measuring gallbladder size and resting and yield pressures 6 days after permanent intubation of the gallbladder in 12 dogs. Gallbladder size was determined planimetrically in a single plane after the drip infusion of 30% Urografin via the cannulation tube. Selective bilateral gastric vagotomy was performed on 8 dogs 1 week after cannulation; in 1 animal, a total vagotomy was performed at this time. Early effects of selective vagotomy (6-12 days later) were studied in 6 dogs and late effects (1½-3 months later) were studied in 2 dogs. Total vagotomy was performed in 6 dogs and the early (6-12 days later) and late effects (1-2 months later) were studied in 6 and 5 dogs, resp. Contraction of the gallbladder was measured after i.v. pancreozymin (2 U/kg) in 2 dogs before vagotomy, in 3 after selective, and in 4 after total vagotomy. Selective vagotomy produced early gallbladder enlargement without any subsequent increase; total vagotomy produced marked early enlargement progressing through the observation period. Resting and yield pressures of the gallbladder were not altered by either procedure, in either the early or late periods of observation. Gallbladder emptying induced by pancreozymin resulted in no difference in the rate of emptying after selective or total vagotomy, but the amount of residual bile was greater after total vagotomy.

6719 THE MOVEMENTS OF THE COLON OF RATS AND GUINEA PIGS.

(E.) Hukuhara, T. (Okayama U. Med. Sch., Japan) and T. Neya. *Jap J Physiol* 18(4):551-562, 1968.

Rats and guinea pigs were utilized to study the pacemaker area (site of origin of peristaltic and antiperistaltic waves) in the colon and the process of transportation of the large intestinal contents in herbivorous animals. The rats and guinea pigs were anesthetized by intraabdominal administration of pentobarbital (50 and 15 mg/kg, resp.), the abdominal cavity was opened, and the jejunum and ileum were removed to expose the large intestine. It was found that the intestinal contents which are inspissated to become scybalum are transported anally through the pacemaker area by antiperistalsis; this may be activated by an intrinsic mucosal reflex causing a rise in excitability in the intestine when the mucosa is stretched by the scybalum. Rhythmic contractions elicited in the pacemaker area, both *in vivo* and *in vitro*, were reduced in strength by administration of hexamethonium and abolished by atropine or tetrodotoxin. Since the pacemaker area had a higher density of Auerbach's plexi and acetylcholine then found in the adjacent areas of the large intestine, it is concluded that cholinergic neurons are more numerous here than in other sites of the colon.

6720 BILE FLOW MECHANISM IN MAN.

(E.) Ono, K. (Hirosaki U. Fac. Med., Japan), N. Watanabe, K. Suzuki, H. Tsuchida, Y. Sugiyama and M. Abo. *Arch Surg* 95(6):869-874, 1968.

By simultaneously recording the cinecholangiogram (contrast media via T-tube) and the electromyogram 5-15 days postoperatively, the mechanism of biliary flow was determined in 16 patients (28-76 yr old) with cholelithiasis who had electrodes implanted into the duodenum and the sphincter of Oddi, plus insertion of a T-tube into the common bile duct, after cholecystectomy. Bile flow was interrupted at the terminal portion of the common bile duct when electrical activity was noted at the sphincter of Oddi. Bile flow into the duodenum occurred only in the absence of electrical activity at the sphincter or during the phase of relaxation of the terminal choledochus. The electrical activity recorded from the muscle layer of the duodenal wall showed no correlation with either the flow of bile into the duodenum or the suppression of flow.

6721 ACTION OF SOME ANTIBIOTICS ON THE EXTRA-HEPATIC BILIARY TRACT. III. TETRACYCLINES.

(E.) Benzi, G. (Dept. Pharmacol., U. Pavia, Italy), A. Crema, F. Berté and G. M. Frigo. *Arch Int Pharmacodyn* 170(2):379-387, 1967.

The effect of tetracycline, chlortetracycline and oxytetracycline on the gallbladder and common bile duct was investigated *in vitro* and *in situ* in various animal species. All three tetracyclines (250-2000 μg/ml) caused decreased biliary flow through the calf's isolated common bile duct in about 50% of the animals; increased flow occurred in 20-30%, and in some experiments a biphasic response was observed. Motility of the isolated cat and guinea-pig gallbladder was depressed, with or without a reduction in tone, depending on the dose of drug used (7.9-500 μg/ml). There were no differences among the three tetracyclines. In the cat, dog, guinea-pig and monkey (*C. hamadryas*), all 3 antibiotics (125-2000 μg/ml) perfused through the common bile duct *in situ* produced a decrease in biliary flow, with little or no increase in duodenal tone and motility. The drugs (125-1000 μg/ml) also produced hypertonus of the guinea-pig common bile duct. A quantitative study of the action of the tetracyclines showed an inverse dose-lag time correlation and a direct, but decreasing, dose-flow correlation. Studies with cross-bile-perfusion preparations in the cat also demonstrated that the choledochus was affected by all the tetracyclines secreted from the tetracycline-treated donors (2000 μg/ml). No changes in response occurred after pretreatment with dibenamine (2-6 mg/kg i.v.), atropine (1-3 mg/kg s.c.), cyproheptadine (50-250 μg/kg i.v.) or hexamethonium (50-300 μg/kg i.v.), suggesting that the tetracyclines act directly on the bile-duct smooth muscle.

6722 THE ELECTRICAL ACTIVITY OF GUINEA-PIG SMALL INTESTINE WITH SPECIAL REFERENCE TO THE SLOW WAVE.

(E.) Hukuhara, T. (Okayama U. Med. Sch., Japan) and H. Fukuda. *Jap J Physiol* 18(1):71-86, 1968.

The electrical activity of isolated guinea-pig jejunal loops was studied by a combination of the ultramicroelectrode technique and Trendelenburg's technique. As the intraluminal pressure was increased, the depolarization of the muscle cell membrane gradually increased, first showing minute oscillations and then becoming summated to develop slow depolarization waves with a more or less long period. When the peak of the depolarizing wave reached a certain critical level (12 mV), a spike was initiated followed successively by an initial after-hyperpolarization, a late after-depolarization, and a delayed after-hyperpolarization which soon returned to the phase of increasing depolarization and finally culminated in the induction of another spike. When the intraluminal pressure was lowered, the spike was abolished and the slow waves declined in amplitude, finally become minute oscillations. This indicates that the spike is responsible for the development of the slow waves. The late after-depolarization could be accentuated by applying acetylcholine (5×10^{-7}-5×10^{-6} g/ml) or by raising the pressure to initiate repetitive spike potentials, and was reduced by applying nicotine (10^{-4} g/ml) or atropine (10^{-4}-10^{-5} g/ml) or by lowering the pressure. Under isometric conditions, the slow waves could be completely abolished with a continued production of spikes. It is felt that the depolarizing waves, ranging from minute oscillations to slow waves, are essentially the same in nature, and are elicited by acetylcholine released from the intramural nervous elements.

6723 POLYPEPTIDES (BRADYKININ, KALLIDIN, ELEDOISIN) AND THE MOVEMENT OF INTESTINAL VILLI.

(Ger.) Ludány, G. (Med. U., Budapest, Hungary), M. Ihász and J. Karika. *Acta Physiol Acad Sci Hung* 34(1/2):85-93, 1968.

When injected into the mesenteric artery, 1 μg/ml/min of bradykinin completely stopped the rhythmic, autonomic movement of intestinal villi in dogs anesthetized with chloralose. The bodies of the villi became shorter, their muscle tonus increased, and their network of capillaries contracted. This was accompanied by mucosal ischemia and a moderate and temporary increase in blood pressure, followed by an increase in autonomic function. When injected into a side branch of the mesenteric artery which did not supply the mucosa, bradykinin inhibited autonomic movement for a short time, but no increase occurred later in autonomic function. Inhibition of the movement of the villi was shown to be caused by a neurally conditioned reflex action occurring over long distances and was prevented by injection of 0.25% procaine into the side branch of the mesenteric artery or 3 mg atropine i.v. and 1 μg/ml/min bradykinin into the mesenteric artery. Similar, but less pronounced effects, were produced by kallidin. Eledoisin in a dose of 2 μg/ml/min inhibited movement of the villi but did not produce a subsequent increase in function.

6724 EFFECT OF ISCHEMIA OF LOWER ESOPHAGUS AND ESOPHAGOGASTRIC JUNCTION ON CANINE ESOPHAGEAL MOTOR FUNCTION.

(E.) Earlam, R. J. (Mayo Clin. Found., Rochester, Minn.), J. F. Schlegel and F. H. Ellis Jr. *J Thorac Cardiovasc Surg* 54(6): 822-831, 1967.

In order to damage or destroy the ganglion cells in Auerbach's plexus, the lower end of the esophagus and the upper part of the stomach of 15 dogs were made ischemic for 4 hr. As a result, the esophagus became dilated in most animals but eventually reverted to normal size. Definite motility disturbances occurred in the early postoperative period, consisting of an absence of peristalsis in the body of the esophagus and failure of the gastroesophageal sphincter to relax; however, recovery from these abnormalities occurred in most animals. After sacrifice, histological examination of the esophagus revealed a decrease in number of and damage to the ganglion cells in Auerbach's plexus. It is clear that the lower esophagus and the esophagogastric junction can function normally in the absence of normal numbers of ganglion cells in Auerbach's plexus, although the precise mechanisms responsible for the abnormalities noted in the early postoperative period are not clear.

6725 EFFECTS OF ANION SUBSTITUTIONS ON POTASSIUM-INDUCED PHASIC AND TONIC CONTRACTION IN TAENIA COLI.

(E.) Urakawa, N. (Dept. Vet. Pharmacol., Fac. Agric., U. Tokyo, Japan), H. Karaki and M. Ikeda. *Jap J Pharmacol* 17(2):258-266, 1967.

The effects of anion substitution on the phasic and tonic responses induced by high K concentrations (42.7 mM KCl in Tyrode's solution) in the isolated guinea pig taenia coli were studied by using Br^-, NO_3^-, I^-, or SCN^- to replace 50% or 100% of the Cl^- of NaCl in the medium. Anion substitution caused an increase in tension of the 1st peak of the K-induced contraction and a potentiation of the phasic contraction (except for SCN^-), as well as a decrease in the tension of the tonic response. The relative effectiveness of the anions in increasing the maximum tension of the phasic contraction was $Cl^- < Br^- < NO_3^- \leq I^-$ for both 50% and 100% replacement. Complete replacement of Cl^- by SCN^- caused prolongation of the duration of contraction, while shortening of duration was cause by the other anions. Calcium contractions (having no phasic response) were also induced by the application of 2.5 mM CaCl to the depolarized smooth muscle kept in the high-K, Ca-free medium at 37C. Complete replacement of Cl^- by NO_3^- decreased the tension of the Ca-contraction to a degree similar to the decrease in tonic tension of the K-induced contraction preceded by a phasic contraction. It appears that the decrease in tension of K-induced tonic contraction by anion substitution may be independent of the potentiation of tension in the 1st peak preceding the tonic response.

6726 CLINICAL AND EXPERIMENTAL STUDIES ON THE MOTILITY OF THE VAGOTOMIZED STOMACH.

(Ger.) Welsch, K. H. (Surg. Polyclin., U. Munich, Germany), W. Hart, W. Brückner and I. Klempa. *Z Gastroent* 6(2):104-113, 1968.

Gastric motility was measured in 58 subjects (16 patients with no stomach diseases, 19 with peptic ulcers, and 23 after gastric surgery and vagotomy). Measurements were made after a 12-hr fast, instillation of 200 ml of physiological saline, and administration of insulin (10 IU i.v.), prostigmine (0.5 mg i.v.), or a combination of insulin and atropine (0.5 mg i.v.). Both insulin and prostigmine stimulated gastric motility to a significantly greater extent in 16 patients with duodenal ulcers than in the controls. Atropine inhibited insulin-induced motility, but to a significantly smaller extent than in the controls. In contrast, insulin- and prostigmine-induced motility was of the same order of magnitude in 3 patients with stomach ulcers and in the controls. Insulin and prostigmine had significantly less effect on gastric motility in 16 patients after vagotomy and pyloroplasty than in the controls. The effects of insulin and prostigmine on gastric motility were about the same before and after gastric resection, vagotomy, and pyloroplasty in 2 cases. Insulin produced no increase in motility in 4 patients who had undergone vagotomy, antral resection, and gastroduodenostomy. Since it acts on postganglionic nerve pathways, prostigmine did. Atropine inhibited insulin-induced motility in all 6 groups of patient These findings agree with the hypothesis that vagal innervation in the antrum prevents liberation of gastrin when the stomach is not digesting food and prevents the retention of food in the antrum.

6727 **CHANGES IN THE HISTAMINE REACTIVITY OF THE SMALL INTESTINE IN HEALTHY AND LEAD-POISONED ANIMALS UNDER THE INFLUENCE OF SOLUTIONS OF LEAD ACETATE.** *(Rus.)* Mambeieva, A. A. (Kazakh Inst. Reg. Path., Acad. Med. Sci. USSR, Alma-Ata) and A. S. Akhmedova. *Biull Eksp Biol Med* 64(8):34-37, 1967.

6728 **EFFECT OF OXYGEN ON GASTRIC FUNCTION IN EXPERIMENTAL ANIMALS.** *(Rus.)* Kosiakova, M. P. (Ivano-Frankovsk Med. Inst., USSR) *Biull Eksp Biol Med* 64(12):19-21, 1967.

6729 **THE MECHANISM OF GASTRIC EMPTYING IN THE HOOK-SHAPED STOMACH.** *(It.)* Celli, L. (Maggiore Hosp., Milan, Italy), E. Gerletti and É. M. Pratelli. *Arch Ital Mal Appar Dig* 34(1):36-43, 1967.

6730 **STUDIES OF TOTAL AND PARTIAL GASTRO-INTESTINAL TRANSIT TIMES.** *(It.)* Cassano, C. C. (Inst. Med. Path., U. Rome, Italy), M. Amoruso, P. Arullani and G. Badalamenti. *Arch Ital Mal Appar Dig* 34(3): 290-293, 1967.

6731 **ATTEMPTS AT QUANTIFICATION IN THE STUDY OF THE MOTILITY OF THE MESENTERIC SMALL INTESTINE: VERIFICATION OF THE "MOTILITY INDEX" AND PROPOSAL OF AN ENTEROTENSIOGRAPHIC INDEX.** *(It.)* Gaglio, M. (Med. Clin., Catania, Italy) and O. Cascone. *Arch Ital Mal Appar Dig* 34(3):294-297, 1967.

6732 **SUMMATION PHENOMENA IN THE REFLEX SWALLOWING CENTER.** *(Rus.)* Kvasov, D. G. (Pediat. Med. Inst., Leningrad, USSR) and A. B. Filimonova. *Fiziol Zh SSSR Sechenov* 54(9):1025-1033, 1968.

6733 **THE EFFECTS OF RESPIRATION ON THE AUTONOMICALLY CONTROLLED MOVEMENTS OF THE STOMACH AND DUODENUM.** *(Ger.)* Schmidt, H. (Inst. Med. Radiol., U. Tubingen, Germany). *Med Welt* 18(47): 2839-2844, 1967.

See also: 6687,6967

6734 **CONCURRENT COLLECTION OF BILE AND PANCREATIC JUICE THROUGH THOMAS CANNULA.** *(E.)* Wyman, J. B. (Mayo Grad. Sch. Med., Rochester, Minn.), K. G. Wakim, L. G. Bartholomew and J. C. Cain. *J Appl Physiol* 25(5):638-640, 1968.

Using aseptic technique, a Thomas cannula was inserted into the duodena of 32 anesthetized female dogs (7.8-16 kg) which were also subjected to cholecystectomy. When the distance between the papilla of Vater and the major pancreatic papilla approximated the diameter of the cannula (1.9 cm), bile and pancreatic juice could be collected concurrently, directing the cannula cephalad for bile collection and caudad for pancreatic juice collection. In larger dogs in which the papillae were further apart (more than 2.8 cm), a permanent tube was placed in the common bile duct. Continuity of the common bile duct was maintained and the nonintubated portion was ligated to prevent bile leakage. The tubes were cleaned every 1-2 weeks to ensure against bile salt deposition. In 15-8-hr studies, bile flow was 31-107 ml; flow rate varied with different dogs and, in separate studies, ranged from 4.1 to 13.4 ml/hr. Intraduodenal HC1 was used to stimulate pancreatic flow through the release of secretin. Pancreatic flow rates ranged from 11 to 225 ml/8 hr in 20 studies.

6735 **FATTY ACID SYNTHESIS BY JEJUNAL SLICES FROM RATS FED MEDIUM AND LONG CHAIN TRIGLYCERIDES.** *(E.)* Reeve, J. (U. Colorado Med. Ctr., Denver) and J. J. Franks. *Amer J Physiol* 214(6):1425-1428, 1968.

The effects of prefeeding long- and medium-chain triglycerides on *in vitro* intestinal fatty acid synthesis were investigated in male albino rats. Jejunal slices from rats prefed lipid, olive oil, palm oil, or an octanoate-decanoate triglyceride preparation were incubated with acetate-1-^{14}C. No stimulation of acetate-1-^{14}C incorporation into fatty acids was observed in animals fed long-chain fat in comparison to animals fed medium-chain triglycerides. Prefeeding fat, especially medium-chain triglycerides, altered the distribution of incorporated radioactivity with most of the radioactivity being found in triglycerides rather than in the phospholipid fraction of the jejunal slices, in animals fed the medium-chain triglycerides, a marked reduction in stearic acid radioactivity was also noted. However, increased amounts of radioactivity were found in fatty acids of 10, 12, 14, and especially 16 carbon atoms, suggesting successive elongation of the prefed octanoic and decanoic acids. Prefeeding long-chain triglycerides resulted in a pattern of labeled fatty acids which differed only slightly from the pattern seen in animals fed no lipid. These results suggest that absorption of long-chain fatty acids does not stimulate endogenous fatty acid synthesis in the jejunum.

6736 **THE STIMULATION OF RNA SYNTHESIS *IN VIVO* AND *IN VITRO* BY RETINOL (VITAMIN A) IN THE INTESTINE OF VITAMIN A DEFICIENT RATS.** *(E.)* Zachman, R. D. (U. Wisconsin Sch. Med., Madison). *Life Sci* 6(20):2207-2213, 1967.

Three- to 4-week-old white female Holtzman rats maintained on a vitamin A deficient diet, which showed a plateau or slight weight loss at 6-8 weeks, were used for all experiments. For *in vivo* studies, paired deficient rats received i.p. injections of 1 ml of normal saline containing 100 μC ^{3}H-uridine and 20 μg retinol in 0.006 ml Tween-80; control rats received the same solution but without retinol. Animals were sacrificed at various time intervals, and the ratio of the specific activities of RNA (retinol treated/control) in the intestine was determined. For *in vitro* studies the colon was divided into sections and placed in solutions containing 20 μC ^{3}H-uridine, 10 μg retinol and 0.003 ml Tween-80; other sections were placed in identical solutions without retinol. The ratio of RNA specific activities 2 hr after ^{3}H-uridine injection in the retinol treated/control rats was 1.3, 1.3, 1.8, 2.0, and 1.7 for liver, kidney, duodenum, ileum, and colon, resp. The ratios for liver and kidney were considered to be not significantly different from 1.0. Retinol also stimulated RNA synthesis in the deficient colon slices, best noted after 10 min of incubation with retinol. The average ratio in 13 experiments was 1.8. Similar incubations for 90-120 min gave an average ratio of only 1.02, thought to be due to degenerative changes in the tissues with time. It is significant that both *in vivo* and *in vitro* stimulatory effects were seen at periods when the time for retinol action was very small, and occurred only in epithelial lined tissues. Retinol did not alter the total uridine pool size, and therefore appears to affect RNA synthesis more directly.

6737 **AMINE FORMATION IN POLYPEPTIDE-PRODUCING ENDOCRINE CELLS OF THE DIGESTIVE TRACT.** *(E.)* Aures, D. (VA Hosp., Sepulveda, Calif.) and R. Hakanson. *Europ J Pharmacol* 3(4):316-321, 1968.

See also: 7085

6738 **THE TROPHIC EFFECT OF CATECHOLAMINES ON THE SECRETORY FUNCTION OF EXHAUSTED STOMACH GLANDS.** *(Rus.)* Anichkov, S. V. (Inst. Exp. Med., Acad. Med. Sci. USSR, Leningrad) and L. L. Grechishkin. *Biull Eksp Biol Med* 64(10):37-39, 1967.

In 8 experiments, 3 dogs were infused i.v. for 7 hr with 1 μg/kg/min histamine followed, 80 min later, by 15 μg/kg/min acetylcholine. The volume and pepsin content of the gastric secretion increased for 3-6 hr and then decreased greatly. The pH of the gastric juice remained unchanged at 1.4-1.5, but total HCl decreased due to the decrease in volume. In another series of 16 experiments, dogs treated as described above received i.v. infusions of 3 μg/kg epinephrine, 3 μg/kg norepinephrine, or 1.5 μg/kg isoproterenol for 1 hr. The volume and pepsin content of the secretion increased, beginning 40-60 min after catecholamine infusion. When acetylcholine and histamine were injected into 2 dogs which had been given 0.05 mg/kg reserpine i.m. for 10 days, gastric secretion fell off more rapidly (within 60-80 min) than in animals which had not been treated with reserpine. The increases in pepsin concentration produced in reserpine-treated dogs were significantly smaller than those produced in control animals, but the volumes of gastric secretion were almost the same. It is suggested that more rapid depletion of the catecholamine reserves, which occurs in reserpine-treated animals, produces a trophic disturbance in the gastric mucosa and more rapid exhaustion of the stomach glands.

6739 **GASTRIC SECRETION OF PEPSIN IN EARLY CHILDHOOD.** *(E.)* Rodbro, P. (Glostrup Hosp., Copenhagen, Denmark), P. A. Krasilnikoff and V. Bitsch. *Scand J Gastroent* 2(4):257-260, 1967.

Gastric secretion of pepsin was studied in 18 children (9-30 months of age) without gastrointestinal disorders, for 30 min before and for four 15-min periods after s.c. injection of 40 μg histamine acid phosphate/kg. The children weighed 8.0-13.9 kg, were 73-93 cm in height, and had a body surface area of 0.39-0.57 m^2. Pepsin secretion was calculated in terms of basal output (1.1-14.8 mg pepsin/hr), stimulated output (2.2-26.0 mg pepsin/hr), and total output (9.6-39.4 mg pepsin/hr). The stimulated pepsin output thus showed a slight increase over the basal pepsin output. While the basal pepsin output correlated significantly with age and body surface area, the stimulated output could not be correlated significantly with either age, body weight, body surface area, the basal pepsin output, or the stimulated gastric acid output.

6740 **INDIVIDUAL VARIATIONS IN SERUM AND URINARY PEPSINOGEN AND RELATIONS TO THE HISTOLOGY OF THE GASTRIC MUCOSA, AS DETERMINED BY A MODIFIED TECHNIQUE.** *(Ger.)* Kehrel, H. (2nd Med. Clin., U. Hamburg, Germany) and H. Schmidt. *Deutsch Z Verdau Stoffwechselkr* 28(1):7-12, 1968.

Serum and urinary pepsinogen levels were determined at 3-4 day intervals for 4 weeks in 10 normal controls (5 males and 5 females) and, in 8 of these same controls, every 3 days for 12 months. Individual scattering was small, particularly for serum values. Analysis of the results with variance statistics showed that serum pepsinogen values were very stable, both over 4- and 12-month periods, while urinary activities varied much more. Pepsinogen determinations were also run on serum and urine from 91 patients with stomach diseases; none had duodenal ulcers, pernicious anemia, or any nongastric diseases. In 39 patients with normal gastric mucosa the range for serum pepsinogen levels was 67-129 μg pepsin equivalents/liter (mean of 98 μg). Serum pepsinogen values averaged about 30% higher among 41 patients with chronic superficial gastritis. Scattering was also greater in this group. Urinary pepsinogen levels were also elevated, but not significantly because of the great scattering. Mean serum pepsinogen values were about the same in 11 patients with atrophic gastritis and in the controls, but scattering was greater among the former group and their urinary pepsinogen levels were significantly elevated. Although serum pepsinogen values are of no diagnostic value in atrophic gastritis, low urinary pepsinogen values are evidence for atrophic changes in the stomach and, in the absence of conclusive histological findings, can help to distinguish between chronic superficial gastritis and atrophic gastritis.

6741 **NEURAL AND HUMORAL PATHWAYS FOR THE TRANSMISSION OF STIMULI FROM THE HYPOTHALAMUS TO THE SECRETORY ACTIVITY OF THE GASTRIC GLANDS.** *(Rus.)* Kosenko, A. F. (Inst. Physiol., T. G. Shevchenko U., Kiev, USSR). *Biull Eksp Biol Med* 65(1):17-20, 1968.

An electric current (50 cycles/sec, 0.5 milliamperes) from a sound generator was used to produce excitation in various regions of the hypothalamus in dogs, and the effects of bilateral vagotomy, splanchnicotomy, and adrenalectomy on the hypothalamic control of gastric secretion were determined. Transmission of positive stimuli from the anterior and median parts of the hypothalamus to the gastric glands involved the vagus nerves, while transmission of negative stimuli from the posterior regions of the hypothalamus did not. The inhibitory effects on gastric secretion from the posterior part of the hypothalamus were transmitted through the splanchnic nerves. When the posterior part of the hypothalamus is excited, gastric secretion is stimulated primarily by a humoral pathway. However, neural pathways play the major role in transmitting impulses from the hypothalamus to the stomach.

6742 **THE BEHAVIOR OF GASTRIC SECRETION AFTER ADMINISTRATION OF THYROID EXTRACT IN NORMAL SUBJECTS.** *(It.)* Ferlito, S. (Inst. Spec. Med. Path., U. Catania, Italy) and M. Calafato. *Riv Gastroent* 20(1):35-45, 1968.

Gastric acid and pepsin secretion were studied in 15 normal men, aged 23-61 yr, under basal conditions and after administration of 40 mg of total thyroid extract i.m. In contrast to other findings reported in the literature, thyroid extract never reduced stomach secretion. While it produced little or no change in 2 subjects with hypochlorhydria, it significantly increased the total volume of gastric juice secreted, pepsin activity, and maximum and total HCl in 10 patients with normal secretory levels. These values reached their highest levels 30 min after administration of the thyroid extract. In some cases, maxima were also reached after 90 and 120 min, giving a bicuspid curve. Values for bound acidity varied, but the mean value tended to increase after administration of thyroid extract. This is probably due to the relatively small amounts of water secreted and the simultaneous increase in mucoprotein production. Similar changes occurred in 3 subjects with hyperchlorhydria, but values obtained after thyroid stimulation were absolutely higher than values obtained in controls or subjects with normochlorhydria. The strongest responses to thyroid stimulation were found in 4 subjects under 30 yr of age. In 4 cases, 1 mg of histamine was given i.m., followed 15 min later by thyroid extract. Initially, histamine inhibited the effects of thyroid stimulation, but a 50% increase occurred in the basal values after the second hr.

6743 NONSECRETORY TRANSPORT OF CHLORIDE THROUGH THE GASTRIC MUCOSA: OBSERVATIONS MADE ON MAN. *(Fr.)*

Zalaru, M. S. (Hosp. No. 1, Ploiesti, Romania). *Acta Gastroent Belg* 30(11):721-729, 1967.

Chloride determinations were made on blood, gastric juice, and mucosa from the gastric fundus and antrum of 37 patients with complicated peptic ulcers. No significant differences were found between the chloride contents of the fundus and antrum in normochlorhydric and hyperchlorhydric patients. These results indicate that chloride transport occurs by electrostatic attraction to hydrogen ions being absorbed back into the plasma and is a purely passive phenomenon.

6744 THE EFFECT OF PHENYLBUTAZONE ON SECRETION OF ^{35}S-LABELED SULFATE BY MUCOSAL CELLS OF THE STOMACH. 1. STUDY OF DIFFERENT FRACTIONS. *(Fr.)*

Lambert, R. (Edouard Herriot Hosp., Lyon, France), C. André, J. P. Léonard and F. Martin. *Acta Gastroent Belg* 30(12):787-806, 1967.

Total, dialyzable, nondialyzable, and cetyl pyridinum chloride-precipitable fractions were determined in gastric juice from male Wistar rats with indwelling cannulas implanted in their rumens. Insoluble mucus, which contains large amounts of glycoproteins, was not analyzed with the method employed. Rats had been given 100 mg/kg phenylbutazone i.m. on 3 consecutive days and 40 μC of $Na_2{}^{35}SO_4$ i.p. at the beginning of the experiment. Phenylbutazone consistently reduced the volume of gastric juice secreted and increased its pH between 7 and 25 hr after injection of the isotope. Phenylbutazone also reduced total secretion of radioactive sulfate by 20-30%, but did not appear to change the ratio of the dialyzable to the nondialyzable fraction. In samples collected during the first 11 hr the fraction of radiosulfate precipitated by cetyl pyridinum chloride at pH 1.5 was 30-54% that of the controls. This reduction was greater than that in the total radioactivity, and the ratio of this fraction to total radiosulfate was lower in treated animals than in controls. This inhibition was also seen in samples of gastric juice collected 7-25 hr after injection of radiosulfate. These results suggest that phenylbutazone not only inhibits incorporation of sulfate into glycoproteins but also prevents it from crossing the membrane of the mucosal cells.

6745 THE EFFECT OF PHENYLBUTAZONE ON SECRETION OF ^{35}S-LABELED SULFATE BY MUCOSAL CELLS OF THE STOMACH. 2. ELECTROPHORETIC STUDY. *(Fr.)*

Lambert, R. (Eduoard Herriot Hosp., Lyon, France), J. P. Léonard, C. André and F. Martin. *Acta Gastroent Belg* 30(12):807-818, 1967.

Male Wistar rats with indwelling cannulas implanted in their rumens were given 100 mg/kg phenylbutazone i.m. on 3 consecutive days and 40 μC of $Na_2{}^{35}SO_4$ i.p. at the beginning of the experiment. Gastric juice, collected at intervals for 16-51 hr after injection of the isotope, was subjected to autoproteolysis for 48 hr at 37C and adjusted to pH 8 to dissolve insolubl mucus which did not undergo proteolysis. In untreated rats insoluble mucus constituted only 3.1% of total radioactivity 16 19 hr after isotope injection but accounted for 23% of nondialyzable radiosulfate. Phenylbutazone greatly reduced total, dialyzable, and nondialyzable radiosulfates in gastric juice collected 16-19 hr after injection of the isotope. In untreated controls paper electrophoresis of the nondialyzable fraction produced 2 bands. The slower one stained more intensely wit Alcian blue and contained more of the total radioactivity. The faster band had an electrophoretic mobility close to that of chondroitin sulfate. In gastric juice collected from treated animals 16-19 hr after injection of the isotope the radioactivity in the slower band was only 9.1% of that of the controls and tha in the faster band was 28.8%. This more marked depression o the faster band persisted. Radiosulfate secretion was much lo in rats which had been repeatedly treated with phenylbutazone

6746 STUDIES ON THE ROLE OF THE ADRENAL CORTEX IN THE INHIBITION OF GASTRIC SECRETION PRODUCED BY OXYTOCIN IN RATS. *(Fr.)*

Groza, P. (D. Danielopolu Inst. Norm. Path. Physiol., Buchares Romania), V. Buzoianu and S. Ionescu. *Rev Roum Physiol* 5(2):111-114, 1968.

Oxytocin (0.01 U) inhibited histamine-stimulated gastric secretion in rats by 60% and pepsin secretion by 65%. The same dose of oxytocin significantly increased the volume and pepsi output in animals which had been adrenalectomized 2 weeks before. Considerable fluctuations occurred in acid secretion, it generally paralleled quantitative changes in the volume of gastric secretion. These findings indicate that corticosteroids play an important role in inhibition of gastric secretion.

6747 CHANGES IN THE ELECTROLYTE COMPOSITION OF THE GASTRIC JUICE UNDER BASA CONDITIONS AND AFTER DRUG STIMULATION. *(It.)*

Gritti, F. M. (Inst. Gen. Med. Clin., U. Bologna, Italy), T. Montini, G. C. Descovich and A. Lancellotti. *Arch Ital Mal Appar Dig* 34(1):10-35, 1967.

Electrolyte concentrations were determined in the gastric juic and plasma from 28 normal subjects (18 women and 10 men, aged 22-43 yr) before and after introduction of an Einhorn tu or administration of 1 mg histamine s.c., 1.5 mg aldosterone i.v., and a combination of histamine and aldosterone. Introduction of an Einhorn tube increased K values slightly in the gastric juice. Histamine increased K concentrations, total acid and free HCl in both gastric juice and plasma. Increases in K concentration occurred simultaneously in plasma and gastric juice and are believed to be due to the release of K from all cells as well as those in the gastric mucosa. Aldosterone reduced the Na:K ratio in both gastric juice and plasma. Increa in total acidity and free HCl were inversely related to Na concentration in the gastric juice after either histamine or aldosterone. A partial additive effect was obtained by administering a combination of histamine and aldosterone. Plasma K increased slightly. The great variation in the electrolyte composition of gastric juice, both from one subject to another and in the same individual, is regarded as a manifestation of physiological adaptation of the gastric mucosa to various functional situations. Changes in plasma electrolytes are regarded as insignificant and due more to the effect of the drug on the body than to changes in electrolyte secretion by the gastric mucosa.

6748 **GASTRIC FUNCTION TESTS CORRELATED TO ABH BLOOD GROUP SUBSTANCES IN GASTRIC JUICE.** *(E.)* Fischermann, K. (Finsen Inst., Copenhagen, Denmark), J. Andersen, P. H. Petersen and P. Rodbro. *Scand J Clin Lab Invest* 20(3):202-206, 1967.

In a series of 28 patients (21 males and 7 females, 26-85 yr old) with various gastrointestinal disorders, gastric acid and intrinsic factor secretion following augmented histamine stimulation and basal uropepsinogen excretion were found to be fairly closely correlated. The secretion of intrinsic factor was above the lowest normal value, ranging from 6020-28190 U/hr, in 12 patients with a maximal acid secretion of 26-106 mEq/liter as well as in 1 patient with hypochlorhydria and 1 patient with achlorhydria; it varied between 76 and 3585 U/hr in 9 with achlorhydria, 2 with hypochlorhydria and 3 with maximal acid responses of 17-36 mEq/liter. The excretion of uropepsinogen was also higher in patients with free acid in the gastric juice (17-153 mU/hr) than in 8 achlorhydric patients (0-48 mU/hr). Only 2 of the 9 patients with gastric cancer, but 4 of 5 with cardiac cancer, had free acid in the gastric secretion, and 3 of the latter 5 had normal acid secretion. Four patients with cardiac cancer and free acid in the gastric juice also showed a normal intrinsic factor secretion, whereas the latter was often low, sometimes reaching the pernicious anemia range, in patients with gastric cancer. Using an immunofluorescence technique, parietal cell antibodies were demonstrated in one patient with duodenal and one with gastric cancer; both had achlorhydria and a very low intrinsic factor secretion. A relatively high incidence of nonsecretors of ABH blood group substance (studied in saliva and gastric juice after histamine stimulation) was found in patients with free acid in the gastric juice (8 of 15 cases), while only 2 of the 13 patients with hypo- or achlorhydria were nonsecretors. The results of ABH secretor studies in saliva and gastric juice were identical. These observations suggest a possible action of the secretor gene on the functional state of cells in the stomach wall other than those which produce ABH blood group substances.

6749 **STUDIES ON THE MECHANISM OF GASTRIN STIMULATION.** *(Rum.)* Groza, P. (D. Danielopolu Inst. Norm. Path. Physiol., Acad. Soc. Rep. Romania, Bucharest), M. Corneanu, S. Ionescu and V. Buzoianu. *Stud Cercet Fiziol* 13(3):217-222, 1968.

The mutual interaction of histamine and gastrin was studied in dogs in order to elucidate the mechanisms of feedback control of gastric secretion. Three dogs with Pavlov pouches were given increasing doses of gastrin extracted from 2 different sources: a) the antrums of 10 fasting dogs, and b) the antrums of 10 dogs given 200 μg of histamine 1 hr before sacrifice. Averaging the results in all 3 dogs, the secretory response to the gastrin extracted from 5 g of antral mucosa was 11% higher (12.5 ± 0.39 ml gastric juice compared to 10.7 ± 0.73 ml) with the gastrin obtained from dogs given histamine. The extracts from 10-20 g of antral mucosa produced a proportionately greater secretory response, followed by a plateau at approximately 21.8 g. Gastric acid secretion into the Pavlov pouches was relatively constant, but the pepsin concentration varied inversely with the volume. The results indicate that a single dose of histamine in dogs increases the gastrin content of the antral mucosa. This positive feedback mechanism may play an important role in the maintenance of gastric secretion.

6750 **TOTAL PROTEIN AND TOTAL NITROGEN IN GASTRIN-STIMULATED GASTRIC SECRETION OF MAN.** *(E.)* Semb, L. S. (Ullevål Hosp., Oslo, Norway), J. Myren and O. P. Foss. *Scand J Gastroent* 2(3):169-174, 1967.

Total protein and total nitrogen levels were determined in 20-min portions of gastric juice withdrawn by continuous suction for 1 hr before and 1 hr after an injection of Gastrin Leo (2 μg/kg s.c.) in 19 patients (6 women and 13 men, 22-70 yr old) with various gastrointestinal and nongastrointestinal diseases. In 9 patients, the procedure was performed twice. Compared to pre-gastrin values, there was an increased concentration and output of protein and a decreased concentration of total nitrogen following gastrin administration; however, the total nitrogen output was increased. There was also a statistically significant correlation between HC1 concentration and total nitrogen concentration after gastrin, and between the outputs of HCl, protein and total nitrogen before as well as after gastrin. The data indicate that gastrin affects the gastric secretion of nitrogenous substances in the same manner as does histamine.

6751 **PARIETAL CELL SECRETORY FUNCTION IN EARLY CHILDHOOD.** *(E.)* Rodbro, P. (Glostrup Hosp., Copenhagen, Denmark), P. A. Krasilnikoff and P. M. Christiansen. *Scand J Gastroent* 2(3):209-213, 1967.

The secretory patterns and hourly outputs of volume, acid and intrinsic factor after augmented histamine stimulation (0.04 mg histaminic acid phosphate/kg s.c.) were studied in 18 children (9-30 months old) without gastrointestinal disorders. The hourly outputs of acid and intrinsic factor correlated significantly with age (r = 0.74 and 0.55, resp.), body-weight (r = 0.68 and 0.59, resp.), and body surface area (r = 0.71 and 0.61, resp.); however, the stimulated acid and intrinsic factor outputs did not correlate significantly with each other. The secretory patterns corresponded to those found in adults, but the peak concentrations of acid and intrinsic factor were half of those found in adults. These findings, and earlier reports, indicate that the gastric secretion of intrinsic factor is more mature than the secretion of acid in the first years of life, and thus seems to be an earlier function of the parietal cell than acid secretion.

6752 **MODE OF ACTION OF A GASTRIC-SECRETION ANTAGONIST.** *(E.)* Kahlson, G. (Inst. Physiol., U. Lund, Sweden), E. Rosengren and S. E. Svensson. *Brit J Pharmacol* 33(3):493-500, 1968.

The effect of antigastrin (2-phenyl-2-[2-pyridyl]-thioacetamide) on hydrochloric acid and pepsin secretion evoked by the infusion of gastrin II or histamine was investigated in unanesthetized rats fitted with total stomach fistulas or Heidenhain-type pouches. Antigastrin inhibited the gastrin- and histamine-induced secretion of HCl by 80% and the secretion of pepsin to a lesser and more varied extent. Infusion of gastrin strikingly enhanced the rate of histamine formation in the glandular region of the stomach mucosa, and also increased the urinary excretion of free histamine. Antigastrin did not inhibit this gastrin-induced elevation of mucosal histamine-forming capacity. It is felt that antigastrin acts by reducing the sensitivity of the parietal and peptic cells to otherwise effective secretory stimulation, and not by interfering with the action of gastrin.

6753 ETHNIC GROUP AND SERUM PEPSINOGEN. *(E.)* Damon, A. (Dept. Anthropol., Harvard U., Cambridge, Mass.) and B. Bell. *J Chron Dis* 29(10):803-808, 1967.

Using Mirsky's procedure (*J. Lab. Clin. Med.* 40:188, 1952), the serum pepsinogen level, was assayed in 236 Irish-American, 196 Italian-American and 64 Jewish middle-aged men. For inclusion in the study all 4 grandparents had to be of the specified ancestry. Most subjects were born in the United States. No significant differences were found in the mean value of the serum pepsinogen levels or in variability among the groups: Irish = 427 ± 134 (standard deviation) or 400 ± 130; Italian = 446 ± 121 or 442 ± 151; Jewish = 431 ± 132. These findings do not support earlier observations of elevated pepsinogen levels in young men of Irish origin.

6754 COMPARATIVE STUDIES ON THE EFFECT OF ATROPINE AND N-BUTYLSCOPOLAMINE BROMIDE ON THE BASAL AND STIMULATED GASTRIC SECRETION OF RATS (SECRETION STUDIES ON SHAY PREPARATIONS). *(Ger.)* Lick, R. F. (Surg. Polyclin., U. Munich, Germany), W. L. Brückner and W. Hart. *Z Gastroent* 5(5):275-281, 1967.

The volume of gastric secretion and the mEq HC1 and units of pepsin secreted in the gastric juice were determined in Sprague-Dawley rats 6 hr after ligation of the pylorus, both under basal conditions and after administration of 10 mg/kg of histamine chloride s.c., 0.625 mg/kg prostigmine s.c., 4.0 μ/kg of insulin s.c., or 37.5 μ/kg of a synthetic gastrin-like tetrapeptide. Animals were also given either 5 mg/kg atropine s.c. or 200 mg/kg n-butylscopolamine s. c. n-Butylscopolamine inhibited basal HC1 production and the volume, acid, and pepsin secretion induced by histamine, insulin, and tetrapeptide significantly more than did atropine. Both drugs had about the same effect on prostigmine-stimulated secretion. Mechanisms underlying the actions of these drugs are discussed.

6755 A STUDY ON THE RELATIONS BETWEEN SOLUBLE TISSUE PROTEIN IN GASTRIC MUCOSA AND GASTRIC SECRETORY FUNCTIONS IN MAN. *(E.)* Sampei, T. (Jikei U. Sch. Med., Tokyo, Japan). *Jikei Med J* 14(1):5-6, 1968.

6756 THE CHEMICAL PROPERTIES AND PHARMACOLOGICAL ACTIVITY OF GASTRIN. *(It.)* Cestari, A. (Inst. Pharmacol., U. Bologna, Italy). *Arch Ital Mal Appar Dig* 34(1):64-76, 1967.

6757 THE ACID-ALKALINE SECRETION OF THE STOMACH IN PATIENTS WITH ACROMEGALY BEFORE AND AFTER MAXIMAL HISTAMINE STIMULATION. *(It.)* Bruno, G. (Inst. Gen. Med. Clin., U. Turin, Italy), G. M. Molinatti and M. de la Pierre. *Arch Ital Mal Appar Dig* 34(1):91-98, 1967.

6758 EFFECT OF RESERPINE ON GASTRIC FUNCTION. *(Fr.)* Reuse, J. (Inst. Pharmacol., Paris, France). *Concours Med* 90(1):488-494, 1968.

6759 EXCRETION OF CATECHOLAMINES BY THE THE GASTRIC MUCOSA. *(Rus.)* Mirzoian, S. A (Erevan Med. Inst., Armenian SSR, USSR), N. A. Iesaian, I. L. Virabian and Ie. K. Kazarova. *Biull Eksp Biol Med* 65(1): 20-23, 1968.

6760 EFFECT OF PARATHYROID HORMONE ON GASTRIC SECRETION IN DOGS. *(Sp.)* Angelillo-Mackinlay, T. (Massachusetts Gen. Hosp., Boston) and G. L. Nardi. *Rev Argent Cir* 13(4/5/6):117-120, 1967.

6761 INHIBITION OF GASTRIC SECRETION IN THE RAT BY SC-15396. *(E.)* Lee, Y. H. (UCLA Sch Med., Los Angeles, Calif.) and J. H. Thompson. *Europ J Pharmacol* 3(4):366-369, 1968.

6762 ANTISECRETORY ACTIVITY OF SOME NEW COMPOUNDS WHICH ARE DEVOID OF ANTICHOLINERGIC ACTIVITY. *(It.)* Rovati, A. L. (Rotta Res. Lab., Milan, Italy), P. L. Casula and G. Da Re. *Minerva Med* 58(86):3651-3653, 1967.

6763 PHARMACOLOGICAL AND CLINICAL EVALUATION OF A NEW NON-ANTICHOLINERGIC ANTISECRETORY AGENT. *(It.)* Rovati, A. L. (Rotta Res. Lab., Milan, Italy), P. L. Casula, L. Lepori and G. Da Re. *Minerva Med* 58(86):3653-3656, 1967.

6764 EXPERIMENTAL TOXICOLOGY AND PHARMACOLOGY OF A NEW NON-ANTICHOLINERGIC COMPOUND WITH ANTISECRETORY AND GASTROPROTECTIVE ACTIVITY (CR 242, XYLAMIDE, MILID). *(It.)* Rovati, A. L. (Rotta Res. Lab., Milan, Italy), P. L. Casula and G. Da Re. *Minerva Med* 58(86):3656-3670, 1967.

6765 EFFECT OF XYLAMIDE (CR 242) ON GASTRIC SECRETION IN THE DOG SUBJECTED TO HYPERSECRETORY STIMULI. *(It.)* Danhof, I. E. (U. Texas Southwest Med. Sch., Dallas). *Minerva Med* 58(86):3670-3677, 1967.

6766 GASTROPROTECTIVE ACTIVITY OF A NEW COMPOUND (XYLAMIDE, MILID) IN THE GASTRIC DISORDERS INDUCED BY ADMINISTRATION OF CORTICOSTEROIDS. *(It.)* Gobbi, F. (Inst. Spec. Med. Path., U. Modena, Italy). *Minerva Med* 58(86):3685-3688, 1967.

6767 **STRUCTURE-FUNCTION RELATIONSHIPS IN GASTRIN-LIKE PEPTIDES.** *(E.)* Morley, J. S. (Imperial Chem. Ind. Ltd., Alderly Park, England). *Proc Roy Soc [Biol]* 170(Series B):97-111, 1968.

6768 **CHEMICAL STUDIES OF SOME MAMMALIAN GASTRINS.** *(E.)* Kenner, G. W. (Robert Robinson Lab., U. Liverpool, England) and R. C. Sheppard. *Proc Roy Soc [Biol]* 170(Series B):89-96, 1968.

6769 **RECENT ADVANCES IN THE PHYSIOLOGY OF GASTRIN.** *(E.)* Gregory, R. A. (Physiol. Lab., U. Liverpool, England). *Proc Roy Soc [Biol]* 81(Series B):81-88, 1968.

See also: 6667,6728,6796,6858,6902,6922,6924,6934,7019,7127,7152,7275,7469

Pancreas

6770 **EFFECTS OF GASTRIC FREEZING ON PANCREATIC AND BILIARY SECRETION.** *(Kor.)* Choi, K. S. (Yonsei U. Coll. Med., Seoul, Korea). *Korean J Intern Med* 11(2):99-107, 1968.

The effects of slow gastric cooling to 15C, 5C, and -10C and of rapid freezing to -10C on secretin- and pancreozymin-stimulated pancreatic secretion and the levels of sodium, potassium, chloride, and bicarbonate in the bile were studied in 16 dogs; pancreatic juice and serum amylase, lipase and trypsin were also measured. The rectal and gastric serosal temperatures fell to -2 to -4C during freezing. Slow freezing to -10C reduced the pancreatic secretory flow 51.4%, while rapid freezing reduced it 18.4%. Pancreatic sodium and potassium secretion was unaffected by freezing, while chloride was slightly reduced and bicarbonate was slightly elevated. Similar changes were seen in bile electrolytes. Slow freezing to -10C reduced the amylase activity in the pancreatic juice by 6.4%, the total amylase output being reduced 54.4%. Rapid freezing also reduced amylase activity 25.2%, and reduced total amylase output 53.8%. Lipase activity was reduced 26.9-30.7% by freezing and total lipase output was reduced 42.4-62.8%. Trypsin activity decreased by 22.0-27.1% and total output was reduced by 42-62.6%. Serum amylase and lipase activities were slightly reduced by slow freezing, but increased 30.4% and 20%, resp., during rapid freezing. Hepatic bile flow decreased 53.8% and 43.4% in slow and rapid freezing, resp.

6771 **THE INFLUENCE OF HYPOTHERMIA ON PANCREATIC SECRETION AND BLOOD FLOW.** *(E.)* Eichelter, P. (Dept. Surg., State U. New York, Buffalo) and W. G. Schenk. *Arch Surg* 96(6):883-886, 1968.

Cardiac output, pancreatic arterial blood flow, and pancreatic secretion were studied in 8 mongrel dogs (19-29 kg) before, during and after whole-body hypothermia, produced by blood cooling to 28C. Cardiac output fell steadily during cooling to 42% of normothermic values at 30C and to 28% at 28C. Pancreatic arterial inflow fell at a slower rate, and was 70% of normothermic values at 30C and 40% at 28C. A preferential shift of blood flow toward the pancreatic area occurred early in hypothermia, but disappeared after 1-2 hr of sustained hypothermia. Pancreatic secretion was promptly reduced to 50% of normothermic values early after the start of hypothermia, but showed a rebound to 85% at 31C, falling again with continued cooling. The possibility of homeostatic mechanisms designed to preserve pancreatic function during hypothermia is suggested by the preferential shift in the distribution of cardiac output to the pancreatic area and by the rebound in pancreatic secretion after the initial hypothermic depression.

6772 **STUDY OF THE EXOCRINE ACTIVITY OF THE HUMAN PANCREAS BY MEANS OF CONTINUOUS INFUSIONS OF SECRETIN. I. EFFECT OF INCREASING DOSES ON FLUID AND ELECTROLYTE SECRETION.** *(Fr.)* Ribet, A. (Purpan Hosp., Toulouse, France), J. P. Pascal and N. Sannou. *Arch Mal Appar Dig* 56(7/8):677-684, 1967.

Secretin was administered by continuous infusion to 20 adults at doses of 1-5 U/kg/hr, and measurements of total volume and electrolytes were made on 3 samples of pancreatic secretion taken between 50 and 80 min. Although values varied widely, maximum volume of secretion (269 ± 90 ml/30 min) and maximum bicarbonate production (711 ± 166 μEq/min) were reached at a dose of 3 U/kg/hr. The ratio of bicarbonate/chloride concentration decreased as the dose of secretin increased from 1-4 U/kg/hr, then increased at doses of 5 U/kg/hr. The values obtained are compared to previous results in animals and those obtained with single injections of secretin.

6773 **STUDY OF THE EXOCRINE ACTIVITY OF THE HUMAN PANCREAS BY MEANS OF CONTINUOUS INFUSIONS OF SECRETIN. II. EFFECT OF INCREASING DOSES ON BASAL LIPASE PRODUCTION AND LIPASE PRODUCTION FOLLOWING INJECTION OF PANCREOZYMIN.** *(Fr.)* Ribet, A. (Purpan Hosp., Toulouse, France), J. P. Pascal and N. Sannou. *Arch Mal Appar Dig* 56(7/8):685-690, 1967.

The lipase response to a single injection of pancreozymin was measured in patients given i.v. infusions of secretin in doses ranging from 1-5 U/kg/hr. There was a significant increase in the mean lipase flow/min during the 40 min following injection at doses of secretin from 1-3 U/kg/hr, followed by a decline at higher doses. Maximum lipase production showed a significa change (decline) only at a dose of 4 U/kg/hr of secretin. Basal output, measured 60-80 min following the start of the secretin infusion and before injection of pancreozymin, showed an increase from doses of 1-3 U, followed by a decline, but values varied too widely for statistical analysis. It is suggested that the increased output results entirely from an increase in volume of flow produced by secretin at the lower doses, rather than from a potentiation. At higher doses, however, secretin may inhibit the response to pancreozymin.

6774 **ETHANOL AND THE INCORPORATION OF ^{32}P INTO THE PHOSPHOLIPIDS OF THE RAT PANCREAS. II. EFFECTS OF THE ACUTE INGESTION AND WITHDRAWAL OF ETHANOL.** *(Sp.)* Ferez, A. (J. J. Aguirre Hosp., Santiago, Chile), L. Costamaillere, H. Orrego and E. Navia. *Rev Med Chile* 96(4):226-229, 1968.

6775 **AN EXPERIMENTAL STUDY OF THE EFFECT OF METOCLOPRAMIDE ON EXOCRINE PANCREATIC SECRETION.** *(Fr.)* Wettendorff, P. (Lab. Pharmacodyn. Ther., Free U. Brussels, Belgium), A. Dumont and A. Delcourt. *Acta Gastroent Belg* 30(11):730-734, 1967.

6776 **THE FLOW OF ENZYMES IN THE PANCREATIC DUCT AND THEIR RELEASE INTO THE BLOOD.** *(Sp.)* Ribera, M. T. (Santa Cruz y San Pablo Hosp., Barcelona, Spain), L. Grammatica and J. Pi-Figueras. *Bull Soc Int Chir* 27(2):89-93, 1968.

See also: 6880,7276,7288

6777 **LATE CHANGES AFTER PARTIAL HEPATECTOMY IN THE NORMAL RAT.** *(E.)* Bengmark, S. (Dept. Surg. Anat., U. Göteborg, Sweden), R. Olsson and B. Rehnström. *Scand J Gastroent* 3(5):525-528, 1968.

Partial hepatectomy, with the removal of 75% of the liver parenchyma, was performed in 82 male Sprague-Dawley rats (300-350 g) and its effects on body, liver and spleen weight, liver nitrogen concentration, serum glutamic oxaloacetic and glutamic pyruvic transaminase activity, and serum albumin and α-, β-, and γ -globulin concentrations were investigated 2, 6, 8, 12, 15 and 18 months after surgery. Liver weight, but not spleen weight, was dependent on body weight, there was no significant difference in liver weight between hepatectomized and control animals, nor was there any significant variation with time. In addition, no significant differences were observed between operated and nonoperated animals in body weight, spleen weight, liver nitrogen concentration, or enzyme activity. Hepatectomized rats had significantly lower serum albumin concentrations and significantly higher β-globulin levels than controls 12 months postoperatively. No age-related changes could be detected using the parameters of this study. The liver which has regenerated after extensive resection does not differ in a biologically significant way from the liver of a nonoperated rat of a similar age.

6778 **STUDIES ON HEPATIC GLYCOLYTIC ENZYMES OF NORMAL AND SCORBUTIC GUINEA PIGS ADMINISTERED ACETOACETATE.** *(E.)* Nayudu, S. G. (Dept. Biochem., Nagpur U., India), V. Krishnamurthi and M. C. Nath. *Proc Soc Exp Biol Med* 128(3):885-887, 1968.

To study the effect of acetoacetate administration on the activities of 3 hepatic enzymes (phosphorylase, phosphoglucomutase and glucose-6-phosphatase) in normal and scorbutic guinea pigs, healthy male animals were divided into 4 groups: normal (group I); normal injected with acetoacetate (group II); scorbutic (group III); and scorbutic injected with acetoacetate (group IV). Animals of groups I and II received 5 mg of ascorbic acid daily; those in groups II and IV received acetoacetate daily. All animals were killed after 21 days and their livers were examined. Scurvy caused an increase in the total available activity of glucose-6-phosphatase and a decrease in the phosphorylase and phosphoglucomutase activities. Acetoacetate produced an increase in the activities of phosphorylase and glucose-6-phosphatase.

6779 **BIOSYNTHESIS OF PHOSPHATIDYLCHOLINES IN RAT LIVER.** *(E.)* Arvidson, G. A. E. (Dept. Med. Chem., Chem. Ctr., U. Lund, Sweden). *Europ J Biochem* 5(3):415-421, 1968.

The incorporation of radioactively labelled choline and methionine into subfractions of the phosphatidylcholines (PC) of liver and blood plasma was studied in Sprague-Dawley rats. Eight different molecular types of PC were isolated by thin-layer chromatography on $AgNO_3$-impregnated plates followed by reversed-phase partition thin layer chromatography. Total incorporation of (1,2-^{14}C) choline into the liver PC of fasting male rats reached a peak 1hr after injection and declined to half its peak value during the next 11 hr. Incorporation into the mono- and dienoic fractions followed a similar time course, while the tetraunsaturated PC continued to incorporate isotope up to 8 hr after injection and the hexaunsaturated fraction rapidly reached a plateau at a low level. The labelled choline was incorporated most rapidly in palmitoyl-oleoyl and palmitoyl linoleoyl PC but was very slow in the stearoyl-arachidonoyl type. The labeling pattern of blood-plasma PC was initially very similar to that of the liver PC; 80% of the incorporated isotope was found in the mono- and dienoic subfractions. The incorporation of labeled choline into subfractions of liver PC of fed rats was similar to that obtained for the fasting animals. Following injection of L-(Me-^{14}C) methionine into fasting and fed rats, 75-85% of the total (^{14}C) phosphatidylcholine was demonstrated in the tetra- and hexaenoic liver PC. The labeling pattern of the PC differed in the plasma, with the mono- and dienoic fractions containing relatively more isotope than corresponding fractions of the liver PC. The incorporation of isotope into the palmitic and docosahexenoic acid fractions of liver PC was greater, and that into the stearoyl-arachidonoyl species was lower, than would be expected from a stoichiometric methylation of all fractions of PC. In both male and female rats, the greatest decrease during fasting occurred in the mono- and dienoic fractions. These data support a heretofore unknown complexity in the metabolism of the 2 major glycerophosphatides in rat liver.

6780 **ON THE SITE OF SYNTHESIS OF ENZYMES TIGHTLY BOUND TO MITOCHONDRIAL STRUCTURE IN RAT LIVER.** *(E.)* Kleitke, B. (Inst. Circul. Res., German Acad. Sci., Berlin-Buch, Germany) and A. Wollenberger. *FEBS Letters* 1(3):187-190, 1968.

In male Wistar rats, i. p. injection of chloramphenicol in 4 successive doses of 53 mg/100 g body weight (16-47 hr prior to death) failed to block the effect of a single i. p. dose of 100 μg/100 g body weight (44 hr prior to death) of 3,3',5-triiodo-L-thyronine on α-glycerophosphate dehydrogenase or succinic dehydrogenase activity in the liver mitochondria. The percentage increase in the activity of both enzymes was the same in chloramphenicol-treated and control rats. In rats given ^{14}C-labeled algal protein hydrolysate 1 hr before death, no specific inhibition of amino acid incorporation into either structural or total mitochondrial protein occurred, and the ratio of structural to total protein was unchanged by chloramphenicol. These findings indicate that the mitochondria synthesize only a very small fraction of their structural proteins by a process which is sensitive to chloramphenicol and that most of their structural proteins are synthesized by a process which takes place outside the organelles. It seems unlikely that mitochondrial α-glycerophosphate and succinic dehydrogenase are synthesized inside the liver mitochondria.

6781 **LYSOSOMAL ENZYME ACTIVITY IN HUMAN LIVER IN RELATION TO THE AGE OF THE PATIENT AND IN CASES WITH OBSTRUCTIVE JAUNDICE.** *(E.)* Björkerud, S. (Sahlgren Hosp., U. Göteborg, Sweden), P. Björntorp and T. Schersten. *Scand J Clin Lab Invest* 20(3):224-230, 1967.

Basal (latent) and maximal (induced by Triton X-100) activities of acid phosphatase, aryl sulfatase, β-glucuronidase, and cathepsin were determined in homogenates of human liver from 23 patients (8 men and 15 women, 19-70 yr old) operated on for uncomplicated gallbladder disease and from 12 patients (7 men and 5 women, 20-70 yr old) operated on for obstructive jaundice due to common duct stones. Maximal activity of acid phosphatase and aryl sulfatase correlated positively with the age of the patient. The ratio between maximal and basal activity (a measure of the degree of enzyme retention by the lysosomes) of aryl sulfatase decreased significantly with increasing age. In patients with obstructive jaundice, the degree of retention of acid phosphatase and cathepsin activity in the lysosomes was significantly decreased. Cholic acid and bilirubin added *in vitro* to the homogenate (10^{-4}-10^{-2} M) caused release of lysosomal enzymes. It is suggested that the retention of bile products in obstructive jaundice causes release of hydrolytic enzymes from liver lysosomes, which may be responsible for the common occurrence of scattered cellular necrosis of the liver in cholestatic conditions.

6782 **KINETICS OF DYE EXCRETION IN THE BILE. I. CRITICAL STUDY OF THE MEASUREMENT OF BILIARY TRACT VOLUME WITH FREE BILE FLOW.** *(Fr.)* Sicot, C. (Beaujon Hosp., Clichy, France), A. Richard and J. P. Benhamou. *Rev Franc Etud Clin Biol* 13(3):270-273, 1968.

A formula was developed to calculate the mean volume of the biliary tract in rabbits from the time required for dye to reach a maximum concentration in the bile after a single i. v. injection of ^{131}I-labeled Rose Bengal, BSP, or indocyanin green. In this formula, it is assumed that the time required for dye to pass through the hepatocyte is negligible. However, values for biliary tract volumes differed significantly with the dye used and the rate of bile flow, being smallest with indocyanin green, somewhat larger with BSP, and largest with Rose Bengal. These differences are attributed to differences in the time required for the dye to pass through the hepatocyte. Values obtained with indocyanin green are considered to be the most accurate. Significantly smaller biliary tract volumes were obtained by decreasing bile flow with larger doses of indocyanin green. Significantly larger volumes were obtained by increasing bile flow with perfusions of sodium dehydrocholate; this is due to error introduced by the relatively large outflow of bile which occurs while the dye is passing through the hepatocyte. The method is concluded to be unreliable since the time required for dye to pass through the hepatocyte is appreciable and cannot be measured.

6783 **THE METABOLISM OF BILE ACIDS IN THE DEVELOPING RAT LIVER.** *(E.)* Danielsson, H. (U. Washington Sch. Med., Seattle) and W. J. Rutter. *Biochemistry* 7(1):346-352, 1968.

The metabolism of tritium-labeled cholic acid, deoxycholic acid, 5β-cholestane- 3α, 7α, 12α-triol, and 7α-hydroxycholest-4-en-3-one was studied in liver hemogenates from rat embryos and suckling rats (Sprague-Dawley strain). The liver homogenates and bile acids were incubated at 37C for either 30 min or 2 hr with ATP, coenzyme A, NADPH, and, when appropriate, taurine. The conversion of the different substrates was calculated from the amount of radioactivity in the appropriate chromatographic zones. The activity of enzymes concerned with bile-acid conjugation increased 30- to 40-fold between 15 days after fertilization and 5 days after birth, with the largest increment occurring between 20 days after fertilization and 2 days after birth; the metabolism of 5β-cholestane-3α, 7α, 12α-triol increased 15-fold during the developmental period studied and that of 7α-hydroxycholest-4-en-3-one increased almost 30-fold between 17 days after fertilization and 4 days after birth. The results suggest that the activities of the enzymes concerned with the conversion of cholesterol to bile acid increase synchronously in the developing rat liver.

6784 **CATIONS OF MITOCHONDRIA IN ETHIONINE INTOXICATION.** *(E.)* Christie, G. S. (Chicago Med. Sch. Inst. Med. Res., Illinois) and J. D. Judah. *Lab Invest* 18(2):108-113, 1968.

Mitochondria isolated from the livers of female Sprague-Dawley rats 4 hr after poisoning with ethionine (1 g/kg i. p.) showed greatly reduced contents of K^+ and Mg^{++} (78.1±6.5 and 17.1 ± 2.9 μM/g dry wt, resp., compared to control levels (142 ± 12.9 and 39.8 ± 3.7 μM/g, resp.). The ATP levels of thes particles were also greatly reduced (from 6.8 ± 2.0 to 2.3 μM/g dry weight), but the Ca^{++} content remained normal (7.1±3.0 μM/g). Prior administration of adenine (250 mg/kg 60 min before sacrifice) restored the cation pattern, but did not produce normal K^+ and Mg^{++} contents. Ethionine-treated male rats showed far less marked changes in the cation content of isolated mitochondria than females. Control rats given carbon tetrachloride by stomach tube (2.5 ml/kg) revealed normal K^+ and Mg^{++} contents and elevated Ca^{++} levels after 4-5 hr. In spite of the altered K^+ and Mg^{++} contents observed following ethionine poisoning, K^+ and Mg^{++} uptake by isolated mitochondria (studied by reisolating the particles after incubation in the appropriate medium) was normal. Valinomycin (2.5 μg) stimulated K^+ uptake in the isolated mitochrondria from intoxicated rats; however, the response to this antibiotic could be abolished by procedures which do not affect mitochondria from normal animals. Normal rates of H^+ secretion in exchange for K^+ were demonstrated at pH 7.15 and 7.50, while increased H^+ uptake and K^+ loss occurred at pH 6.3 in mitochondria from poisoned rats. In animals given 250 mg/kg of adenine s. c. 3 hr after administration of ethionine, the H^+ entry and loss of K^+ were within normal limits over a pH range of 6.3 to 7.1. Electron microscopy of isolated mitochondria revealed no abnormalities attributable to ethionine poisoning or to the isolation procedure. The addition of ATP to the homogenizing medium had no effect on the cation content of the isolated mitochondria. The unusual occurrence of low cation content in the presence of normal transport systems is discussed and a hypothesis to account for these observations is presented.

6785 **STERO-BILE ACIDS AND BILE ALCOHOLS. CII. METABOLISM OF LITHOCHOLIC ACID IN MOUSE AND DOG.** *(E.)* Kurata, Y. (Hiroshima U. Sch. Med., Japan). *Hiroshima J Med Sci* 16(3/4):281-285, 1967.

Bile acids extracted 16 hr after i. p. injection of the sodium salt of tritium-labeled lithocholic acid (40 μg) into mice (2) or dogs (1) were separated by thin-layer chromatography (isooctane:ethyl acetate:acetic acid, 10:10:2) or reversed-phase column partition chromatography (mobile phase-methanol:water, 165:135; stationary phase-chloroform:heptane, 45:5) and the radioactive components were identified. In mice, lithocholic acid was hydroxylated at C_6 to yield 3α, 6β-dihydroxycholanic acid, which could be further hydroxylated to trihydroxycholanic acid. In dogs, lithocholic acid was hydroxylated at C_7 to yield chenodeoxycholic acid, which was not hydroxylated further.

6786 **STUDIES ON CARBOHYDRATE METABOLISM IN THE LIVER. 1) PATTERNS OF GLUCOSE METABOLIZING ENZYMES IN THE LIVER IN PATIENTS WITH HYPERTHYROIDISM.** *(Jap.)* Iwasaki, Y. (Fac. Med., Kyoto U., Japan). *Jap Arch Intern Med* 14(6):159-165, 1967.

Carbohydrate metabolism in the liver of 14 patients with hyperthyroidism was studied by determining the glucose-6-phosphatase (G6P-ase), fructose-1, 6-diphosphatase (FDP-ase), phosphohexoseisomerase (PHI), glucose-6-phosphate dehydrogenase (G6PDH), and lactic dehydrogenase (LDH) activities in percutaneous liver biopsy specimens. Four of the 14 patients were restudied after treatment with ^{131}I. Liver specimens from 7 patients without liver damage were used as controls. In the hyperthyroid group, the activities of G6P-ase, FDP-ase, and PHI showed significant elevation (276%, 250% and 136%, resp. as compared with controls) whereas G6PDH and LDH showed no significant differences from the control group. In the 4 cases of treated hyperthyroidism, the elevated PHI activity quickly returned to normal, but G6P-ase and FDP-ase showed delayed recovery and remained slighty elevated. The elevations of G6P-ase, FDP-ase, and PHI are similar to to the changes seen in experimentally induced hyperthyroid rat liver, and are thought to reflect an increase in hepatic gluconeogenesis. However, the activities of G6PDH and LDH noted in human hyperthyroid subjects are different from the elevated values seen in the experimental situation; further observations are needed to explain this discrepancy.

6787 **STUDIES ON CARBOHYDRATE METABOLISM IN THE LIVER. 2) PATTERNS OF GLUCOSE METABOLIZING ENZYMES IN THE LIVER IN PATIENTS WITH LIVER DISEASE.** *(Jap.)* Iwasaki, Y. (Fac. Med., Kyoto U., Japan). *Jap Arch Intern Med* 14(6):167-176, 1967.

The changes in carbohydrate metabolism in the livers of 10 patients with acute viral hepatitis, 14 with chronic active hepatitis, 9 with chronic inactive hepatitis, and 9 with hepatic cirrhosis were studied by determining the glucose-6-phosphatase (G6P-ase), fructose-1, 6-diphosphatase (FDP-ase), phosphohexoseisomerase (PHI), glucose-6-phosphate dehydrogenase (G6PDH), and lactic dehydrogenase (LDH) activities in percutaneous liver biopsy specimens. Liver specimens from 7 patients without liver disease were used as controls. In acute hepatitis, G6P-ase, FDP-ase, and PHI were significantly reduced (34%, 38%, and 80%, resp. compared with controls), whereas G6PDH was significantly elevated (266%). In chronic active hepatitis, G6PDH (156%) was significantly elevated, whereas G6P-ase (68%) and FDP-ase (74%) were significantly reduced. PHI showed no significant change. In chronic inactive hepatitis, no significant alterations in enzyme activities were observed. In hepatic cirrhosis, G6P-ase (67%) and FDP-ase (66%) were significantly reduced, while the other enzymes showed no significant differences from the control group. The elevation of G6PDH in acute and chronic active hepatitis is attributed to the activation of regenerative hepatocellular mechanisms through production of $NADPH_2$ and pentose-phosphate.

6788 **STUDIES ON CARBOHYDRATE METABOLISM IN THE LIVER. 3) PATTERNS OF GLUCOSE METABOLIZING ENZYMES IN THE LIVER IN PATIENTS WITH PRIMARY HEPATOMA AND IN TUMOR BEARERS.** *(Jap.)* Iwasaki, Y. (Fac. Med., Kyoto U., Japan). *Jap Arch Intern Med* 14(7):193-199, 1967.

The changes in carbohydrate metabolism in malignant liver tissue from 8 patients (7 men and 1 woman) with primary hepatoma and 10 patients with hepatic metastasis (9 with gastric carcinoma and 1 with colonic carcinoma) were studied by determining the glucose-6-phosphatase (G6P-ase), fructose-1, 6-diphosphatase (FDP-ase), phosphohexoseisomerase (PHI), glucose-6-phosphate dehydrogenase (G6PDH), and lactic dehydrogenase (LDH) activities in liver biopsy specimens obtained at laparotomy or with the aid of peritoneoscopy. None of the primary hepatoma specimens showed any G6P-ase or FDP-ase activity, which correlates with the absence of gluconeogenesis in hepatoma tissue. PHI and LDH activities were in the normal range. G6PDH (236% of control) showed significant elevation, reflecting the active synthesis of nucleic acid and protein by tumor tissue. In specimens of metastatic carcinoma, G6P-ase, FDP-ase, PHI, and LDH showed no significant difference from control values; however, G6PDH was significantly elevated (238%). This is attributed to the increased proteins produced by the liver which are readily consumed by the tumor cells.

6789 **A CONTRIBUTION TO THE PROBLEM OF THE SECRETION OF TRANSAMINASES INTO THE BILE.** *(Cz.)* Volek, V. (Fac. Gen. Med., Charles U., Prague, Czechoslovakia), V. Baláš and R. Papežová. *Cesk Gastroent Vyz* 21(7):450-454, 1967.

The transaminase activity of both choledochal (T-tube) bile and bile obtained from the duodenum was investigated in 7 cholecystectomized subjects and 45 patients with various hepatobiliary diseases. The results showed that the transaminase activity (especially pyruvic transaminase) was higher in drained bile, and significantly higher in "B" bile from the duodenum than in "A" bile. The transaminase activity of the bile remained fairly constant during the day, regardless of the volume of bile secreted, and corresponded roughly to the total transaminase activity of the serum, although there was no correlation between serum and biliary transaminase levels. The transaminase activity in duodenal bile from patients with viral hepatitis, cirrhosis, or diseases of the gallbladder and bile ducts was not significantly different from that in controls.

6790 **EFFECT OF PAPAIN ON MITOTIC ACTIVITY AND REGENERATION IN THE LIVER.** *(Hun.)* Bartók, I. (U. Med. Sci., Szeged, Hungary), I. Piukovich, M. Tényi, A. Tószegi and G. Thury. *Kiserl Orvostud* 19(4):413-418, 1967.

In healthy rats weighing 180-200 g, a single i. m. injection of papain (20 mg/kg) was followed by significant stimulation of mitosis, an increase in liver weight, and the development of histological changes typical of hepatic regeneration. At 24 hr, for example, the liver weight had increased to 4.1 g/100 g compared to 3.3 g/100 g in untreated rats, and at 48 hr this was 4.6 g/100 g; the mitotic index was zero in all rats except those sacrificed 48 hr after papain injection, in which it had increased to 11. There were no signs of liver damage. When rats were partially hepatectomized (70%) and the same dose of papain was given 6 hr later, regeneration was more rapid in treated animals and the regenerating liver showed a larger number of mitotic figures. At 30 hr after hepatectomy, for example, the extent of regeneration was 29% in treated animals and 24% in controls, while at 54 hr these values were 65% and 58%, resp. The mitotic index at 30 hr was 45 in rats given papain 24 hr before and 11 in control rats, but by 54 hr the two values were 9 and 10, resp.

6791 EFFECT OF INSULIN AND OTHER HORMONES ON THE PENETRATION OF COBALT IONS AND VITAMIN B$_{12}$ INTO THE LIVER CELL.

(Pol.) Kaliciński, A. (2nd Clin. Int. Dis., Acad. Med. Bialystok, Poland), I. Kinalska and J. Chlebowski. *Pol Arch Med Wewnet* 39(2):175-180, 1967.

The uptake of ^{60}Co-labeled vitamin B$_{12}$ and $CoCl_2$ into the liver cell was studied in the livers of male Wistar rats (130-180 g body weight), both *in vivo* and *in vitro*. The penetration of $CoCl_2$ was significantly increased by both endogenous and exogenous insulin, as well as by tolbutamide or chlorpropamide, but the action of insulin was abolished by ouabain or cortisone, penetration of $CoCl_2$ was decreased by growth hormone. Insulin also significantly stimulated the penetration of labeled vitamin B$_{12}$. It is suggested that ATPase and insulin may play a role in the active transport of micromolecules within the hepatocyte.

6792 BILIARY EXCRETION OF ALCOHOL DEHYDROGENASE.

(E.) Mezey, E. (St. Luke's Hosp. Ctr., New York, N. Y.), G. R. Cherrick and P. R. Holt. *J Lab Clin Med* 71(5):798-806, 1968.

The tissue distribution and fate of alcohol dehydrogenase (ADH) released from the liver following acute hepatocellular damage due to CCl_4 was investigated in rats. Female Sprague-Dawley rats (175-250 g) were divided into 4 groups: 1) bile duct fistula; 2) bile duct fistula followed by CCl_4 (2 ml/kg via a nasogastric tube); 3) bile duct ligation; and 4) bile duct ligation followed by CCl_4 as in group 2. Rat pairs were killed at 2-16 hr, 4-28 hr, and 8-72 hr after CCl_4 administration or after operation, the activity of ADH in various tissues, including bile and blood, was determined, and the liver was examined histologically. Plasma and bile GOT and lactic dehydrogenase (LDH) activities were also determined. The total quantity of ADH in organs other than the liver was very small, although significant activity was detected in the mucosa of the stomach and small intestine. Hepatic ADH levels/mg protein fell 10% in 8-10 hr, 20% in 24 hr, and 30% in 28 hr in both the bile fistula and the bile-duct ligated rats; however, the total hepatic ADH content remained at the lower limits of that found in normal animals since the decrease in unit enzyme activity was accompanied by an increase in total liver weight. In controls (no CCl_4 given) there was no change in hepatic ADH activity. CCl_4 accentuated the decrease in bile flow that occurs after bile duct cannulation; at 16 hr almost no bile was secreted. In animals with bile fistulas and CCl_4 treatment, ADH appeared in the plasma at 4 hr, peaked at 28 hr, and disappeared by 72 hr. Plasma GOT and LDH elevations occurred during this same period; however, ADH plasma activity showed a slower increase and delayed peak as compared to LDH. No ADH was detected in the plasma or bile of control bile-fistula rats, and no significant changes in plasma GOT or LDH occurred after bile-duct cannulation. In the bile of bile-fistula rats receiving CCl_4, ADH appeared at 4-5 hr, peaked at 8-10 hr, and disappeared at 16 hr, with the fall in ADH activity paralleling the reduction of total bile flow Only moderate increases in GOT and LDH were noted. The concentration of ADH in bile was greater (5 times) than that in plasma, whereas GOT and LDH were greater (100 times) in plasma than in bile. It is concluded that ADH is principally found in the liver and differs from other enzymes whose presence in serum may result from release from several different organs. These observations also explain the finding that plasm ADH activity is highest in patients with liver-cell necrosis and intrahepatic cholestasis.

6793 INTRINSIC HEPATIC CONTROL OF PLASMA ALBUMIN CONCENTRATION.

(E.) Tracht, M. E. (Mount Sinai Hosp., New York, N. Y.), L. Tallal and D. G. Tracht. *Life Sci* 6(24):2621-2628, 1967.

To determine whether plasma albumin metabolism is controlle directly by the colloidal osmotic pressure of the plasma, livers from female Sherman rats were excised and perfused for 2 hr with 3 different solutions: the control group, in which poole rat blood adjusted to a hematocrit with 0.85% saline was used as the perfusate; the plasmaphoresis group, in which pooled ra erythrocytes suspended in 0.85% saline containing 5 mEq/l potassium, with the hematocrit adjusted to 35%, was used as the perfusate; and the dextran group, in which pooled erythrocytes suspended in 6% dextran solution containing 5 mEq/l potassium, with the hematocrit adjusted to 35%, was used as the perfusate. In all cases, the perfusate volume was 45 cc. After 15 min of perfusion, 6.1 µC of DL-leucine-1-^{14}C was added to the perfusate. At the end of the perfusion period, the perfusate total protein, albumin and globulin concentratio were determined, total bile output was measured, and liver sections were examined histologically. No statistically significant alterations in bile production were noted in the 3 groups, and no noteworthy differences were observed on gross or mic scopic examination of the liver, all shwoing relatively good preservastion of normal hepatic structure. Both the plasmaphoresis and dextran groups showed an equivalent decrease in protein and albumin concentrations. The incorporation of radioactive leucine into the serum albumin was doubled in the plasmaphoresis group and unchanged in the dextran group, as compared to the control group. By contrast, plasmaphoresis exerted no significant effect on the incorporation of radioactivity into liver protein, although this was significantly decreased in the dextran group. These findings suggest that the liver is directly responsive to the oncotic pressure of the perfusing fluid and that the rate of appearance of radioactive plasma albumin in the circulation is inversely related to the colloid pressure of the plasma.

6794 DIETARY AND HORMONAL EFFECTS ON ADENOSINE TRIPHOSPHATE:ADENOSINE MONOPHOSPHATE PHOSPHOTRANSFERASE ACTIVITY

IN RAT LIVER. *(E.)* Adelman, R. C. (Temple U. Sch. Med., Philadelphia, Pa.), C. H. Lo and S. Weinhouse. *J Biol Chem* 243(10):2538-2544, 1968.

Adenylate kinase activity was measured in rat liver tissue by a spectrophotometric procedure in which ADP formation from ATP and AMP was coupled to oxidation of NADH via pyruvate kinase, phosphoenolpyruvate, and lactate dehydrogenase. Activity was extremely high (135 μmoles/min/g tissue) in the supernatant fraction obtained by centrifugation of the 0.25 M sucrose homogenate at 100,000 X gravity and was much lower in the mitochondria (13 U/g). No activity was found in the microsomes or nuclei. Both supernatant and mitochondrial fractions were nearly as active with other nucleoside monophosphates as cosubstrates as with AMP, but were much less active with other nucleoside triphosphates than with ATP. A 48-hr fast increased the liver supernatant enzyme activity to 374 U/g, which was lowered in 24 hr to 40 U/g by feeding a high glucose diet. The high fasting level was lowered only slightly by feeding a high fat or high protein diet. In fed alloxan-diabetic rats, activity was about as high as in fasted normal rats, and was lowered by insulin treatment to levels found in normal fed rats. Activities were somewhat lower in adrenalectomized rats, and the effects of fasting and glucose feeding were not as pronounced as in normal rats. The mitochondrial enzyme activity remained essentially unchanged by dietary and hormonal alterations. A clearer picture of the physiological significance of these alterations will be attained when the individual enzymes can be isolated and studied separately.

6795 **EFFECT OF PHENOBARBITAL TREATMENT ON THE ACTIVITIES OF NADPH-DEPENDENT ENZYMES OF LIVER MICROSOMES IN FASTED OR SUCROSE FED RATS.** *(E.)* Kato, R. (Natl. Inst. Hyg. Sci., Tokyo, Japan). *Jap J Pharmacol* 17(2):181-198, 1967.

Female and male Sprague-Dawley rats were divided into 3 groups and fed a standard chow diet, sucrose, or fasted for 72 hr; one-half of each group was treated with phenobarbital (80 mg/kg) or methylcholanthrene (40 mg/kg) 72 and 48 hr prior to sacrifice. The administration of phenobarbital markedly increased the activities of liver microsomal NADPH oxidase, NADPH-cytochrome c reductase, NADPH-ferricyanide reductase, NADPH-neotetrazolium and NADPH-dichloroindophenol diaphorase and the amounts of microsomal protein and P-450. Starvation of female rats markedly increased the activities of some NADPH-dependent enzymes and the amount of P-450. Sucrose feeding of female and male rats markedly depressed the activities of all microsomal NADPH-dependent enzymes and the amount of P-450. The effect of phenobarbital on the microsomal enzymes and P-450 was potentiated by starvation in females and additive in males, while methylcholanthrene and starvation had an additive effect on enzyme activities in females. Phenobarbital had similar effects on the microsomal enzymes and P-450 in sucrose fed rats as in the controls. The incorporation of amino acids into liver microsomal protein was decreased by starvation; however, phenobarbital increased this incorporation in the starved rats. These results suggest that the activities of microsomal NADPH-dependent enzymes are related to protein synthesis in liver microsomes.

6796 **THE EFFECT OF A SINGLE LARGE DOSE OF SALICYLATE AND PHENYLBUTAZONE ON THE GLUCOSAMINE-6-PHOSPHATE SYNTHESIS *IN VIVO* BY THE LIVER AND BY THE GASTRIC MUCOSA OF RATS.** *(E.)* Schönhöfer, P. (Inst. Pharmacol., U. Bonn, Germany) and K. H. Perrey. *Med Pharmacol Exp (Basel)* 17(2):175-182, 1967.

In 2 groups of 25 female Wistar rats (250-300 g), 20 animals received phenylbutazone (200 mg/kg) or sodium salicylate (1000 mg/kg) p.o., resp., and 5 rats in each group served as untreated controls. Animals were sacrificed 3-24 hr after drug administration, and liver and gastric mucosal glucosamine-6-phosphate (GA-6-P) levels were determined. The activity of the liver enzyme was not influenced by either drug, whereas the activity of the gastric mucosal enzyme was distinctly inhibited by both: phenylbutazone decreased the ability of the gastric mucosa to synthesize GA-6-P to 37.4% of the control value and salicylate reduced GA-6-P synthesis to 15.6% of the control value 24 hr after drug administration, even though salicylates induced less macroscopically detectable damage to the gastric mucosa than phenylbutazone. The inhibition of GA-6-P synthesis by these drugs may play a part in the impairment of mucoprotein production by the gastric mucosa; this may account for the symptoms of gastric intolerance observed with the use of these drugs in some patients.

6797 **FATTY LIVER CAUSED BY THE REPETITIVE ADMINISTRATION OF RELATIVELY SMALL DOSES OF CARBON TETRACHLORIDE IN RATS.** *(E.)* Osumi, Y. (Fac. Med., Kyoto U., Japan), Y. Amano and K. Shimamoto. *Jap J Pharmacol* 17(2):298-307, 1967.

Male Wistar rats were treated with 0.025, 0.075, and 0.15 ml/kg of CCl_4 dissolved in sesame oil twice weekly for 8 weeks in an attempt to establish a dose-response curve for the development of CCl_4-induced fatty liver. Liver phospholipids were increased by the administration of CCl_4 for 2 weeks, but were decreased after 8 weeks. Serum lipoprotein levels were reduced at 2 weeks but elevated at 8 weeks. The level of liver fatty acids was increased proportionately to the dosage of CCl_4; however, serum fatty acids were decreased initially and elevated only at the end of the 8-week period. An increase of oleic acid and a decrease of stearic and arachidonic acids among the liver fatty acids also correlated positively with the CCl_4 dosage; the serum fatty acid composition did not show these changes. The histological changes in the liver, such as fatty degeneration and fibrous proliferation, coincided well with the biochemical changes noted in the liver and serum lipids.

6798 **EFFECT OF BLOOD FLOW REVERSAL IN LIVER AUTOTRANSPLANTS UPON THE SITE OF HEPATOCYTE REGENERATION.** *(E.)* Sigel, B. (Woman's Med. Coll. Pennsylvania, Philadelphia), L. B. Baldia, S. A. Brightman, M. R. Dunn and R. I. M. Price. *J Clin Invest* 47(6): 1231-1237, 1968.

The role of the direction of intrahepatic blood flow in determining the location of hepatocyte formation in regenerating liver was studied in dogs. Single liver lobes were autotransplanted to the neck area with reestablishment of the blood flow so as to perfuse the hepatic lobule either from portal tract to central vein or, in a reverse direction, from central vein to portal tract. Partial resection of the nontransplanted liver was later performed to induce regeneration in the grafts by humoral means. Tritiated thymidine was administered, and radioautographs were prepared from excised graft and nontransplanted liver. In the "straight" blood flow grafts as well as in all nontransplanted livers, labeled hepatocytes indicating DNA synthesis were found predominantly in the vicinity of the portal tracts. In the "reverse" blood flow grafts, labeled hepatocytes were more prevalent about the central veins. Thus, the localization of hepatocyte formation in the lobule during active liver regeneration cannot be attributed to an inherently greater capacity of periportal liver cells to divide but is probably related to their preferential exposure to blood constituent changes (humoral mechanisms). Hepatocyte regeneration in the presence of abnormal directional circulation might lead to lobular disorganization resulting in consequent biochemical aberrations despite the formation of new cells.

6799 COMPARISON OF THE EFFECT OF 1-[2'-HYDROXY-5'-CARBOXYPHENYL]-PENTANOL-1 AND OTHER CHOLERETIC AGENTS ON SECRETION OF ALKALINE PHOSPHATASE IN THE BILE. *(Fr.)* Trebaul, L. (Fac. Med. Pharm., U. Rennes, France), L. Guesnier and J. van den Driessche. *Therapie* 23(2):341-346, 1968.

Alkaline phosphatase determinations were made on bile collected from rats with biliary fistulas for 2 hr after administration of 200 mg/kg sodium dehydrocholate, 100 mg/kg cholic acid, 3 g/kg sorbitol, 100 mg/kg phenylpentanol, or 100 mg/kg 1-[2'-hydroxy-5'-carboxyphenyl]-pentanol-1 (m-[1-hydroxypentyl]-p-hydroxybenzoic acid; HCPP) through a tube placed in the duodenum. A 17% reduction occurred in the volume of bile secreted by controls, but alkaline phosphatase concentrations increased so that there was little change in the hourly output of enzyme. After 1 hr animals receiving sodium dehydrocholate had secreted 217% more bile and 63% more alkaline phosphatase than controls so that the hourly output of enzyme was 21% higher. After 2 hr the volume of bile had decreased to values approaching those in the controls and the hourly output of alkaline phosphatase was 37% lower. Cholic acid and sorbitol caused almost no change in the volume of bile secreted and produced only slight changes in enzyme output. Phenylpentanol produced a significant increase in the volume of bile secreted and a significant decrease in the alkaline phosphatase concentration; little change occurred in the hourly output of enzyme. In animals given HCPP, a significant increase in both bile flow and alkaline phosphatase concentration was associated with a very large increase in the enzyme output (104% after 1 hr, 195% after 2 hr). These results do not show whether HCPP acts on the mechanism regulating secretion of alkaline phosphatase by the liver or whether it increases enzyme synthesis by the liver.

6800 ENZYME INDUCTION IN PERFUSED RAT LIVER BY GLUCAGON AND OTHER AGENTS. *(E.)* Knox, W. E. (Harvard Med. Sch., Boston, Mass.) and C. Sharma. *Enzym Biol Clin (Basel)* 9(1):21-30, 1968.

In isolated rat livers perfused with whole rat blood and a mixture containing 327 mg of essential and nonessential amino acids (32.2 mg α-amino nitrogen per 100 ml), hydrocortisone phosphate (1.5 mg in 1 ml initially, then 0.25 mg in 1 ml/hr) induced tryptophan oxygenase (TO) and tyrosine aminotransferase (TA) activity amounting in 4 hr to about half the levels reached after 5 hr *in vivo*. In the absence of added amino acids, the steroid induced only TA. Addition of α-methyltryptophan (10^{-3} M) to the perfusion fluid induced TO to about the same extent as hydrocortisone, approaching values obtained with these inducers *in vivo*. α-Methyltryptophan had little or no effect on *in vitro* TA activity. Only 12-17% of TO was conjugated in the livers perfused with hydrocortisone (as measured by its activity with globin in the incubation mixture), whereas 33-64% became conjugated in the livers perfused with α-methyl tryptophan. Glucagon (300 μg initially, then 150 μg/hr) induced significant TA, but not TO, activity in isolated livers fro adrenalectomized rats, as it does *in vivo*. However, glucagon, unlike α-methyltryptophan, did not enhance the induction of TA or TO by hydrocortisone *in vitro*.

6801 PHOSPHOPROTEIN PHOSPHATASE ACTIVITY DURING REGENERATION OF THE LIVER IN RATS. *(Ser.)* Rotović, B. (Med. Fac., U. Belgrade, Yugoslavia) R. Vlajnić, R. Živkovic, I. Kulić-Japundžić and M. Japundžić. *Acta Med Iugosl* 21(2/3):158-164, 1967.

6802 EFFECT OF MAGNESIUM DEFICIENCY ON LIPID METABOLISM IN RATS. *(Sl.)* Červeň, J. (Res. Inst. Human Nutrit., Bratislava, Czechoslovakia) and E. Ginter. *Cesk Gastroent Vyz* 21(6):376-382, 1967.

6803 EFFECT OF STAPHYLOCOCCAL TOXIN ON THE SECRETION AND CHEMICAL COMPOSITION OF THE BILE IN ALBINO RATS. *(Rus.)* Drogovoz, S. M. (Ternopol Med. Inst., USSR). *Biull Eksp Biol Med* 64(8):37-38, 1967.

6804 INTERRELATIONS BETWEEN AN INCREASED HEAT PRODUCTION WHICH IS INADEQUATE FOR GAS EXCHANGE AND THE DISSOCIATION OF RESPIRATION AND PHOSPHORYLATION IN THE LIVER CELL IN CASES OF DIPHTHERIA INTOXICATION. *(Rus.)* Veselkin, P. N. (Inst. Exp. Med., Acad. Med. Sci. USSR, Moscow), Ie. S. Gramenitskaia and L. I. Niselovskaia. *Biull Eksp Biol Med* 64(11):56-60, 1967.

6805 EFFECT OF HYDROXYUREA ANALOGUES IN REGENERATING RAT LIVER. *(E.)* Hill, R. B. (U. Colorado Sch. Med., Denver) and J. A. Gordon. *Exp Mole Path* 9(1):71-76, 1968.

6806 DETERMINATIONS OF THE DNA CONTENT IN THE NUCLEI OF THE HEPATOCYTES OF NORMAL AND REGENERATING RAT LIVER. *(Rus.)* Riabinina, Z. A. (Inst. Exp. Biol., Acad. Med. Sci. USSR, Moscow) and V. A. Beniush. *Biull Eksp Biol Med* 65(1):100-104, 1968.

6807 THE UTILIZATION OF TRYPTOPHAN AND ITS METABOLITES FOR PYRIDINE NUCLEOTIDE SYNTHESIS IN TUMORS AND HOST LIVER. *(E.)* McDonald, J. W. D. (U. Western Ontario, London, Canada), F. J. Rathbun and H. B. Stewart. *Cancer Res* 28(4):666-671, 1968.

6808 METHIONINE AND GLYCINE LEVELS IN THE LIVER OF RIBOFLAVIN DEFICIENT RATS. *(E.)* Arakawa, T. (Tohuku U., Sendai, Japan), T. Tamura,

. Tada and H. Hirono. *Tohoku J Exp Med* 95(3):203-205, 968.

809 EFFECT OF GALASCORBIN (VITAMINS C AND P) ON OXIDATIVE PHOSPHORYLATION THE LIVER MITOCHONDRIA UNDER CONDITIONS OF YPOTHYROIDISM. *(Rus.)* Pokotilenko, G. M. (Kiev Med. st., USSR). *Vrach Delo* (12):23-25, 1967.

810 THE EFFECT OF ETHANOL ON GALACTOSE ELIMINATION IN RATS WITH NORMAL AND HOLINE-DEFICIENT FATTY LIVERS. *(E.)* Salaspuro, M. P. es. Labs. State Alcohol Monopoly, Helsinki, Finland) and . E. Salaspuro. *Scand J Clin Lab Invest* 22(1):49-53, 1968.

811 THE ROLE OF THE LIVER IN DETOXICATION. *(Ger.)* Gramlich, F. (1st Med. Clin., U. ainz, Germany). *Landarzt* 43(31):1530-1535, 1967.

812 INDUCTION OF DRUG-METABOLIZING ENZYMES IN THE ENDOPLASMIC RETICULUM F THE HEPATOCYTE BY MEANS OF DRUGS. *(Ger.)* emmer, H. (Inst. Toxicol., U. Tubingen, Germany). *Deutsch ed Wschr* 92(44):2001-2008, 1967.

813 EFFECT OF CHLOROPHENOXYISOBUTYRATE ON RAT LIVER NON-SAPONIFIABLES. *.)* Phillips, W. E. J. (Dept. Natl. Hlth. Welfare, Ottawa, anada), M. R. Lakshmanan and R. L. Brien. *Canad J hysiol Pharmacol* 46(1):81-84, 1968.

814 THE EFFECT OF HEPATIC BLOOD SUPPLY ON ARGINASE ACTIVITY IN THE DOG IVER. *(E.)* Hirano, A. (Jikei U. Sch. Med., Tokyo, Japan). *kei Med J* 14(1):27-29, 1967.

815 THE ACTIVITY OF CARNITINE PALMITOYL TRANSFERASE IN THE LIVER MITOCHONRIA OF RATS FED THROMBOGENIC DIETS. *(E.)* uquesne, N. S. (Montreal Heart Inst., Quebec, Canada). *Canad Physiol Pharmacol* 46(1):120-123, 1968.

816 EFFECT OF PERIARTERIAL HEPATIC NEURECTOMY ON LIVER IRON METABOLISM. *Fr.)* Lenart, I. F. (Inst. Path. Anat., Ljubljana, Yugoslavia), . Rigler and V. Simsic. *Lyon Chir* 63(6):821-824, 1967.

6817 THE EFFECT OF NEPHRECTOMY AND RENAL ISCHEMIA ON LIVER REGENERATION. *(E.)* Hyde, T. A. (Endocrine Unit, U. Liverpool, England). *J Path Bact* 96(1):131-136, 1968.

6818 STIMULATION OF SEVERAL MICROSOMAL DRUG OXIDATIONS BY PHENOBARBITAL METHYLCHOLANTHRENE AND CHLOROPHENOTHANE, SINGLE AND IN COMBINATIONS. *(E.)* Uehleke, H. (Pharmacol. Inst., U. Tubingen, Germany). *Naunyn-Schmiedebergs Arch Pharmakol Exp Path* 259(1):66-90, 1967.

6819 ISOENZYMES OF HUMAN AND RAT LIVER MONOAMINE OXIDASE. *(E.)* Collins, G. G. S. (Queen Charlotte's Maternity Hosp., London, England), M. B. H. Youdim and M. Sandler. *FEBS Letters* 1(4):215-218, 1968.

6820 REGULATION OF PRODUCTION AND RELEASE OF LIPOPROTEIN BY THE PERFUSED RAT LIVER. *(E.)* Ruderman, N. B. (Harvard Med. Sch., Boston, Mass.), K. C. Richards, V. V. de Bourges and A. L. Jones. *J Lipid Res* 9(5):613-619, 1968.

6821 EFFECT OF ETHIONINE ON THE METABOLISM OF PHOSPHOLIPID IN RAT TISSUES. I. THE INCORPORATION OF ^{32}P-ORTHOPHOSPHATE INTO PHOSPHOLIPIDS OF RAT TISSUES. *(E.)* Sato, R. (Sapporo Med. Coll., Japan). *Sapporo Med J* 31(1):41-55, 1967.

6822 ON THE ETHIONINE-INDUCED FATTY LIVER IN MALE AND FEMALE RATS. *(E.)* Schlunk, F. F. (U. Pittsburgh Sch. Med., Pa.) and B. Lombardi. *Lab Invest* 17(3):299-307, 1967.

6823 CYTOPLASMIC CHANGES IN RAT LIVER AFTER PROLONGED TREATMENT WITH LOW DOSES OF ETHIONINE AND ADENINE: AN ULTRASTRUCTURAL AND BIOCHEMICAL STUDY. *(E.)* Meldolesi, J. (Inst. Pharmacol. U. Milan, Italy), F. Clementi, E. Chiesara, F. Conti and A. Fanti. *Lab Invest* 17(3):265-275, 1967.

6824 CHOLINE DEFICIENCY FATTY LIVER. *(E.)* Lombardi, B. (U. Pittsburgh Sch. Med., Pa.) and A. Oler. *Lab Invest* 17(3):308-321, 1967.

See also: 6692,6770,7419,7428,7432,7437,7439

6825 **THE METABOLISM OF DRUGS AND OTHER ORGANIC COMPOUNDS BY THE INTESTINAL MICROFLORA.** *(E.)* Scheline, R. R. (Dept. Pharmacol., U. Bergen, Norway). *Acta Pharmacol (Kobenhavn)* 26(4):332-342, 1968.

A number of drugs and other organic compounds were incubated anaerobically and aerobically with rat cecal extracts in a medium containing glucose, yeast extract, and peptone in pH 7.4 phosphate buffer in order to study the metabolism of these substances by the intestinal microflora. After incubation, the samples were extracted with ether and examined by thin-layer chromatography. The intestinal microflora brought about: hydrolysis of 4-methylumbelliferone glucuronide, phenol-β-D-glucoside, hesperidin, rutin, p-aminohippuric acid and methyl gallate; reduction of methyl red; dehydroxylation of xanthurenic acid and pyrogallol; ring fission of (+)-catechol, hesperetin and quercetin; and aromatization of shikimic acid and quinic acid. No hydrolysis of p-nitrocatechol sulfate or N^4-acetylsulfanilamide was observed and no metabolites were detected in the incubation mixtures which contained cyanidin chloride.

6826 **METABOLIC EFFECTS OF GLUCOSE INFUSION IN MAN.** *(E.)* Svedmyr, N. (Dept. Pharmacol., U. Gothenburg, Sweden). *Acta Physiol Scand* 73(4): 407-410, 1968.

The possible importance of the hyperglycemic effect of epinephrine for its calorigenic action was studied in experiments on 5 healthy adult males. When glucose was infused i.v. at 5 mg/kg/min for 30 min (total dose 22 g), the concentration of glucose in the blood increased slightly more (78 ± 4.2 mg/100 ml) than after an infusion of epinephrine at a dose of 0.10 μg/kg/min for 30 min (54 ± 4.3 mg/100 ml). The oxygen consumption was not stimulated during or after the glucose infusion, but the CO_2 elimination and respiratory quotient increased. The plasma free fatty acid content decreased but the lactate concentration was not altered. It is suggested that the hyperglycemic effect of epinephrine is of no importance for its calorigenic action.

6827 **EFFECT OF DIETARY FATS ON CHOLESTEROL ESTERASE ACTIVITY IN THE ALBINO RAT.** *(E.)* Divakaran, P. (Dept. Nutrit., Haffkine Inst., Bombay, India) and A. Venkataraman. *Indian J Exp Biol* 6(1): 15-18, 1967.

The effect of feeding various dietary fats (vanaspati, coconut oil, safflower oil, peanut oil), constituting 20% of the diet, for 6 weeks on the cholesterol esterifying activity in plasma, liver, intestine, and pancreas was studied in young male albino rats. The absolute amount of "newly esterified" cholesterol in the plasma of the vanaspati (hydrogenated vegetable fat) and coconut-oil fed groups was significantly elevated (54% and 50%, resp.) as compared to the groups fed safflower oil and peanut oil (17% and 25%, resp.), indicating increased cholesterol esterification *in vitro;* however, the cholesterol esterifying activity in the liver was decreased as compared to the safflower and peanut oil groups. In the intestine and pancreas, cholesterol was preferentially esterified with unsaturated rather than saturated fatty acids. The absolute values for cholesterol esterase activity in the pancreas were much higher than in the intestine (ratio of 10:1). It is concluded that coconut oil, with its high content of saturated fatty acids, and the commercially prepared hydrogenated vegetable fat (vanaspati) behave similarly as far as the plasma and liver esterase activities are concerned. Some of the other differences observed need further study.

6828 **BEHAVIOR OF THE pH IN VARIOUS SEGMENTS OF THE SMALL AND LARGE INTESTIN CONTINUOUS RECORDING BY MEANS OF AN ENDORADIOSONDE.** *(It.)* Arullani, P. (Inst. Med. Path., U. Rome, Italy), R. Caprilli and G. Spaziani. *Arch Ital Mal Appar Dig* 34(3):279-289, 1967.

6829 **COMPARATIVE STUDIES OF BACTERICIDAL ACTIVITY WITH SIX DIFFERENT ENTEROBACTERIACEAE.** *(Ger.)* Urbach, H. (Inst. Hyg., Friedrich-Schiller U., Jena, Germany), G. Schabinski and A. Stelzner. *Arch Hyg Bakt* 151(7):630-640, 1967.

6830 **INFECTIVE MULTIPLE RESISTANCE AMONG THE ENTEROBACTERIA.** *(Ser.)* Zajc-Satler, J. (Fac. Med., Ljubljana, Yugoslavia) and S. Banič. *Lijecn Vjesn* 90(3):237-245, 1968.

6831 **FORMATION OF EXOTOXIN BY *ESCHERICHIA COLI* UNDER VARIOUS CONDITIONS OF INCUBATION.** *(Rus.)* Smirnova, V. I. (Mechnikov Inst. Virol. Epidem., Odessa, USSR). *Zh Mikrobiol* 45(7):48-50, 1968.

6832 **THE ANTIBIOGRAM OF THE TYPHOID FEVER BACTERIA. IV. A COMPARATIVE STUDY OF THE SENSITIVITY OF *SALMONELLA TYPHI* TO VARIOUS ANTIBIOTICS.** *(Rus.)* Kokhanovskaia, T. M. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Zh Mikrobiol* 45(7):42-44, 1968.

6833 **THE SUBSTRATE BINDING SITE OF PEPSIN.** *(E.)* Humphreys, R. E. (Yale U., New Haven, Conn.) and J. S. Fruton. *Proc Nat Acad Sci USA* 59(2):519-525, 1968.

6834 **VAGOTOMY AND MESENTERIC BLOOD FLOW IN THE DOG.** *(E.)* Scheinin, T. M. (Dept. Surg., U. Turku, Finland) and M. V. Inberg. *Ann Med Exp Fenn* 45(4):381-383, 1967.

6835 **PHYSIOLOGICAL BASIS OF GAS EXCHANGE IN THE GUT.** *(E.)* Forster, R. E. (U. Pennsylvai Med. Sch., Philadelphia). *Ann NY Acad Sci* 150(Art.1):4-12, 1968.

DIAGNOSTIC PROCEDURES

6836 USE OF RAFFINOSE LOADING IN THE DIAGNOSIS OF MALABSORPTION. *(Ger.)*

ust, L. (Inst. Nutrit., Potsdam-Rehbrucke, Germany), K. etter and M. Borchardt. *Deutsch Gesundh* 22(45):2150-2152, 967.

ogether with 500-750 ml of tea, 4 g raffinose were given p.o. o 10 normal subjects and 20 patients with malabsorption syn- lromes due to gastrectomy, ileocecal resection, or sprue. After hr, normal subjects had excreted 0.3-0.8% of the raffinose n their urine and after 24 hr, 0.7-1.3%. The 20 patients were ivided into 3 groups on the basis of small intestine biopsies:) 10 patients with no mucosal inflammation or atrophy, 2) with slight mucosal inflammation, and 3) 6 with severe mu- osal inflammation and, in some cases, atrophy. A comparison f the results of xylose and raffinose tolerance tests showed hat some results were obtained from patients in groups (1) nd (2) which did not agree with the histological findings. Raf- inose absorption was increased for all 6 patients in group (3) vhile xylose absorption was increased in only 2 patients. These indings indicate that the raffinose tolerance test is a truer re- lection of the state of the small intestine since its absorption epends only on the permeability of the intestinal wall.

6837 DIAGNOSIS OF LIVER METASTASES BY ULTRASOUND. *(Ger.)*

Schentke, K. U. (2nd led. Clin., U. Berlin, Germany), K. Henkert and F. Renger. *eutsch Gesundh* 22(45):2113-2116, 1967.

Jltrasound hepatograms were obtained for 167 patients sus- ected of having liver metastases. Results were positive in 52 ases and negative in 115. An analysis of these results from 129 atients in which the diagnosis was anatomically or histologically onfirmed showed that the results of the ultrasound hepatogram vere correct in 29 of 33 positive cases, the other 4 being false ositives. In 105 patients in whom liver metastases had been lefinitely or probably ruled out by other methods, the ultra- ound hepatogram agreed in 86 cases and gave false-positive re- ults in 10. However, in 8 of these 10 cases there were tumors n the immediate vicinity of the liver which could not be con- idered extrahepatic.

6838 PARTIAL STUDY OF MEGAKARYOCYTES FROM THE BONE MARROW OF PATIENTS VITH ESOPHAGEAL AND GASTRIC CANCER. *(Rus.)*

nikin, B. S. (Kuibyshev Med. Inst., USSR). *Lab Delo* (1):11- 3, 1968.

legakaryocytes were studied in bone marrow specimens from 1 cancer patients (32 men and 19 women, aged 27-75 yr old); 5 had esophageal cancer and 36 had stomach cancer. Changes, vhich were especially evident in the late stages of cancer, were een in all forms of megakaryocytes. There was a significant eduction in both the most functionally active mature forms nd in forms from which blood platelets originate, with a com- ensatory increase in the number of juvenile megakaryoplasts. decrease also occurred in the number of intermediate forms, he promegakaryocytes, which was more marked in stomach ancer. This reduction in the more mature cell forms was due o involution and degenerative cell changes. Involution was articularly evident in the early stages while degeneration was more marked in the later stages. Degenerative changes were seen at all stages of cell development, but were particularly frequent among promegakaryocytes and mature megakaryocytes. Examination of megakaryocytes is recommended for the early diagnosis of cancer.

6839 COMPARISON BETWEEN THE WOFAVERDIN AND BSP TESTS AS LIVER FUNCTION TESTS. *(Ger.)*

Renger, F. (Carl Gustav Carus Med. Acad., Dresden, Germany), B. Richter and W. Rose. *Deutsch Gesundh* 22(43): 2035-2040, 1967.

A comparison was made of the rate at which indocyanin green, in the form of Wofaverdin, and BSP disappeared from the blood after i.v. injection. With a dose of 0.3 mg/kg Wofaverdin the half-time averaged 3 min, 2 sec ± 22 sec in 20 subjects with no histories of liver, biliary, or cardiovascular disease. The same type of two-stage curve was obtained with Wofaverdin, the hydroiodide of indocyanin green in 1,2-propanol, and Cardiogreen, the lyophilized powder. Both curves deviated somewhat from the theoretical for a 1st-order reaction. BSP gave a one-stage curve. Simultaneous tests were then run on 54 patients with a variety of liver and biliary diseases by simultaneous administration of 0.3 mg/kg Wofaverdin and 5 mg/kg BSP i.v. Results from these 2 methods were shown to be equivalent by analysis of variance.

6840 HEMODYNAMIC AND RADIOLOGICAL EVALUATION OF PATIENTS WITH HEPATIC OR PANCREATIC DISEASE BY COMBINED UMBILICO-PORTAL AND SYSTEMIC VENOUS CATHETERIZATION. *(E.)*

Joly, J. G. (Saint-Luc Hosp., Montreal, P.Q., Canada), J. Bernier, P. Lavoie, A. Legare and A. Viallet. *Canad Med Ass J* 98(1):16-24, 1968.

Nineteen cirrhotic patients (18 males and 1 female, 34-58 yr old) and 10 noncirrhotic patients (8 males and 2 females, 26-68 yr old, including 4 with alcoholic hepatitis and 2 with chronic pancreatitis) were evaluated by combined portal-hepatic venous catheterization. Among the 19 cirrhotic patients, a complete hepatoportogram was obtained in 12, a hepatogram with partial portogram in 2, a portogram and partial hepatogram in 1, and a hepatogram only in 4; a complete hepatoportogram was obtained in 8 of the 10 noncirrhotic patients. Esophageal varices were diagnosed in 6 patients by barium X-ray and in 12 patients by portohepatography. Phlebography revealed stenosis in the subdiaphragmatic region of the inferior vena cava in 5 cirrhotics. In the cirrhotic and noncirrhotic groups, resp., average wedged hepatic venous pressures (WHVP) were 23.1 ± 5.7 mm Hg and 12.4 ± 4.9 mm Hg, while average free hepatic venous pressures (FPVP) were 22.5 ± 6.6 mm Hg and 11.9 ± 4.4 mm Hg. Although both WHVP and FPVP were significantly correlated [$r = 0.94206$ ($p < .001$) and $r = 0.95287$ ($p < .001$), resp.] the WHVP-FHVP gradient of the cirrhotic group was significantly higher than that of the noncirrhotic group: $t = 6.37242$ ($p < .001$). Earlier literature regarding the diagnosis of portal hypertension is discussed, and the radiological and physiological superiority of the described method is emphasized.

DIAGNOSTIC PROCEDURES

6841 **STUDIES ON UROPEPSIN EXCRETION, MAXIMAL AND BASAL ACID OUTPUT IN RECURRENT PEPTIC ULCER.** *(E.)* Hartkopp, O. (State Hosp., Copenhagen, Denmark) and P. Aagaard. *Gastroenterologia* 107(6):344-353, 1967.

The diagnostic value of testing the uropepsin excretion, basal acid output, and maximal acid output following histamine was investigated in 20 patients (15 males and 5 females) with and 22 patients (15 males and 7 females) without recurrent peptic ulcers following gastrectomy. Determination of the 24-hr uropepsin excretion was found to be of definite value in diagnosing recurrent peptic ulcer, since values exceeding 150 U/24 hr were found in 65% of the ulcer group as compared with only 9% of the control group. Values in excess of 200 U/24 hr are strongly suggestive of recurrent ulcer, while values below 70 U/24 hr definitely indicate that it is not present. Although determination of the basal acid output is of no diagnostic value in recurrent peptic ulcer, the peak acid output may be of significance since a peak acid output greater than 10 mEq/hr was found in 50% of the ulcer group but in only 9% of the control group. Achlorhydria was not found in any case in the ulcer group. The combined evaluation of uropepsin excretion and peak acid output increases the diagnostic value, presumably because pepsin and acid production do not occur in exactly the same parts of the stomach. A peak acid output greater than 7 mEq/hr or a uropepsin value greater than 150 U/24 hr was found in 95% of the recurrence group and 23% of the control group.

6842 **A COMPARATIVE STUDY OF ARTERIOGRAPHY AND SCINTILLATION SCANNING IN SPACE-OCCUPYING LESIONS OF THE LIVER.** *(E.)* Kreel, L. (Royal Free Hosp., London, England), E. A. Jones and A. S. Tavill. *Brit J Radiol* 41(486):401-411, 1968.

The results of scintillation scanning (using colloidal ^{198}Au, ^{99m}Tc or ^{131}I-labeled BSP) and of selective celiac axis arteriography (using Conray 420 and Hypaque 45%) were directly compared in 23 patients with space-occupying lesions of the liver. These patients were among 33 with space-occupying lesions detected among 160 examined by selective arteriography and 38 with space-occupying lesions among 320 examined by hepatic scintigraphy. Lesions included primary hepatoma, metastatic liver cancer, and liver cysts. Ten illustrative cases are presented in detail. Based on this experience, it is concluded that colloidal gold or technetium sulfide are superior to radioiodinated BSP or Rose Bengal in the detection of space-occupying hepatic lesions. Scintiscanning in general, however, is superior to arteriography in the detection of such lesions, since arteriography can only reveal displacement of vessels. On the other hand, arteriography may make possible the distinction between cysts and malignant tumors, which is generally impossible on the scintiscan. The smallest space-occupying lesion detectable on a hepatic scintiscan is about 2.5 cm in diameter, although larger lesions may be missed. As a result, some highly vascular tumor nodules or cirrhotic nodules may be more easily detected by arteriography. Correlations of the findings obtained by both techniques is essential to a correct interpretation of the nature of most space-occupying lesions, since the differential diagnosis between benign and malignant conditions is extremely difficult without biopsy. Some of the specific findings in the various types of hepatic disease are described.

6843 **AN EVALUATION OF 600 EXAMINATIONS WITH THE GASTRODUODENAL FIBERSCOP** *(Sl.)* Dvorsky, A. (Res. Inst. Human Nutrit., Bratislava, Czechoslovakia). *Cesk Gastroent Vyz* 21(6):360-368, 1967.

The results of 600 fiberscope studies performed in 459 patien (319 males and 140 females) during a 10-month period were compared with the results of X-ray examination, and in 83 ca the diagnostic value of the fiberscope was compared with that of the conventional semiflexible gastroscope. Among the 459 patients, 111 had normal X-rays and in 33 no X-ray was take other radiological findings included duodenal ulcer (65 cases), deformation of the duodenal bulb (56), gastric ulcer (58), pyloric ulcer, stenosis or diverticula (6); achalasia (2), gastric polyps (17), gastric neoplasms (50), and a number of patients with previous gastrointestinal surgery. Fiberscopic findings w normal in 206 cases, while in 5 the fiberscope could not be i troduced into the stomach; other fiberscopic findings include duodenal ulcer (22 cases), duodenitis (5), pyloric or antral inflammation or erosion (33), gastric ulcers (44), gastric neopla (51), gastric polyps (19), mucosal bleeding (7), atrophic gastr (29), and inflammation of the anastomosis or jejunum follow surgery (46). The semiflexible gastroscope detected only 75% of the lesions detected by the fiberscope, which was particula valuable for the examination of older children and for the dia nosis of upper gastrointestinal bleeding. This instrument elim nates blind spots in the lesser curvature of the antrum near th pylorus, but not in the fundic region below the cardia. The cases of duodenal ulcer detected (17% of 124 cases) included 3 with negative X-ray findings and 4 in which no X-ray was done. However, some difficulty was encountered in inserting the instrument into the duodenal bulb. There was only one serious complication during 230 examinations, this being non fatal cardiac arrest.

6844 **ABSORPTION OF ^{58}Co-LABELED VITAMIN B_{12} IN DISEASES OF THE SMALL INTESTIN** *(Cz.)* Kojecky, Z. (Fac. Med., Purkinje U., Olomouc, Czechoslovakia), J. Blatny and M. Wiedermann. *Cesk Gastroe Vyz* 21(8):508-513, 1967.

Schilling's test (absorption and urinary excretion of ^{58}Co-labe vitamin B_{12} following oral administration) was given to 52 patients with various diseases of the small intestine and to 30 controls. A significant decrease in vitamin B_{12} absorption wa noted in 14 patients with resection of more than 50 cm of th terminal ileum, but not in patients with resection of less than 50 cm of terminal ileum or patients with previous resection o the jejunum. Absorption was also decreased in 6 patients wit the blind-loop syndrome, 5 with regional enteritis, and 6 with primary malabsorption, although in the latter condition there was poor correlation between the results of the Schilling test and other findings, such as fecal fat excretion or the results of biopsy. Schilling's test was very useful for the differential diagnosis of pernicious anemia and megaloblastic anemia of enteric origin.

6845 **PAPER ELECTROPHORESIS AND FLOCCULATION TESTS OF THE SERUM PROTEINS IN NORMAL NEGROES.** *(Por.)* Martinez, J. O. (Inst. Gastroent São Paulo, Brazil), L. R. Trabulsi, W. P. Rothstein and L. C. d Silva. *Arq Gastroent* 4(3):207-213, 1967.

The plasma proteins were studied by means of paper electrophoresis, chemical titration, and flocculation tests (cephalin-cholesterol flocculation, thymol turbidity, thymol flocculation, zinc or ammonium sulfate turbidity, and Lugol's precipitation) in 37 normal male Brazilian Negroes, 19-21 yr old, with no history of hepatobiliary disease, dietary deficiencies, hepatosplenomegaly or abnormalities in the blood or urine picture or chest X-ray. Paper electrophoresis yielded average values of 7.99 ± 0.44, 4.23 ± 0.31, 0.41 ± 0.08, 0.67 ± 0.11, 0.88 ± 0.12 and 1.77 ± 0.21 g% for total protein, albumin, and α_1-, α_2-, β- and γ-globulin, resp. The average percentage of γ-globulin was thus 22.45 (range 18.12-28.62), which is significantly higher than in normal Caucasians. There was no correlation between the results of paper electrophoresis and those of chemical protein determinations (biuret method), which paralleled the results in normal Caucasians (5.77 ± 0.36 g% albumin and 2.23 ± 0.33 g% globulins). The results of the "liver function tests" (flocculation tests), however, were abnormally high, especially those affected by the globulin level. It is therefore recommended that liver function in Negroes be evaluated by techniques which do not depend on the alterations in plasma proteins.

6846 STUDIES ON GASTROPHOTOGRAPHY IN VARIOUS POSITIONS. REPORT 3. GASTROPHOTOGRAPHY IN THE RESECTED STOMACH. *(Jap.)*

Miyake, T. (Tenri Hosp., Nara, Japan), Y. Yamamoto, T. Suzaki, I. Ariyoshi, Y. Yagiri, K. Hajiro, A. Miyoshi and M. Watanabe. *Jap Arch Intern Med* 14(11/12):309-316, 1967.

In 32 patients who had previous gastrectomies (Billroth I or II), serial photographs of the stomach were taken with a gastrocamera V in 2 stages: 1) the camera was aimed at the stoma at a depth of 50 cm with the angle down, at 3 orientations with respect to the lesser curvature (10:30, 12:00, and 1:30), and in 6 different patient positions—supine, prone, right and left lateral, and right and left oblique prone; 2) 8 panoramic views in the supine position were taken at maximal depth with the angle up. The percentage of pictures satisfactory for evaluation was calculated for each position and camera direction for patients subjected to Billroth I and II, resp. The results in the 1st series were: left oblique prone, 72% and 48%; left lateral, 66% and 40%; supine, 54% and 28%; prone, 50% and 24%; and right lateral and oblique prone, less than 10%. The most satisfactory orientation was at 10:30, the least at 1:30. Satisfactory pictures were obtained in 65% of cases with a cardia-stoma length of 6-11 cm and in 21% when the length was 3-4 cm. In the 2nd series, satisfactory pictures were obtained in 70-89% and the stoma was visualized satisfactorily in 5-21%. The left oblique prone, left lateral, supine, and prone positions are recommended in gastrophotography of the resected stomach. The difference between patients with a Billroth I or II gastrectomy is ascribed to the length of the gastric stump.

6847 CINE-ESOPHAGOSCOPY AS AN AID IN THE DIAGNOSIS OF PEPTIC ESOPHAGITIS. *(E.)*

Katz, D. (New York Med. Coll. , N. Y.). *Gastroint Endosc* 14(3):162-165, 1968.

Esophageal examination by means of (a) a 4-power proximal lens and standard incandescent bulb lighting (b) (Eder-Hufford esophagoscope), a telescopic obdurator with fiberannular lighting from an external light source (ACMI telescope), and (c) cinematography through the telescopic obdurator were compared in 16 patients with hiatus hernia. The telescopic obdurator and fiberannular lighting were superior to the proximal telescope and standard incandescent bulb revealing 10 cases of esophagitis compared to only 7. Cinematography through the telescopic obdurator documented these findings well and revealed 3 additional mild cases. These results are ascribed to the increased intensity of light through the telescopic obdurator, the increased magnification of the telescopic obdurator, and the increased sensitivity of Ektachrome EF film to the color red.

6848 A CONTROLLED COMPARISON OF RIGID AND FIBER OPTIC ESOPHAGOSCOPY. *(E.)*

Zimmon, D. S. (VA Hosp., New York, N. Y.) and M. A. Tesler. *Gastroint Endosc* 14(4):220-221, 1968.

In 51 male patients with suspected or proven hepatic cirrhosis, esophagoscopies were performed by two experienced endoscopists using both the Eder-Hufford Rigid Esophagoscope and the American Cystoscope Makers, Inc. Foroblique Fiber Optic Esophagoscope for the evaluation of esophageal varices. Fiber optic esophagoscopy proved to be technically easy, was preferred by the patient, and gave an excellent view of esophageal mucosa, cardioesophageal junction and the gastric, cardia and fundus. However, the rigid esophagoscope was distinctly superior for the identification of esophageal varices. Although there was close agreement between both endoscopists using both instruments in 33 cases, in 14% of cases in which rigid esophagoscopy demonstrated esophageal varices they were overlooked when the fiber optic instrument was used.

6849 FIBEROPTIC GASTROSCOPY IN BENIGN PYLORO-DUODENAL STENOSIS. *(E.)*

Stempien, S. J. (VA Hosp., Long Beach, Calif.), J. J. Madigan and A. E. Dagradi. *Gastroint Endosc* 14(4):194-196, 1968.

Results of fiberoptic gastroscopy in 50 patients suspected of having outlet stenosis were compared with X-ray and surgical findings. The pyloric opening was well visualized in 44 cases by the methods used, the 6 failures being due to displacement posteriorly and superiorly or crossing of a distal antral fold proximal to the pylorus. Of these 44 cases, 28 showed pyloric stenosis. There was 72% agreement between the results of gastroscopy and X-ray, which showed varying degrees of barium retention in 43 cases and pyloric or pyloroduodenal stenosis in 35. X-ray studies tended to give more total information, but gastroscopy gave more information on the exact location of the stenosis. There was a high degree of correlation (84%) between endoscopic and surgical findings in the 29 patients who came to surgery. Associated lesions in these 50 patients included only 3 cases of gastric ulcer, but 16 of hypertrophic hypersecretory gastritis and 14 of atrophic gastritis; 32-36% also had antral gastritis and/or pyloritis. Gastroscopy is highly recommended in all cases of outlet stenosis, both to identify and localize the causative lesion and to assess the results of therapy.

6850 THE RELATIVE VALUE OF THE OLYMPUS MODEL GT-5 GASTROCAMERA AND OLYMPUS MODEL GT-F GASTROCAMERA FIBERSCOPE. *(E.)*

Morrissey, J. F. (Dept. Med., U. Hosp., Madison, Wis.), Y. Tanaka and W. B. Thorsen. *Gastroint Endosc* 14(4):197-200, 1968.

A comparative study of the gastrocamera and gastrocamera fiberscope was performed in 267 patients, 46 of whom had 2 areas of interest, resulting in a total of 313 comparisons. Only 11 of these 313 areas were unsatisfactorily visualized, including 7 with an X-ray diagnosis of ulcer and one carcinoma, showing that both are effective instruments for examining the stomach. Among 87 patients with gastric ulcer, the gastrocamera provided better visualization in 10 and the fiberscope was better in 26, a statistically significant difference in favor of the fiberscope. This instrument was also superior for examining the distal antrum and pylorus, while the gastrocamera was superior for examining the high body of the stomach. In many cases, there was an advantage to the use of both instruments. Thus, 12% of patients examined by the fiberscope and 17% of those examined by the gastrocamera benefitted from an additional examination using the other instrument. Among 141 X-ray-negative patients, endoscopy revealed 59 lesions (including 24 ulcers and 5 carcinomas), 45 being revealed by the gastrocamera and 44 by the fiberscope.

6851 LACTOSE LOADING: A SIMPLE TEST FOR DETECTING INTESTINAL LACTASE. EVALUATION OF DIFFERENT METHODS.

(E.) Desai, H. G. (Tata Mem. Hosp., Parel, Bombay, India), A. V. Chitre and K. N. Jeejeebhoy. *Gastroenterologia* 108(4):177-188, 1967.

Comparison with established techniques for measuring lactase deficiency (lactose tolerance test, peroral jejunal biopsy analysis) showed that the oral lactose loading test is a simple and reliable method for estimating intestinal lactase. The comparisons were made in 57 adult patients (17-70 yr old) with tropical sprue (41), gastroenteritis (7), giardiasis (5), and Polya-gastrectomy (4), and in 18 normal controls (18-50 yr old). The test involves determination of the minimum quantity of oral lactose causing 1 or more liquid stools within 4 hr, by administration of doses increasing or decreasing daily in the amount of 10 g from an initial starting dose of 50 g. Tolerance to 40 g or less is considered a sign of lactase deficiency. The correlation between the maximum rise in blood sugar during the lactose tolerance test and the minimum amount of lactose causing liquid stools was 0.70 ($p = 0.0005$), compared to only 0.5 ($p = 0.005$) between the latter value and the lactase activity in a single jejunal biopsy specimen. False positives for lactase deficiency are more common with both jejunal biopsy and lactose tolerance than with oral lactose loading.

6852 SERUM ALKALINE PHOSPHATASE LEVELS IN INFECTIVE HEPATITIS.

(E.) Knox, J. D. E. (Ruchill Hosp., Glasgow, Scotland) and D. Reid. *Scot Med J* 13(6):202-205, 1968.

The serum alkaline phosphatase levels were determined in 222 patients (116 males and 106 females, 2-60+ yr old; 2/3 less than 15 yr old) with infectious hepatitis; 5'-nucleotidase levels were also determined in 24 of these patients. Patients were divided into 3 grades of clinical severity: 115 being mildly, 95 moderately, and 12 severely ill. Alkaline phosphatase levels in patients less than 15 yr old were significantly higher than in older patients. The correlation between alkaline phosphatase levels and patient age persisted regardless of the different grades of severity of illness. In 24 mildly ill patients, serum 5'-nucleotidase levels showed little correlation with alkaline phosphatase levels in those less than 15 yr old; in older patients, however, a statistically significant correlation between the 2 enzymes was noted. It is concluded that, in infectious hepatitis, the serum alkaline phosphatase shows a better correlation with the age of the patient than with the severity of the illness. Nevertheless, the test can be of diagnostic importance, particularly in children and particularly when combined with determinations of 5'-nucleotidase levels.

6853 ENZYME STUDIES AND CLINICAL OBSERVATIONS AFTER INFUSION CHOLANGIOGRAPHY

(Ger.) Zastrow, R. (Med. Clin., U. Rostock, Germany), R. Arendt and H. J. Hütter. *Deutsch Z Verdau Stoffwechselkr* 28(1):37-51, 1968.

Serum GPT, GOT, sorbitol dehydrogenase, glutamic dehydrogenase, and α-amylase activities were determined on 31 patients before and at various intervals after infusion cholangiography. Serum transaminase levels were elevated in all 12 patients who were given 60 ml adipiodone in 250 ml of 5% glucose or 0.9% NaCl and 5 ml of 0.4% codeine syrup p.o. Severe subjective signs of intolerance, mostly gastrointestinal in nature, were seen in 7 of these patients and mild to moderate reactions in 5 others. Sorbitol dehydrogenase and glutamic dehydrogenase levels were elevated in 11 and 7 cases, resp. Subjective symptoms developed in the 10 patients with the highest serum enzyme activities. Serum amylase activities were elevated in 2 patients, 1 of whom had a history of chronic recurrent pancreatitis. Of the 19 patients given adipiodone alone, 6 had severe symptoms of intolerance and 6 had mild to moderate symptoms. All patients in this group had elevated liver enzyme activities at least once during the observation period, but simultaneous increases in all enzyme activities occurred in fewer cases than among those given both adipiodone and codeine. Gastrointestinal symptoms developed after 10 min to 5 hr in 5 of 11 controls given codeine alone. SGOT and SGPT levels were elevated in 4 cases each and serum sorbitol dehydrogenase and glutamic dehydrogenase levels in 2 cases each. Codeine produced increases in alkaline phosphatase activities in 5 of these 11 patients. These findings suggest that infusion cholangiography should be used with caution in patients with any form of pancreatitis and that codeine must be avoided in patients recovering from acute pancreatitis or in those suspected of having papillary or prepapillary obstructions of the biliary tract.

6854 THE PRACTICAL VALUE OF SOME BIOCHEMICAL TESTS IN THE DIAGNOSIS OF VIRAL HEPATITIS IN CHILDREN.

(Rus.) Burnatseva, V. V. *Pediatriia* 47(1):19-23, 1968.

Clinical and laboratory studies were made of 126 children, aged 1-14 yr, with viral hepatitis and 14 controls with other diseases who were initially suspected of having viral hepatitis. Of those with viral hepatitis, 5 had severe forms, 53 moderately severe forms, 53 mild forms, and 13 anicteric forms. Associated diseases included chronic tonsillitis in 25 cases, *Giardia lamblia* cholecystitis in 7, ascariasis in 10, acute respiratory diseases in 5, and chronic pneumonia in 4. The most sensitive biochemical test run on these children was Jirgl's test which was positive in 119 patients (94.4%), including 10 of 13 with anicteric hepatitis. The seromucoid concentration was reduced in 72.5%, elevated in 17.1%, and within normal limits in 10.4%. Aldolase was elevated in 65%. The calomel test was positive in only 45% and the cholesterol test in 30.8%. Jirgl's test was most markedly ositive and seromucoid concentrations reached a maximum later the course of the disease and remained abnormal longer than d bilirubin and aldolase values. All of these biochemical tests ere negative in the controls except for Jirgl's test, which was ositive in 1 child with infectious mononucleosis. Jirgl's test recommended for the early diagnosis of viral hepatitis in ildren.

855 **NEW SERUM ENZYME TESTS FOR CHILDREN WITH CHRONIC LIVER DISEASES.** *(Rus.)* ordeieva, G. F. (Inst. Pediat., Moscow, USSR), Iu. A. Iurkov nd L. V. Chistova. *Pediatriia* 47(2):34-37, 1968.

otal serum lactic dehydrogenase (LDH) and LDH isoenzyme vels were measured on 55 children, aged 3-15 yr (36 with rrhosis, 6 with fatty dystrophy and 13 with chronic hepatitis). otal LDH and LDH$_5$ levels were elevated in 24 children with ymptoms of severe cirrhosis and, to a lesser extent, in 12 with itial symptoms of cirrhosis. LDH$_4$ levels were also increased, ut not as much. All these values returned to normal as the atient's clinical condition improved. A simultaneous decrease ccurred in serum transaminase values. Increases in total LDH nd LDH$_5$ levels were also seen in patients with fatty dystrophy, ut these changes persisted even after treatment. Serum transminase values remained unchanged in these patients. Slight creases in total LDH activity and in LDH$_3$, LDH$_4$, and LDH$_5$ evels were found in patients with chronic hepatitis. The LDH pectrum is concluded to be a sensitive index of the degree of ver damage in children.

856 **COMPARATIVE EVALUATION OF SOME METHODS FOR DETECTING TYPHOID AND ARATYPHOID CARRIERS.** *(Rus.)* Litinskii, Iu. I. (Cent. ci.-Res. Inst. Epidemiol., Min. Hlth., Moscow, USSR), A. V. asinskii, L. S. Koretskaia, M. A. Smirnova-Mutusheva and B. A. odovannyi. *Lab Delo* (5):299-304, 1968.

omparisons were made of the number of *Salmonella typhosa* nd *S. paratyphi* A and B strains isolated on various solid and iquid culture media from 1865 apparently healthy workers in he food industry and from a group of known carriers (43 with *typhosa*, 10 with *S. paratyphi* A, and 16 with *S. paratyphi* B). Stool specimens were collected with a glass rectal tube. n addition, specimens were collected from some subjects after hey had taken laxatives. Specimens from apparently healthy ubjects were inoculated into Ploskirev's and Levin's solid media nd into Müller's concentrating medium followed by transfers nto bismuth sulfite. No statistically significant differences were ound between the number of isolations on these media, but here were few carriers among this group. When specimens rom known carriers were inoculated into Ploskirev's and Levin's nedia and into selenite medium followed by transfers into loskirev's and Levin's media, a significantly larger number of ositive cultures was obtained by the last method. No differences were noted between stool specimens obtained with the se of laxatives and those which were not. However, 5 of 40 *paratyphi* A strains grew only when inoculated directly into olid media. It is concluded that both solid and liquid media hould be used in the detection of *S. typhosa* and *S. paratyphi* arriers.

6857 **DUODENOGRAPHY (WITHOUT HYPOTONIA) AND ITS ROLE IN THE DIAGNOSIS OF DISEASES OF THE LIVER, PANCREAS, AND DUODENUM.** *(Rus.)* Slobozhankin, A. D. (S. M. Kirov Acad. Milit. Med., Leningrad, USSR), K. G. Magrilova and A. I. Iutin. *Vestn Khir Grekov* 100(6):26-30, 1968.

In 470 patients, radiological examinations were performed by inserting a tube into the duodenum through which a BaSO$_4$ suspension was injected with a syringe. After removal of the tube, the diameter and motor function of the duodenum were studied together with the passage of contrast medium. In patients with duodenal stasis the diameter of the descending part of the duodenum, which is normally 2-3 cm, was increased to 3-10 cm. In a few cases of spastic duodenal stasis the diameter of the duodenum was 1-1.5 cm. Evacuation of contrast medium was delayed from 2.5 to 40 min. Slow pendular movements, very slow evacuation of contrast medium into the jejunum, and antiperistalsis and gastroduodenal reflux were seen in all cases. Possible infection ascending from the duodenum into the extrahepatic biliary tract can be diagnosed by the presence of reflux into the common bile duct. Reflux into the pancreatic duct was found in 1 case. Reflux into the common bile duct and occasionally into the extrahepatic biliary tract was seen in patients who had undergone choledochoduodenostomies, cholecystoduodenostomies, sphincteropapillotomies, and suprapapillary choledochoduodenostomies.

6858 **EVALUATION OF TUBE AND TUBELESS METHODS FOR STUDYING THE ACID SECRETORY FUNCTION OF THE STOMACH IN CHILDREN.** *(Rus.)* Umanskaia, E. M. (Children's Hosp. No. 2, Tallin, USSR) and S. Sh. Umanskii. *Pediatriia* 47(6):11-14, 1968.

Gastric acid secretion after ingestion of fresh cabbage juice was studied in 107 children (58 girls and 49 boys, aged 4-15 yr). These children included 56 normal controls and 51 with a variety of gastrointestinal disorders. In all cases, acid secretion was determined by using the ion-exchange resin KB-4-2p with a quinine indicator. In addition, 49 of the children were subjected to gastric intubation and fractional study of the stomach contents. With the ion-exchange method, 5 of the 56 controls were found to have achlorhydria, 13 hypochlorhydria, 40 normochlorhydria, and 3 hyperchlorhydria. There were no differences in acid secretion among the 4-6 yr-old and 7-12 yr-old age groups. Of 29 children with chronic cholecystitis, only 13 were found to have normochlorhydria, another 11 had hypochlorhydria and 5 had hyperchlorhydria. Among 16 children with chronic gastritis, 4 had hyperchlorhydria, 8 hypochlorhydria, and 4 achlorhydria. Results of the ion-exchange method agreed with those of the intubation method in 34 cases (69.4%). The intubation method failed to show the presence of free HCl in most of the 8 cases in which results did not agree completely. In another 7 cases, in which results differed completely, intubation had not been carried to completion. Uropepsin determinations, performed on all 107 children, showed distinct age variations. Abnormal values were obtained much less frequently than during studies of acid secretion.

6859 **GASTRIC LAVAGE CYTOLOGY AND BIOPSY FOR EARLY GASTRIC CANCER UNDER DIRECT VISION BY THE FIBERGASTROSCOPE.** *(E.)* Kasugai, T. (Aichi Cancer Ctr. Hosp., Nagoya, Japan). *Gastroint Endosc* 14(4):205-208, 1968.

A high degree of accuracy in the diagnosis of early stomach cancer was obtained by using gastric lavage cytology and biopsy under direct vision by the fibergastroscope. Using a fibergastroscope with an attached nasogastric tube, a diagnostic accuracy of 94.5% was obtained in 146 cases studied by gastric lavage cytology. Use of the FGS-K, FGS-C and GFC fibergastroscopes gave a 99.1% accuracy in 348 cases studied by gastric lavage cytology. Taking both groups together, gastric lavage cytology correctly identified 274 of 283 known cancers and 222 of 227 known negatives. Results in washing the lesser curvature of the antrum and the posterior wall of the upper corpus were improved by using models (FGS-C, GFC) equipped with remote control for the axis of observation and the spouting angle. Tissue-culture solution (Hanks') was markedly superior to other solutions for clinical and cytochemical studies. Gastric biopsy under direct vision gave an accurate diagnosis in 85.9% of 255 cases; the accuracy improved sharply with study of 4 or more specimens, and with samples of 1 mm or more. The dangers of false negatives, due to inadequate biopsy site, or of false positives can be minimized by combination of both methods. A diagnostic accuracy of almost 100% can be achieved by combining cytology and biopsy with endoscopic and X-ray examinations.

6860 **GASTROFIBROCINEMATOGRAPHY AND CONTROLLED STOMACH BIOPSIES: PERSONAL EXPERIENCE WITH 357 GASTROFIBROSCOPIES AND 33 CONTROLLED STOMACH BIOPSIES.** *(Fr.)* Stoichita, S. (Ctr. Gastroent., Bucharest, Romania), L. Boicesco and M. Constantinescu. *Acta Gastroent Belg* 30(11):735-740, 1967.

The results of 205 diagnoses made by gastrofibrocinematography with the Hirschowitz gastrofibroscope and hot light and 152 made with the Wolf gastrofibroscope, cold light, and fixed-focus optics are tabulated. In addition, the results of 33 controlled stomach biopsies made with the Wolf gastrofibroscope are shown and illustrative case reports presented. Gastrofibroscopy is recommended for the diagnosis of peptic ulcers and lesions too small to be detected radiologically, tumors located deep in the walls of the stomach, hemorrhagic gastritis, Mallory-Weiss syndrome, and gastric varices. Controlled stomach biopsies are useful in differentiating between benign and malignant tumors and between the different forms of gastritis.

6861 **PROPOSED MODIFICATION OF JIRGL'S TEST FOR IMPROVED DIFFERENTIATION OF JAUNDICE.** *(Fr.)* Padolecchia, N. (Inst. Med. Path., U. Bari, Italy), S. Maggi, G. Maiorano, V. Fasano and V. M. Campese. *Arch Franc Mal Appar Dig* 57(4/5):437-446, 1968.

A modification of Jirgl's test was run on serum from 28 normal controls (14 men, 14 women) and 165 patients with jaundice who had been diagnosed at surgery or by punch liver biopsies or laparoscopy. After 2 ml of serum had been treated with 5 ml of 0.1 N KOH for 45 min, 5 ml of 20% sulfosalicylic acid were added with stirring. After allowing the mixture to stand for 20 min and filtering, 1 ml of 5% phosphotungstic acid in 2 N HCl was added dropwise to 5 ml of the filtrate and the solution was centrifuged. The precipitate was dissolved in 5 ml of 10% Na_2CO_3, and 0.5 ml of a 1:3 dilution of Folin-Ciocalteau's reagent was added. After allowing the mixture to stand for 20 min mucoproteins were determined in the supernatant by spectrophotometric determination of tyrosine. Positive Jirgl reactions were obtained on 30 of 33 sera from patients with obstructive jaundice and on 26 of 105 sera from patients with hepatocellular jaundice. No positive tests were obtained on serum from 23 patients with cirrhosis or 4 with hemolytic jaundice. Mucoproteins, as measured by tyrosine values, were increased in 31 of 33 patients with obstructive jaundice, in 17 of 105 with hepatocellular jaundice, and in 3 of 23 patients with cirrhosis. The use of this test in the differential diagnosis of jaundice is discussed.

6862 **COMPARATIVE EVALUATION OF PARASITOLOGICAL EXAMINATION OF THE FECES AND SCRAPINGS FROM THE RECTAL MUCOSA IN INTESTINAL PROTOZOAN INFESTATIONS.** *(Por.)* Penteado, J. F. (Natl. Inst. Soc. Security, Guanabara, Brazil), R. R. Gonçalves, P. R. de Carvalho and N. Zacharias. *Folha Med* 56(2):239-242, 1968.

Stools from 215 patients were examined by 4 coprological techniques. In addition, rectal mucosa was obtained from 97 of these patients by curettage during routine rectosigmoidoscopy Material was examined both in the native state and after fixation in Schaudin's fluid and staining with iron hematoxylin. Negative results were obtained in 95 cases when native specimens were examined and in 97 cases when stained specimens were employed. Coprological examinations showed that 35 of the patients were infested with *Endolimax nana*, 3 with *Entamoeba histolytica*, 16 with *Entamoeba coli*, and 1 with *Iodamoeba buetschlii*. Similar values were obtained for the other 118 patients with coprological techniques. These results indicate that examination of rectal mucosa is of little diagnostic value for protozoan infestations.

6863 **THE USE OF LYOPHILIZED PANCREAS IN THE DIFFERENTIAL DIAGNOSIS OF STEATORRHEA.** *(It.)* Sategna-Guidetti, C. (Inst. Gen. Med. Clin., U. Turin, Italy), R. Bertero, A. Pera and M. de la Pierre. *Minerva Gastroent* 13(2):23-25, 1967.

The effect of lyophilized pancreas was evaluated in 16 patients with steatorrhea and azotorrhea of pancreatic and intestinal etiology. Fecal lipids and nitrogen were determined before and after 8-10 days of therapy with 2-10 g/day of lyophilized pancreas divided into 2 doses. Both subjective and objective improvements were noted in all 9 patients with primary exocrine pancreatic insufficiency (3 with mucoviscidosis, 3 with calcerous pancreatitis, and 3 with chronic pancreatitis) and in 2 patients with gastrectomy-induced steatorrhea. Eight of these 9 patients with pancreatic insufficiency had previously failed to respond to other treatment. No improvements were noted in 2 patients with celiac disease, 1 with an extensive resection of the ileum, and 2 who developed intestinal fistulas after a stomach resection The use of lyophilized pancreas is recommended as a screening test in the differential diagnosis of steatorrhea, and also as substitution therapy for pancreatic insufficiency.

6864 **THE ROENTGENOLOGICAL AND SCINTIGRAPHIC VISUALIZATION OF THE PORTAL CIRCULATION FOLLOWING INJECTION OF CONTRAST MEDIUM INTO THE UMBILICAL VEIN.** *(Ger.)* Wirbatz, W. (Inst. Cancer Res., German Acad. Sci., Berlin, Germany), B. Mateev, G. Bauke, J. Kiessling, P. Georgi, H. J. Altenbrunn and W. Richter. *Arch Geschwulstforsch* 32(1/2):126-136, 1968.

In situ casts of the liver are illustrated which demonstrate that the commonly held view as to the configuration of the umbilical vein at its entry into the portal system is incorrect. Rather than entering the main trunk or the left main branch of the portal vein, it flows into the so-called *ramus ventroflexus* or *pars umbilicus.* An umbilical catheter thus enters the left main branch of the portal vein in a countercurrent direction, which provides optimal conditions for hepatic angiography and scintigraphy. Based on a correct understanding of the relevant anatomy, catherization of the umbilical vein becomes an easy matter. Illustrative results in 2 patients with hepatic metastases from an esophageal carcinoma and an inoperable carcinoma of the cardia are presented. Based on experience with a total of 46 cases, it is concluded that, compared to the usual angiographic techniques or splenoportography, omphaloportography yields better contrast and makes possible the selective catheterization of the portal vein or its main branches. However, injury can occur at the point of entry of the umbilical vein into the portal circulation. The umbilical route is of no advantage in scintillation scanning of the liver, since the limits of this technique are set by its low resolving power. A more accurate diagnosis of disturbances in hepatic circulation can be achieved, however, by injecting isotopes directly into the portal system.

6865 USEFULNESS OF THE ENDOGENOUS SERUM BILIRUBIN CLEARANCE RATE IN THE FOLLOW-UP OF PATIENTS RECOVERING FROM VIRAL HEPATITIS.

(Pol.) Myśliwicz-Rychlik, H. (Infect. Dis. Clin., Pomeranian Acad. Med., Szczecin, Poland). *Roczn Pom Akad Med Swierczewski* 13:437-467, 1967.

The results of periodic endogenous serum bilirubin clearance (ESBC) tests were compared with those of BSP clearance and determinations of SGOT and SGPT levels in 215 patients (15-67 yr old) followed for 0.5-4 yr after successful treatment of viral hepatitis, as well as in 50 normal controls and 18 patients with clinically-diagnosed cirrhosis of the liver. The ESBC was determined by the method of Campeanu (*Munchen Med Wschr* 102:196, 1960), which involves the administration of 1 ml of 3% nicotinic acid; no side effects due to the nicotinic acid were observed. The ESBC was found to reflect the actual functional state of the liver and the dynamics of the pathological process. In the early posticteric period, the ESBC paralleled the BSP clearance and transaminase levels, with results resembling those in cirrhosis. Following the return of SGPT and SGOT to normal levels, the BSP clearance and ESBC gradually normalized as well, but any relapse was reflected by a rapid drop in ESBC. While the transaminase levels returned to normal in all 215 patients, BSP clearance returned to normal in only 80.5% and ESBC was normalized in only 74.0%. The persistence of low ESBC values, even 1.5 yr after discharge from the hospital, suggests a chronic pathological process in the posthepatitic liver. The ESBC is particularly useful in patients with a disturbance in bilirubin balance, in whom it makes possible a differentiation between disturbances in bilirubin metabolism and those in bilirubin excretion.

6866 THE EFFECT OF INTRAARTERIAL SECRETIN ON CELIAC ANGIOGRAPHY (ARTERIOGRAPHY COMBINED WITH DUODENAL INTUBATION).

(Fr.) Plessier, J., D. Doyon, J. Benett, M. Stoopen, P. Economopoulos, E. Chérigié and J. Caroli. *Arch Franc Mal Appar Dig* 57(3):307-315, 1968.

Celiac arteriograms were made on 7 biologically normal patients, 17 with benign pancreatic diseases, and 1 with a carcinomatous invasion of the pancreas. After insertion of a duodenal catheter, 75 U of secretin were injected into the celiac trunk via a catheter inserted in the femoral artery followed, 1.5 hr later, by 30-50 ml of an iodine-containing contrast medium. Arteriograms from 6 of the 7 controls showed the entire pancreas. In the remaining case, the patient had just recovered from acute pancreatitis. In patients with pancreatic disease, arterial changes were more evident than without secretin due to better filling of the blood vessels. The most consistent finding was the absence of parenchymal opacification in patients with pancreatic disease and opacification in patients with normal pancreatic secretion. The maximum lipase and bicarbonate concentrations in duodenal juice were the same, regardless of whether secretin was injected i.v. or i.a., but stimulation produced by intraarterial injections lasted longer. No patients developed signs of intolerance, and no changes occurred in blood amylase levels in controls or diabetic subjects with chronic pancreatitis. Some changes did occur in patients with obstructions at the head of the pancreas and some transitory but large variations did occur in blood sugar levels.

6867 THE DIAGNOSTIC AND PROGNOSTIC VALUE OF DETERMINING THE UROCANINASE ACTIVITY IN VIRAL HEPATITIS.

(Rus.) Blinova, T. V. (Gor'ki Sci.-Res. Inst. Epidem. Microbiol., USSR). *Sovet Med* 31(3):41-44, 1968.

Urocaninase activity was determined spectrophotometrically by a modification of the method of Tabor and Mehler on serum from 152 patients with viral hepatitis, 102 with jaundice due to other causes, and 110 normal controls; all were adults. In contrast to the controls, who had no demonstrable serum urocaninase activity, 97.7% of the patients with viral hepatitis did. Activity ranged from 3-15 U in 75.7% and was 2 U or less in 9.4%. Urocaninase activity was low (2.5 U or less) or absent in 39 patients with obstructive jaundice; 58 patients with cholecystitis, cholelithiasis, and chronic hepatitis; 2 patients with toxemia of pregnancy; and 2 with leptospirosis. In patients with viral hepatitis a correlation was observed between serum urocaninase activity, serum bilirubin values, and clinical symptoms of jaundice. Serum urocaninase activity decreased rapidly and was absent at discharge in patients with mild forms of viral hepatitis. In patients with severe and moderately severe forms this decrease was slower and enzyme activity was still present in 70% 30-40 days after the onset of jaundice. Prednisolone therapy, used on 7 patients, caused rapid decreases in urocaninase activity, but increases occurred in enzyme activity after the drug was discontinued. The effect of prednisolone was probably due to a reduction in hepatocellular permeability.

6868 EXPERIENCE WITH THE USE OF IMMUNOFLUORESCENCE IN THE DIFFERENTIAL DIAGNOSIS OF VIRAL HEPATITIS.

(Rus.) Barinskii, I. F. (D. I. Ivanovskii Inst. Virol., Acad. Med. Sci. USSR, Moscow), B. K. Bezprozvannyi and M. Ie. Semendiaieva. *Sovet Med* 31(3): 34-36, 1968.

An immunofluorescence technique was used to examine smears from punch liver biopsies made on 35 patients, aged 18-53 yr, with signs of viral hepatitis. Material, fixed in absolute methanol, was stained by the direct Coons' method with a conjugate of placental γ-globulin and fluorescein-isothiocyanate containing no more than 2 molecules of dye per molecule protein. Intense diffuse fluorescence was observed in the cytoplasm, but not in the nuclei, of liver cells from 7 patients with acute viral hepatitis which had been confirmed by histological methods. Occasional mesenchymal cells displayed fluorescence of the same intensity. This same fluorescence persisted during recovery in 2 of the 3 patients examined; 1 of these patients developed signs of chronic hepatitis. No immunofluorescence was seen in biopsy material from 5 patients with drug-induced toxic-allergic hepatitis or in protein-deficiency cirrhosis even though parenchymal cirrhosis was present in some patients in the former group. This method was used to distinguish between residual symptoms of viral hepatitis and chronic hepatitis in 21 cases. No immunofluorescence was seen in 9 of 16 patients with residual fibrosis, hyperbilirubinemia, and fatty dystrophy of the liver or in 1 patient with chronic balloon dystrophy. Immunofluorescence was observed in 2 patients with clinical symptoms of chronic hepatitis who had atypical histological changes.

6869 SERUM LIVER ENZYMES IN DIFFERENT FORMS OF VIRAL HEPATITIS IN CHILDREN.

(Rus.) Braginskii, D. M. (Munic. Clin. Hosp. Infect. Dis. No. 82, Moscow, USSR) and I. V. Shakhgil'dian. *Sovet Med* 31(1):46-51, 1968.

Serum fructose-1-phosphate aldolase and sorbitol dehydrogenase activities were studied on 253 children with viral hepatitis (51 with anicteric and 202 with icteric hepatitis) and on 60 children with other diseases. Parallel studies were made of other nonspecific enzymes. The children with jaundice included 133 with mild, 59 with moderately severe, and 10 with severe hepatitis. During the first 5 days of the disease, fructose-1-phosphate aldolase activities were increased in all patients with mild and moderately severe hepatitis and in 93.5% of those with anicteric hepatitis. Correlations were observed between mean values for enzyme activities and the severity of the hepatitis during this period. Similar results were obtained for sorbitol dehydrogenase. Activities of both enzymes decreased rapidly and mean values were significantly lower between the 6th and 10th days of the disease than during the first 5 days. The mean activity of sorbitol dehydrogenase decreased more rapidly than that of fructose-1-phosphate aldolase and returned to normal 11-15 days after the onset of jaundice. During early stages of infection fructose-1-phosphate aldolase activities were of greater diagnostic value than those of fructose diphosphate aldolase, glucose phosphate isomerase, malic dehydrogenase, or alkaline phosphatase. Transaminase activities were of greatest diagnostic value later in the course of the infection. The diagnostic value of sorbitol dehydrogenase activities was about the same as that of nonspecific aldolase. Among the 60 children with other diseases, specific and nonspecific enzyme activities were increased in 6 with toxic or bacterial hepatitis. Slight increases in nonspecific enzyme activities were associated with normal fructose-1-phosphate aldolase and sorbitol dehydrogenase activities in 8 of the remaining 54 patients with other diseases.

6870 CYTOCHEMICAL STUDY OF LEUKOCYTE ALKALINE PHOSPHATASE ACTIVITY IN SOME INFECTIOUS DISEASES OF THE INTESTINE.

(Rus.) Nagoiev, B. S. (Kuban Med. Inst., Krasnodar, USSR). *Sovet Med* 31(1):52-56, 1968.

Alkaline phosphatase activity in the neutrophils from 200 patients with various gastrointestinal disorders was studied with an azo coupling reaction. On admission and 1-4 days after the onset of symptoms, significant increases in alkaline phosphatas levels were found in 50 of 57 patients with dysentery, 44 of 50 patients with salmonellosis, and 3 of 3 patients with typhoi fever. Increased enzyme activity was seen with normal, high, and low white-blood counts. No relation was found between the increase in enzyme activity and the presence of a shift to the left, increased sedimentation rates, or the type of bacterial infection. In contrast to these findings, alkaline phosphatase values were normal in 74 of 88 patients with other gastrointestinal diseases (chronic gastritis, acute gastroenteritis, enterocolitis, chronic colitis, and nonspecific ulcerative colitis). In patients with dysentery and salmonellosis a significant decrease occurred in alkaline phosphatase activity between the 5th and 8th day. In most cases levels returned to normal between the 9th and 17th days. At this time a significant decrease in alkaline phosphatase activity occurred in patients with typhoid fev but values were still above normal. In dysentery and salmonell sis, correlations were observed between the increase in alkaline phosphatase levels and the severity of the clinical symptoms.

6871 DETERMINATION OF AMYLASE ON DILUTE SERUM AS A TEST FOR CHRONIC PANCREATITIS.

(Rus.) Tuzhilin, S. A. (Inst. Nutrit., Acad. Med. Sci. USSR, Moscow) and A. A. Petrov. *Lab Delo* (7):424-426, 1968.

Amylase activity was determined by the method of Smith and Roe on whole serum and 1:2 dilutions of serum from 72 patients with surgically confirmed chronic pancreatitis and from 11 normal controls. Significant differences between pancreatit patients and controls were found for whole serum amylase (WSA) levels and for the ratio of the difference between amylase activities in whole and dilute serum (WSA-DSA) to WSA levels (WSA-DSA/WSA = 0.52 and 1.28, resp.). Of the 72 patients, 54 (75%) had abnormal WSA-DSA/WSA values, 40 (56 had abnormal WSA values, and 44 (61%) had abnormal WSA-DSA values. Statistically significant decreases occurred in all these values when serum was incubated with Trasylol, confirming literature reports that increased amylase values in dilute serum are due to decreases in inhibitor concentrations. In 4 rabbits with acute pancreatitis induced by ligation of the pancreatic ducts, significant changes in WSA values were found 1 day after surgery and in WSA, DSA, and WSA-DSA/WSA value 2 days after surgery. All values returned to normal 5 days after surgery. Similar, but more persistent changes occurred in 4 dogs with chronic pancreatitis produced by insufficiency of the sphincter of Oddi.

6872 DUODENAL KINESIOGRAPHY IN THE DIAGNOSIS OF DISEASES IN THE PANCREATICODUODENAL REGION.

(Rus.) Sokolov, V. I. (Sci.-Res. Inst. Clin. Exp. Surg., Min. Hlth. USSR, Moscow), V. F. Smirnov and M. M. Oreshenkov. *Khirurgiia (Moskva)* 44(3):119-124, 1968.

Duodenal motility was measured in 38 patients (32 women and 6 men, aged 22-74 yr), using a balloon-tipped catheter connected to a manometer. The tracings obtained depended on th extent of the disease and the distance between the duodenum and the affected organ. Tracings were divided into 4 different categories depending upon the amplitude of the small waves: 1) normal (38-42 mm H_2O), 2) hypokinetic (18-26 mm H_2O), 3) hyperkinetic (60-65 mm H_2O), and 4) akinetic (5-15 mm H_2O). Normal motility was seen in 2 patients with chronic appendicitis, 1 with a partial intestinal obstruction due to adhesions, and 1 with acute cholecystitis. Hyperkinetic tracings were found in 6 patients (2 with cholelithiasis, 2 with biliary tract dyskinesia, and 2 with stomach cancer). Hypokinetic activity was observed in 12 patients (5 with acute cholecystitis, 2 with duodenitis, 2 with chronic cholecystitis and pancreatitis, 2 with acute pancreatitis, and 1 with papillitis). Akinetic motility, which has the greatest diagnostic value, was found in 13 patients with chronic pancreatitis and in 2 with papillitis. In addition to its use in diagnosis, duodenal motility measurements can be made to control electrical or pharmacological stimulation of the duodenum for duodenal stasis, postoperative ileus, and other conditions of this type.

6873 SECRETION OF ^{131}I-LABELED ALBUMIN INTO THE INTESTINE IN ENTEROPATHIES.

(Ger.) Fodor, O. (3rd Med. Clin., Cluj, Romania), D. Dumitrascu, J. Szantay, S. Tragor and G. Ciofu. *Z Gastroent* 6(2):114-120, 1968.

In 25 controls and 57 patients with chronic nonspecific enteropathies (enterocolitis, gastric resections, and achlorhydric diarrhea), radioactivity was measured in plasma and juice aspirated from the proximal jejunum for 3 hr after they were given 2 μC/kg of ^{131}I-labeled albumin i.v. Curves for total ^{131}I radioactivity in jejunal juice were about the same for controls and patients with gastrointestinal disorders, and individual values were widely scattered. Values for protein-bound radioactivity in jejunal juice were very low, and those of about half of the patients overlapped with control values. However, 37 of the 57 patients secreted larger amounts of ^{131}I-labeled albumin, and mean values for controls and patients differed significantly. Plasma radioactivity levels were about the same in controls and patients. Values for albumin clearance, calculated by van Slyke's method, were also similar in controls and patients. The quantity of ^{131}I-labeled albumin, expressed as a percentage of the quantity in plasma, was significantly higher in patients. When jejunal juice was subjected to electrophoresis, radioactivity almost always appeared in the prealbumin region and sometimes in the albumin region. Differences between controls and patients were small. The intestinal loss of protein-bound radioactivity, which cannot always be accounted for by microscopically visible lesions, is concluded to be the only valid criterion of protein-losing enteropathy.

6874 INDIRECT HAEMAGGLUTINATION AND AMOEBA IMMOBILIZATION TESTS AND THEIR EVALUATION IN INTESTINAL AND EXTRA-INTESTINAL AMOEBIASIS.

(E.) Prakash, O. (All-India Inst. Med. Sci., New Delhi), B. N. Tandon, I. Bhalla, A. K. Ray and V. K. Vinayak. *Indian Pract* 21(10):637-645, 1968.

The results of hemagglutination (h.a.) and ameba immobilization (a.i.) tests were compared in 46 patients with amebic diseases of the liver and/or intestine, 32 patients with miscellaneous disorders, and 27 controls. Antigen was prepared from 5 strains of *Entamoeba histolytica* grown with a strain of *Klebsiella* and subjected to sonic disintegration following purification. The ameba immobilization test was considered positive if at least 50% of cells showed no mobility for at least 10 sec after incubation with serum (40-45 min). Cases of amebic liver abscess (16) were diagnosed positive in 12 cases by h.a. and in 14 cases by a.i. Out of 13 cases of amebic hepatitis, 7 were positive by h.a. and 8 positive by a.i. All 3 cases of intestinal amebiasis with hepatitis tested gave positive results with both tests, and in the group with intestinal amebiasis alone (14), 7 and 8 were positive by h.a. and a.i., resp. The h.a. test gave a false positive in 5 control patients and 1 normal control; the a.i. test gave 4 false positives in control patients. It is noted that the a.i. test is more specific in detecting antibodies against *E. histolytica*, particularly in cases of chronic amebiasis. The serologic tests are of special diagnostic value in cases of extra-intestinal amebiasis, in which attempts to isolate the organism are often negative (13 out of 16 cases of liver abscess), and should be used in addition to parasitologic studies.

6875 PANCREATIC SCANNING: CLEVELAND CLINIC EXPERIENCE.

(E.) Rodriguez-Antunez, A. (Cleveland Clin. Found., Ohio), T. A. Egleston, E. J. Fillson, B. H. Sullivan Jr. and C. H. Brown. *Lahey Clin Found Bull* 17(1):29-33, 1968.

The results of pancreatic scanning in 165 patients with suspected pancreatic neoplasms or advanced pancreatitis, of which 144 were suitable for analysis, are presented. Of 107 pancreatic scintigrams diagnosed as normal, 103 were correctly diagnosed with 4 false negatives (pancreatic tumors), verified surgically or clinically. Of 37 scans diagnosed as abnormal due to a partial or distorted visualization of the pancreas, 21 were correctly diagnosed with 16 false positives. No significant improvement in the accuracy of interpretation of the abnormal pancreatic scan was noted when compared to a previously reported series. It was noted that if the pancreatic outline on the scan appears distorted or has an abnormal configuration, the presence of radioactivity within the ileum is suggestive of normalcy, whereas serious pancreatic disease is suggested when the pancreas cannot be identified and there is no radioactivity in the intestine. Pancreatic scanning is a reliable way to exclude pancreatic disease if the pancreas is well visualized.

6876 A CORRELATION OF INFUSION HEPATIC ANGIOGRAPHY AND LIVER SCANNING.

(E.) Wise, R. E. (Lahey Clin. Found., Boston, Mass.) and F. J. Siber. *Lahey Clin Found Bull* 17(1):21-27, 1968.

A total of 70 infusion hepatic angiograms were performed in 45 patients with surgically proven primary or secondary hepatic malignancies. The method consists of the pressure injection of 75-90 ml of iodinated contrast material (flow rate of 2 ml/sec) via an indwelling catheter positioned in the hepatic artery, followed by the recording of 6 successive exposures, beginning 5 sec after the start of the infusion. In 35 of the 45 patients, 60 liver scans (150 μC of ^{198}Au) and 63 angiograms were performed. Discrepancies in liver morphology were noted in 15 of the 35 patients; in 10 cases, 1 or more segments of the liver were not opacified due to arterial occlusion. In these cases, the liver scan outlined the nonopacified segment. In 1 case, neither the scan nor the angiogram depicted the right lobe (occlusion of right hepatic artery). In 6 cases, a deficiency in scanning technique was noted, in which the left lobe of the liver was not delineated (left lobe was hypertrophied). This was attributed to the frequently noted inadequate count rate obtained for the left lobe by using the maximal count rate over the right lobe and the minimal count rate over the usual position of the left lobe. In no instances were lesions of 0.5-3.0 cm seen as discrete lesions on the scan; in contrast, avascular lesions as small as 0.5 cm were noted with the angiograms. Good correlation was generally found between the angiogram and the liver scan in the evaluation of lesion size. If the infusion hepatic angiogram, which gives an accurate estimate of liver morphology, is obtained before scanning, useful information is obtained, since proper interpretation of the scan demands prior knowledge of liver size and shape. The angiogram also provides accurate information with respect to lesion size and is a good method of following the effectiveness of chemotherapy for liver lesions. The 2 techniques are thus complimentary to each other.

6877 INTRADERMAL TEST IN THE DIAGNOSIS OF *SCHISTOSOMA MANSONI* INFECTION.

(E.) McMahon, J. E. (East Afr. Inst. Med. Res., Mwanza, Tanzania). *E Afr Med J* 44(10):438-440, 1967.

On Ukerewe Island, a known hyperendemic area of *S. mansoni* infection, stool cultures for *S. mansoni* ova were made on 160 African subjects who were also tested for *S. mansoni* with intradermal injections of 0.05 ml of Melcher's sterile acid-soluble protein fraction of adult worms. There were 75 schoolchildren and 25 adults with positive stools; skin tests were positive in 77.3% of these schoolchildren and 100% of the adults. There were 30 schoolchildren and 30 adults with negative stools; 33.3% of these schoolchildren and 43.3% of the adults had positive skin tests. Positive skin tests were also obtained in 5 European adults with positive stools. There was no difference in the skin reactions of males and females of either group; those over 30 yr old tended to have larger skin reactions than young adults or children. No correlation was noted between the worm burden as measured by egg output and the intensity of the skin reaction as measured by the wheal. When skin tests were repeated in 70 subjects 6 months after curative treatment with Ambilhar or after 4 months of suppressive therapy with lucanthone and Ambilhar, none of the previously positive skin reactions became negative, and a tendency for a more intense post-therapeutic skin reaction was noted. Intradermal testing is felt to be of value as a diagnostic aid in individual patients, in epidemiological studies, and prior to mass treatment in adults; however, the test is of no use for evaluating chemotherapeutic effectiveness.

6878 CINECHOLANGIOGRAPHIC OBSERVATIONS AFTER BILE DUCT SURGERY.

(E.) Scheinin, T. M. (Dept. Surg., U. Turku, Finland), E. Lehtinen and M. I. Linna. *Scand J Gastroent* 2(3):175-180, 1967.

Solitary film cholangiograms and cinecholangiograms were made using indwelling T-tubes on 45 patients after biliary tract surgery. Conventional cholangiograms revealed various abnormalities in 14 patients: filling defects, deformities, and apparent obstructions to bile flow; repeated examinations failed to clarify their significance. Cinefluorography, however, showed them to be due to clinically insignificant anatomic and physiologic mechanisms or to technical errors during spot film examination. Cinecholangiography also provided information as to the kinetics, sphincteric activity, and bile flow in the surgically explored biliary tract; after transduodenal sphincterotomy not exceeding 20 mm in length, the cinefluorographic behavior of the choledochoduodenal junction was similar to that of the normal, intact sphincter apparatus, and the responses to morphine and nitroglycerine were also similar. In all patients, removal of the T-tube was followed by complete recovery. Cinecholangiograp should not be regarded as a substitute for conventional cholangiography, but it can add significant information to the latte

6879 THE SIGNIFICANCE OF COMMON DUCT VISUALIZATION IN THE ORAL CHOLECYSTOGRAM WITHOUT VISIBLE GALL BLADDER.

(E.) Sieniewicz, D. J. (Montreal Gen. Hosp., Quebec, Canada) and J. Kiss. *J Canad Ass Radiol* 18(4):459-463, 1967.

In a series of 4551 patients without previous gallbladder surgery, oral cholecystography revealed contrast medium (iopanoi acid) in the common bile duct without visualization of the gall bladder in 59 cases (1.1%). Repeat examinations demonstrated improved concentration of contrast medium in the common duct in about half of the patients but did not provide addition significant information. The cystic duct was visualized in 13 and a portion of the hepatic duct system was identified in 23 patients. No common clinical features characterized the group, except for the usual varied pain pattern of patients wit cholelithiasis. Thirty-five of the 59 patients underwent cholecystectomy. All revealed cholelithiasis with acute, subacute o chronic cholecystitis. The incidence of stones in the common duct or of strictures was no higher than expected for an opera tive group of this type. Acute hydrops of the gallbladder was found in 1 case. Three case reports are presented in detail. It is concluded that when the common duct is outlined by contrast medium without a visible gallbladder or opaque calcul the patient probably has gallbladder disease with cholelithiasis and reexamination is not indicated.

6880 THE SECRETIN TEST IN THE EVALUATION OF PANCREATIC EXOCRINE FUNCTION.

(E.) Bertero, R. (Dept. Gen. Clin. Med., U. Turin, Italy), C. Saracco, G. Bruno and M. De La Pierre. *Panminerva Med* 10(6):250-258, 1968.

Pancreatic juice obtained by duodenal intubation (Dreiling's technique) was evaluated for pH, volume, maximum bicarbona concentration, and total bicarbonates in 27 normal subjects, 11 with chronic pancreatitis, 8 with pancreatic carcinoma, 30 with diabetes, 11 with ulcerohemorrhagic rectocolitis, 8 with cholelithiasis, 3 with malabsorption due to primary villopathy, and 1 with moniliasis. Each patient received Boots secretin (1 U/kg i.v. over a 2-min period), and collections were made for 10 min before secretin injection (basal period) and then for 2 10-min followed by 2 20-min periods. In the normals, total secretion was 139 ml ± 37, volume per kg was 2.29 ml ± 0.87, maximum bicarbonate concentration was 100.8 mEq/liter ± 18.8, and total bicarbonate was 12.18 mEq ± 3.61 In chronic pancreatitis, a reduction in total pancreatic juice was noted in only 3 of the 11 cases (49-98 ml), and volume/k was decreased in only 2 cases (0.96-1.1 ml), whereas maximu bicarbonate concentration and total bicarbonates were consistently lower than in the normals. In the 8 patients with pancreatic carcinoma, 6 cases involved the head of the pancrea while 1 involved the body and tail and 1 involved only the tai tal secretion was decreased in the 1st 6 cases and was normal r high in the last 2 cases. Maximum bicarbonate concentraons were low in 6 and total bicarbonates were low in 7 cases. the 30 patients with diabetes, total secretion was low in 12, the lower limits of normal in 7, and normal in 11. Abnorally low ml/kg values were noted in 15, maximum bicarbonate oncentration was below normal in 12, and total bicarbonate cretion was depressed in 20 cases. In the 11 cases of ulcerave colitis, decreased total secretion was noted in 3, ml/kg values ere increased in 9, maximum bicarbonate concentration was w in 4 and above normal in 3, and total bicarbonates were w in 1 and above normal in 3 cases. In the 8 patients with olelithiasis, low total secretion was noted in 6, low ml/kg alues were noted in 5, low maximum bicarbonate concentraon was noted in 7, and low total bicarbonate values were seen 6. In those with malabsorption, total secretion and ml/kg alues were high in all 3, low maximum bicarbonate concentraon was noted in 1, but total bicarbonates were normal in all ses. The single case of moniliasis showed low maximum conentration and low total values for bicarbonate. These observaons indicate the usefulness of the secretin test in the diagnosis f pancreatic diseases.

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ELECTRON MICROSCOPIC OBSERVATIONS OF NON-SPECIFIC LEAD STAINING IN THE MALL INTESTINE OF THE CAT AFTER LIPID OR CAROHYDRATE FEEDING. *(E.)* Napolitano, L. (U. New lexico Sch. Med., Albuquerque) and J. Howard. *Anat Rec* 60(1):25-35, 1968.

he specificity of Rostgaard and Barrnett's marker technique *lnat. Rec.* 152:325, 1965) for tracing lipid absorption in the nall intestine by the demonstration of lipid-lead complexes as evaluated using jejunal mucosa from adult cats fasted 48 r and fed corn oil (10 -15 ml) or 5% dextrose by stomach ibe. In fasted fat-fed animals electron-microscopic observaons revealed dense lead deposits of varying size located in, n and about the microvillus border of intestinal columnar aborptive cells. Occasionally, the reaction product was either ound to the luminal surface of mucous cells or outlined the iucous inclusions throughout the cytoplasm. At times, the itercellular channel between a mucous cell and an adjacent bsorptive cell was outlined by a dense precipitate; lipid acimulations in these regions are unlikely. Lead deposits having ie same morphology and similar anatomic distribution were ound in or on small intestine epithelial cells of fasted dextrosed cats. Tissues from fat- or dextrose-fed animals extracted in pid solvents prior to incubation in tris-maleate buffer + lead itrate still showed lead deposits related to the microvillus order. Animals fasted for 48 hr and fed neither corn oil nor extrose possessed few lead precipitates related to the microillus border of the epithelial absorptive cells. These findings ndicate that the occurrence of lead deposits following a fatty ieal does not provide a specific demonstration of lipid aborption.

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A HIGH-FREQUENCY METHOD FOR JUDGING THE EXTENT OF DEVIATION IN THE VICINITY F RECTOSIGMOID CARCINOMA. *(E.)* Setka, J. (Charles U. Iosp., Prague, Czechoslovakia), J. Vrana and A. Zwinger. *Amer Proctol* 18(5):369-375, 1967.

he electrical impedance of rectal tissue in the direction of the nal sphincter was determined by application of high-frequency currents in 7 patients (3 females, 4 males) with cancer of the rectum or polyposis of the large intestine, 8 patients (2 females, 6 males) with systemic diseases, ulcerative colitis or irritable colon, and 9 normal subjects (7 females, 2 males). In cases of rectal cancer the impedance values in the vicinity of the carcinoma were generally less than 0.7 mV (0.45-0.72 mV), increasing with distance from the tumor. In one patient with benign polyposis and another studied after resection of the sigmoid for rectal cancer, normal values were obtained (0.7-1.3 mV). The control subjects always showed vales equal to or higher than 0.7 mV (0.7-1.1 mV). In the patients with systemic diseases the impedance values were borderline: irritable colon, 0.89 mV; ulcerative colitis, 0.82 mV; polycythemia vera, 0.55-0.75 mV; eosinophilia, 0.75 mV; pyelonephritis, 0.5 mV; acute myeloblastic leukemia, 0.17-1.0 mV; malignant reticuloblastoma with perianal and perirectal infiltration, 0.45 mV. In one patient with rectal cancer a striking correlation was observed between impedance values and biopsy results at 3 sites: at the base of the tumor (0.55 mV) where poorly differentiated adenocarcinoma with plasmocellular inflammatory infiltration was found, at a site 3 cm distant from the tumor (0.62 mV), where there were moderate, nonspecific, chronic inflammatory changes, and 5 cm from the tumor (0.7 mV), where core irregularities but no convincing carcinoma were observed in the mucosa. It is suggested that this electrometric technique could serve as a guide for determining the required extent of resection in rectal cancer.

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TUBELESS VAGOTOMY AND PYLOROPLASTY: EARLY RADIOLOGIC DETECTION OF GASTRIC RETENTION. *(E.)* Williams, J. A. (Dept. Surg., U. Birmingham, England), A. D. Barnes and D. K. M. Toye. *Amer J Surg* 115(4):454-456, 1968.

A simple radiologic test for the early detection of delayed gastric emptying was performed on 55 patients treated by vagotomy and pyloroplasty for duodenal ulcer. In 35 patients, more than 75% of Gastrografin contrast medium administered the day after surgery (30 ml) left the stomach within 1 hr. In an additional 8 patients, gastric emptying occurred between 1 and 2 hr after administration of Gastrografin. Four subjects still retained about 50% of the total amount of medium at 2 hr, probably due to failure to lie on the right side. X-rays taken at 3 hr in 3 patients (delayed because of administrative difficulties) revealed an empty stomach by that time. No clinical signs of delayed gastric empyting occurred in any of these 50 patients through their postoperative course. Five patients retained all or nearly all of the Gastrografin at 1 and 2 hr after administration. One of these 5 showed satisfactory emptying at 3 hr and 3 more were normal by 24 hr, but one patient required gastric aspiration and i.v. fluids for 3 weeks; this patient did not show gaseous distension. In fact, of 5 patients with marked gaseous distension, only 2 had delayed gastric emptying, while 11 of 12 cases with moderate gaseous distension showed normal emptying. The 1st bowel movement in the 55 patients given Gastrografin occurred on the 2nd to 8th postoperative day (mean 3.6 days), while in an additional 50 vagotomized patients treated without nasogastric suction but given no Gastrografin, it occurred on the 2nd-6th postoperative day (mean 3.5 days). This Gastrografin test provides a simple aid to postoperative management when delayed gastric emptying may be expected and makes it possible to dispense with the routine use of postoperative nasogastric suction.

6884 **EXAMINATION OF THE LIVER USING THOROTRAST: A STUDY OF THE HEPATIC LESIONS PRODUCED IN MAN BY A PARENTERAL INJECTION OF THORIUM DIOXIDE.** *(Fr.)* Collette, J. M. and J. da Silva Horta. pp. 42-48 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

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6963 COLOR LYMPHOGRAPHY DURING ABDOMINAL SURGERY. *(Ger.)* Vojtisek, V. (Fac. Hyg., Charles U., Prague, Czechoslovakia), B. Kasalický, A. Haasová, V. Puchta and A. Chlumská. *Zbl Chir* 92(26a):1892-1905, 1967.

6964 RADIOISOTOPES IN THE DIAGNOSIS OF POSTOPERATIVE BILE PERITONITIS. *(Ger.)* Jirán, B. (Fac. Med. Hyg., Charles U., Prague, Czechoslovakia) and J. Prokopec. *Zbl Chir* 92(26a):1882-1892, 1967.

6965 CLINICAL AND MORPHOLOGICAL ASPECTS OF THE INJECTION OF CONTRAST MEDIA AND DRUGS INTO THE LIVER VIA THE UMBILICAL VEI *(Ger.)* Matiashin, I. M. (Donetsk State Med. Inst., USSR), D. G Doviner, L. I. Gnilitskaia, Iu. V. Baltaitis and V. N. Korobov. *Zbl Chir* 92(26a):1872-1876, 1967.

6966 CLEARANCE OF INDOCYANIN GREEN ("WOFAVERDIN") AS A METHOD FOR EVALUATING THE SEVERITY OF HEPATIC CIRRHOSIS. *(Ger.)* Ehrke, D. (Schwerin Distr. Hosp., Germany), J. Janecki, A. Seifert and H. Balzer. *Acta Hepatosplen* 15(2):102-107, 1968.

See also: 6740,6782,6789,6971,7019,7025,7076,7279,7286,7322,7354,7442,7459,7469,7525

GASTROINTESTINAL DISEASES

ESOPHAGUS

6967 ELECTRICAL POTENTIALS IN THE NORMAL ESOPHAGUS AND IN HIATUS HERNIA. *(E.)*

Goodman, E. N. (Coll. Phys. Surg., Columbia U., New York, N. Y.), C. A. Flood, B. T. Sandler and M. R. Sullivan. *Arch Surg* 96(6):924-928, 1968.

Recordings of esophageal electrical potentials and pressures were made in 56 patients with radiographically evident hiatus hernia and 40 normal controls, using a perorally introduced combined pressure-electrode probe with electrodes spaced 2.5 or 5 cm apart. Electrical potentials were recorded during rest, dry swallowing, swallowing 15 cc water and swallowing 15 g of gelatin. In the controls, the normal electrical response was indicated by an abrupt positive deflection of 10-30 millivolts, lasting 6-15 sec, following the initiation of swallowing. Negative deflections in the distal esophagus (related to the sphincteric zone) were noted in 12 subjects. In the main body of the esophagus, an orderly downward propagation of electrical signals was observed with a time delay of 0.3-1.5 sec between electrodes spaced 5 cm apart; in the gastroesophageal region, deflections were less orderly. The electrical potentials were correlated with intraluminal pressure recordings, preceding the latter by 0.5-2.0 sec. In patients with hiatus hernia, 3 groups were identified. In group A (12 cases), a normal response was seen as far down as 30 cm from the incisor teeth; below this level, the downward propagation of the electrical wave was impaired. In 8 patients, propagation stopped at 30 cm and in 4 it stopped at 35 cm; pressure responses, in most cases, were interrupted at the same level as the electrical responses. In group B (38 cases), greater abnormality was noted, with the height, shape, and downward propagation of the electrical response being inconsistent in the same individual. Random activity unrelated to swallowing was commonly seen. However, good correspondence between electrical responses and pressure recordings was usually obtained. In group C (7 cases), absent or weak and irregular responses were commonly noted, with intraluminal pressures corresponding to the electrical pattern. It is concluded that in hiatus hernia, there is an interruption of the normal sequence of electrical response signals from various levels in the esophagus.

6968 COURSE OF ESOPHAGEAL VARICES IN CIRRHOSIS, IN THE ABSENCE OF PORTAL DECOMPRESSION. *(E.)*

Palmer, E. D. (VA Hosp., East Orange, N. J.). *Gastroenterologia* 107(6):337-343, 1967.

Reported measurements of the esophageal varices were made by esophagoscopy in 256 patients (230 males and 26 females, 24-81 yr old) with histologically proven nutritional or coarse nodular cirrhosis who were not treated by shunt procedures. Of these patients, 230 were considered to be chronic alcoholics, while 8 had probably had viral hepatitis; none had schistosomiasis. There was a total of 709 successful examinations, repeated at an average interval of 15.3 months. Based on the severity of the varices at final esophagoscopic examination, 62 had improved, 106 had not changed, and 88 were worsening. In 46 instances varices were absent on the initial examination but appeared later. In 59 instances varices disappeared spontaneously, but in 28 of these cases at least one further examination was made, and it was found that the varices had returned in 16 patients. It is concluded that disappearance must be considered a temporary phenomenon. Among the behavioral oddities was the occasional observation that the diameter of the varices had become larger, while at the same time their distribution had become restricted, or vice versa. Cessation of bleeding remains the only reliable criterion for the success of shunt surgery.

6969 EXPERIENCE WITH TRANSTHORACIC CARDIOMYOTOMY FOR CARDIOSPASM. *(Ger.)*

Köle, W. (Provincial Hosp., Graz, Austria). *Chirurg* 39(2):86-89, 1968.

Heller's procedure for cardiomyotomy was modified by using a thoracic approach via the 8th intercostal space on the left, with paravertebral resection of about 2 cm of the 8th rib. A 10-15 cm incision was made, and a spindle-shaped piece of tissue was removed from the longitudinal and transverse musculature at the lateral edge of the incision. This modified procedure was successfully employed on 37 of 39 patients with an average age of 36 yr. Of the 2 failures, 1 occurred in a patient who had previously undergone Heller's procedure via an abdominal approach and had cardial scar tissue; good results were obtained with a cardiofundectomy using an end-to-side esophagogastrostomy. The other failure was in a man who developed a recurrence with chronic hypertrophic esophagogastritis; good results were obtained with a resection of the esophagus and cardia and an end-to-side esophagogastrostomy. There were no operative deaths and no complications.

6970 PORTAL HYPERTENSION IN CHILDHOOD: EXPERIENCES WITH VOSSSCHULTE'S TECHNIC OF DISSECTION-LIGATION. *(E.)*

Kundert, J. G. (Munic. Hosp., Bremen, Germany) and F. Rehbein. *J Pediat Surg* 3(4):494-502, 1968.

Eight patients (2 girls and 6 boys, 1.5-14 yr old) with extrahepatic portal hypertension were treated by Vossschulte's technique for dissection-ligation of esophageal varices 1 month after splenectomy, portography, measurement of portal pressure (elevated in all cases) and liver biopsy. The technique consists of esophageal ligation with a catgut suture over a metallic cylinder, introduced into the lower esophagus through a gastrostomy. The result is a circumferential scar which completely interrupts communication of the esophageal veins with the portal venous system. After resolution of the catgut sutures, the cylinder is removed through the gastrostomy by withdrawal of nylon strings attached to the prosthesis. Postoperatively, there were no instances of esophageal leakage. Four of the 8 children developed esophageal stenosis 3-4 months postoperatively. Sufficient dilatation of the scarred area was achieved in all cases by esophageal bouginage. Esophageal varices present preoperatively could not be demonstrated postoperatively in 7 patients available for follow-up (2 months-7.5 yr). One child lost to follow-up died of general infection 7 months after the operation. There have been no episodes of recurrent bleeding from esophageal varices in any of the patients. This technique is simple and safe in comparison to the ligation of individual varices or colonic interposition.

6971 THE ETIOLOGY AND DIAGNOSIS OF ESOPHAGITIS IN PATIENTS WITH PROGRESSIVE SYSTEMIC SCLEROSIS (SCLERODERMA).

(Por.) Bettarello, A. (Fac. Med., U. São Paulo, Brazil) and S. Zaterka. *Arq Gastroent* 5(1):19-28, 1968.

Esophageal histology, the response to acid perfusion, gastric acid secretion (basal and Histalog-stimulated), and the manometric characteristics of the lower esophageal sphincter were studied in 24 patients (5 men and 19 women, 21-56 yr old) with clinically diagnosed disseminated scleroderma in an effort to determine the pathogenesis of esophagitis in such patients. There was no significant difference in gastric secretion between the scleroderma patients and 20 healthy controls. Nevertheless, radiology revealed esophagitis in 3 of 21 cases, and alterations such as esophageal dilatation and reduced peristalsis in 6 others. The acid perfusion test was positive in 14 of 15 patients with retrosternal pyrosis, but was negative in all 7 patients without pyrosis. Blind esophageal biopsy confirmed the diagnosis of esophagitis in 11 of 12 patients with retrosternal pyrosis and in 2 of 7 without pyrosis. This technique is recommended as the most reliable method of diagnosing esophagitis, particularly if done under fluoroscopic control. Electromanometric studies revealed a significant decrease in tone of the lower esophageal sphincter in patients with scleroderma and esophagitis (to 5.9 cm H_2O over an extent of 2.0 cm compared to 12.2 cm H_2O over an extent of 2.4 cm in 10 normal controls); in 6 scleroderma patients without esophagitis, the comparable values were 9.8 cm H_2O and 2.0 cm. It is concluded that this decrease in tone is one (but probably not the only) factor responsible for esophageal reflux in patients with scleroderma. Refluxed gastric juice, coming into contact with an esophageal mucosa already weakened by the basic disease process, probably explains the frequent occurrence of esophagitis in these patients.

6972 CARCINOMA OF THE THORACIC ESOPHAGUS.

(E.) Baker, R. R. (Johns Hopkins U. Sch. Med., Baltimore, Md.) and S. Lott. *John Hopkins Med J* 121(3): 153-161, 1967.

Clinical findings, methods of management, and autopsy observations are reviewed for 156 cases of squamous cell carcinoma of the thoracic esophagus (133 males and 23 females, 63% between 50 and 60 yr old) admitted to the Johns Hopkins Hospital during the period 1952-63. Dysphagia was the most common presenting symptom (135 of 156 cases); complaints in the remaining 21 patients were, in order of decreasing frequency: epigastric pain, chest pain, pain from osseous metastasis, anorexia and weight loss, hoarseness and hematemesis. Only 5 patients (3.2%) survived 3 yr or more. In 12 patients with lesions of the mid third of the esophagus and without evidence of distant metastases, the operative mortality was 33%. Radiotherapy of 41 patients with mid-third lesions and no distant metastases resulted in 39 deaths attibutable to the tumor; radiation was a contributing cause of death in 8 cases. Only one patient survived for 3 yr without signs of recurrence. Among patients with lesions of the mid third of the esophagus and signs of distant metastases, the operative mortality was 41% and there were no long-term survivors. Eleven patients with mid-third lesions associated with distant metastases who received no treatment had an average survival of less than one month. Three of 27 patients with tumors of the lower third of the esophagus survived without evidence of recurrence for 5 yr postoperatively. The operative mortality was 14%. Survival averaged 11 months in 5 patients with curable lower-third lesions treated by radiation. Resectio was considered curative in 7 and palliative in 6 of 13 autopsies classified as postoperative deaths. In a series of 17 autopsies on patients treated by radiation, 6 of 7 classified as potentially curable had evidence of local disease, and one showed distant metastasis, while 10 of 10 classified as palliative had histological evidence of local disease and distant metastases. Any significant increase in survival rate depends on the diagnosis of these tumors before they produce dysphagia; increased use of exfoliative cytology, radiography and esophagoscopy is indicated. A general plan of management of esophageal carcinoma is outlined, and it is concluded that palliation is generally best accomplished by radiation alone.

6973 EXPERIENCE AND RESULTS WITH THE SURGICAL TREATMENT OF ACUTE BLEEDING FROM ESOPHAGEAL VARICES.

(Ger.) Fischer, R. (Munic. Hosp., Stuttgart-Bad Canstatt, Germany) and C. Friedel. *Med Welt* 40(47):2823-2828, 1967.

Of 53 cirrhotic patients (39 men and 14 women, aged 23-70 yr) with bleeding esophageal varices, 37 underwent shunt surgery and 16 were given palliative surgery; 2 additional patients were inoperable. Of the 37 patients in the first group, 16 had acute and 21 had chronic recurrent bleeding. Portacaval shunts were established in 34 cases and splenorenal shunts in 3. Seven of these patients died after surgery. Of the 16 patients treated palliatively, 14 had acute bleeding and 2 had chronic recurrent bleeding. Treatment consisted of splenectomy and subdiaphragmatic block in 6 cases, transgastric acupressure in 6 cases, transthoracic acupressure on the open esophagus in 3 cases, and the dissection ligation technique of Vossschulte in 1. Seven of these patients also died after surgery. Of the total of 53 operated patients, 23 eventually died and 16 survived. Of 15 cirrhotics followed for 1 yr or more after portacaval decompression, 8 were in good condition.

6974 THE ANATOMY OF THE LOWER ESOPHAGEAL SPHINCTER IN NORMAL SUBJECTS AND IN TWO PATIENTS WITH MEGAESOPHAGUS AND MUSCULAR HYPERTROPHY OF THE CARDIA.

(It.) Luccioli, G. M. (S. Giovanni Evangelista Munic. Hosp., Tivoli, Rome, Italy). *Gazz Int Med Chir* 72(24-bis):3441-3455, 1967.

The anatomy of the distal esophagus is described in detail on the basis of autopsy studies of the esophagogastric junction in 20 fresh cadavers and radiological and clinical findings in 2 cases of megaesophagus (1 man and 1 woman, both 58 yr old with a 10-yr history of dysphagia) accompanied by cardial hypertrophy, hyperactive peristalsis and (in one case) an epiphrenic diverticulum. Both patients were treated successfully by esophagocardiomyotomy, without pyloroplasty, resulting in disappearance of the symptoms despite persistence of the esophageal dilatation and disturbances in motility. Longitudinal sections from the distal esophagus of the 20 cadavers revealed no sign of muscular thickening, and it is concluded that no true esophageal sphincter in the anatomical sense exists. The type of megaesophagus encountered in these 2 cases is felt to be distinct from both idiopathic esophageal muscular hypertrophy, which is quite rare, and congenital hypertrophic stenosis of the cardia.

6975 **OESOPHAGEAL ABNORMALITIES IN SJÖGREN'S SYNDROME.** *(E.)* Hradský, M. (Fac. Med., Hradec Kralove, Czechoslovakia), I. Hybášek, V. Černoch, V. Sazmová and J. Juran. *Scand J Gastroent* 2(3):200-203, 1967.

Esophagoscopic and X-ray examinations performed in 30 patients (25 females and 5 males, 21-75 yr old) with Sjögren's syndrome revealed mucosal atrophy throughout the entire ength of the esophagus in all cases. No malignant changes were noted on X-ray examination; however, in 4 patients, a characteristic incisure was found by contrast filling in the anterior wall of the pharynx close below Killian's sphincter. Dysphagia during the ingestion of solid food in these patients was not due to pathological esophageal membranes, but to lack of saliva and dryness of the mucosa, making the passage of solid food difficult. It is believed that the mucosal atrophy of the esophagus in patients with Sjogren's syndrome should be included among the major features on which the diagnosis of this entity is based.

6976 **SECTION OF THE CRICOPHARYNGEUS MUSCLE FOR DYSPHAGIA.** *(E.)* Blakeley, W. R. (U. New Mexico Sch. Med., Albuquerque), E. J. Garety and D. E. Smith. *Arch Surg* 96(5):745-762, 1968.

The clinical significance of the cricopharyngeus muscle in dysphagia localized to the neck and not due to a foreign body or a neoplasm is reviewed on the basis of an extensive literature survey and the authors' experience with 7 patients (51-75 yr old) treated surgically for nonobstructive cervical dysphagia. Dysphagia can arise not only from failure of the cricopharyngeus to relax properly (achalasia), but also from weakness or paralysis of pharyngeal constrictors (resulting from myopathic, central neuropathic or peripheral neuropathic lesions). The 7 patients, representing each of these forms of dysphagia, had the following findings in common: advanced age, dysphagia definitely symptomatically localized to the neck, strongly positive history of aspiration and pneumonitis in the majority, and characteristic "butterfly" pooling of barium in valleculae and pyriform sinuses of the megapharynx. Six patients (1 refused surgery) were treated by extramucosal, midline section of the cricopharyngeus muscle with no operative morbidity or mortality. Five of these patients had complete objective and subjective relief of dysphagia, as shown by cine-esophagograms, weight gain and cessation of aspiration. The only failure occurred in an elderly man, hemiplegic for 4 years; in a case of such profound motor and sensory loss, any treatment short of laryngectomy seems unlikely to succeed. The surgical procedure, as well as the anatomy and physiology of the region, is described in detail.

6977 **EXPERIMENTAL REPLACEMENT OF A SEGMENT OF ESOPHAGUS WITH SMALL INTESTINE.** *(Hun.)* Szabó, L. E. (U. Med. Sci., Budapest, Hungary), S. Karácsonyi and I. Katona. *Magy Sebesz* 20(4): 209-217, 1967.

6978 **SOME PROBLEMS CAUSED BY GASTROESOPHAGEAL REFLUX.** *(Cz.)* Šistek, V. (Fac. Gen. Med., Prague, Czechoslovakia) and O. Vaněčková. *Cesk Gastroent Vyz* 21(6):356-359, 1967.

6979 **THE HIDDEN DANGERS OF HIATAL HERNIA.** *(Fr.)* Leymarios, J. *Concours Med* 90(1):845-852, 1968.

6980 **COMPRESSION OF ESOPHAGEAL VARICES BY MEANS OF THE SENGSTAKEN-BLAKEMORE TWIN-BALLOON CATHETER.** *(Fr.)* Testart, J. (Lariboisiere Hosp., Paris, France). *Concours Med* 90(1):647-653, 1968.

6981 **LEIOMYOMA OF THE ESOPHAGUS: REPORT OF A CASE.** *(Por.)* Roselli, A., F. Paulino and G. Gonzalez. *Arq Gastroent* 5(3):141-145, 1968.

6982 **INCOMPETENCE OF THE DISTAL ESOPHAGEAL SPHINCTER IN INFANTS: ITS RELATIONSHIP TO HIATAL HERNIA.** *(E.)* Lilly, J. R. (George Washington U. Sch. Med., Washington, D. C.) and W. P. Tunell. *Clin Proc Child Hosp DC* 24(9):301-307, 1968.

6983 **THE SURGICAL MANAGEMENT OF OLD ESOPHAGEAL PERFORATIONS.** *(E.)* Keen, G. (Frenchay Hosp., Bristol, England.). *J Thorac Cardiovasc Surg* 56(4):603-606, 1968.

6984 **PULMONARY COMPLICATIONS OF HIATUS HERNIA: CASE REPORT.** *(E.)* Tobias, J. A. (Virginia Mason Hosp. Seattle, Wash.) and L. D. Hill. *Bull Mason Clin* 22(3):91-98, 1968.

6985 **ESOPHAGEAL PERFORATION BY CERVICAL SPUR DURING GASTROSCOPY.** *(E.)* Davidson, J. H. *Gastroint Endosc* 15(2):79-80, 1968.

6986 **REOPERATIONS FOR HIATAL HERNIA.** *(Fr.)* Fékété, F. (Beaujon Hosp., Clichy, Paris, France). *Entret Bichat [Chir]* pp. 13- 14, 1967.

6987 **CANCER COMPLICATING IDIOPATHIC MEGAESOPHAGUS.** *(Fr.)* Debray, C. (Bichat Hosp., Paris, France), J. Leymarios and J. P. Cuq. *Entret Bichat [Med]* pp. 449-454, 1967.

6988 **FOLLOW-UP STUDY OF 76 PATIENTS OPERATED ON FOR "ISOLATED" SLIDING HIATUS HERNIAS.** *(Ger.)* Escher, J. (Waid Munic. Hosp., Zurich, Switzerland), A. Lather, M. Landolt and E. Kaiser. *Praxis* 56(42):1426-1430, 1967.

6989 **DUPLICATION OF THE CERVICAL ESOPHAGUS.** *(Ger.)* Morger, R. (East Swiss Hosp. Infants Child., St. Gallen, Switzerland). *Schweiz Med Wschr* 98(3):86-88, 1968.

6990 **CARCINOMA OF THE ESOPHAGUS: A STUDY OF 238 CASES.** *(Turk.)* Dirvana, S. (Med. Fac., U. Istanbul, Turkey), H. K. Cilingiroglu, Y. Kefeli and N. Gürkan. *Turk Tip Cem Mec* 34(6):394-398, 1968.

6991 **STRANGULATED POSTTRAUMATIC DIAPHRAGMATIC HERNIA INVOLVING THE STOMACH AND COLON.** *(Sp.)* Durán Broqués, F. and A. Serradel Terreres. *Cir Ginec Urol* 22(4):479-484, 1968.

6992 **SPONTANEOUS RUPTURE OF THE ESOPHAGUS WITH GASTROINTESTINAL BLEEDING IN CALCAREOUS PANCREATITIS WITH DIFFUSE SPLENOPORTAL THROMBOSIS.** *(Fr.)* Rautureau, J. (Franco-Moslem Hosp., Bobigny, France), M. Benozio and J. Texier. *Gaz Med France* 75(17):3625-3630, 1968.

6993 **HOMOTRANSPLANTATION OF THE ESOPHAGUS.** *(Ger.)* Rainov, R. (4th Surg. Clin., Inst. Med., Sofia, Bulgaria). *Zbl Chir* 92(26a):1178-1183, 1967.

6994 **RARE DISORDERS OF THE DIAPHRAGM: CLINICAL AND RADIOLOGICAL ASPECTS OF THE HERNIA OF MORGAGNI-LARREY AND HERNIAS OF THE AORTOESOPHAGEAL HIATUS.** *(It.)* Tilli, R. (Inst. Gen. Surg. Clin., U. Florence, Italy), P. Gonnelli and D. Borrelli. *Nunt Radiol* 33(7):789-808, 1967.

6995 **THE ROLE OF SUBMUCOSAL ESOPHAGEAL TRANSECTION IN THE TREATMENT OF BLEEDING ESOPHAGEAL VARICES.** *(Ger.)* Gschnitzer, F. (Surg. Clin., U. Innsbruck, Austria). *Med Welt* 18(48):2909-2911, 1967.

6996 **THE DETERMINATION OF OPERABILITY IN CANCER OF THE ESOPHAGUS.** *(Ger.)* Krause, F. (Surg. Clin., U. Tubingen, Germany). *Med Welt* 18(47):2829-2835, 1967.

6997 **SURGICAL TECHNIQUES FOR PARTIAL INTRATHORACIC REPLACEMENT OF THE ESOPHAGUS.** *(Ger.)* Hannak, H. (Surg. Clin., U. Tubingen, Germany). *Med Welt* 18(47):2835-2839, 1967.

6998 **CLINICAL, PATHOGENETIC AND THERAPEUTIC PROBLEMS IN A CASE OF DYSPHAGIA PROBABLY DUE TO ESOPHAGEAL SCLERODERMA.** *(Fr.)* Leonard, P. (517 Brand-Whitlock Blvd., Brussels, Belgium). *Acta Gastroent Belg* 30(11):753-775, 1967.

6999 **INCARCERATED POSTTRAUMATIC DIAPHRA MATIC HERNIA. [CASE REPORT]** *(It.)* Scultetus, R. (S. Maria Nuova Hosp., Florence, Italy), F. Petrucci C. Massimo and G. Gallenga. *Osped Ital Chir* 18(4):343-347, 1968.

7000 **TRANSABDOMINAL REPAIR OF ESOPHAGEAL HIATAL HERNIA ANTERIOR TO THE ESOPHAGUS.** *(E.)* Hermann, R. E. (Cleveland Clin. Found., Ohio). *Surg Gynec Obstet* 126(4):844-847, 1968.

7001 **ESOPHAGEAL MANOMETRICS AS A GUIDE TO THERAPEUTIC DILATION IN ACHALASIA: A PRELIMINARY REPORT. [CASE REPORT]** *(E.)* Rakatansky, H. (Dept. Med., U. Chicago, Ill.). *Gastroint Endosc* 14(4):182-185, 1968.

7002 **MILD GASTRIC FUNDAL IRRADIATION IN THE TREATMENT OF PEPTIC ESOPHAGITIS.** *(E.)* Cooper, J. N. (Dept. Med., U. Chicago, Ill.), E. A. Gelzayd and J. B. Kirsner. *Gastroint Endosc* 14(4):222-224, 1968.

7003 **SEGMENTAL RESECTION FOR CARCINOMA OF THE ESOPHAGUS: REPORT OF A CASE WITH FIVE-YEAR SURVIVAL.** *(E.)* Hopkins, S. M. (St. John Hosp., Detroit, Mich.) and H. J. Vandenberg Jr. *Arch Surg* 96(6):936-938, 1968.

7004 **BLIND ESOPHAGEAL COIN REMOVAL USING A FOLEY CATHETER. [CASE REPORT]** *(E.)* Brown, L. P. (Francis E. Warren AFB Hosp., Cheyenne, Wyo.). *Arch Surg* 96(6):931-935, 1968.

7005 **CLINICAL STUDIES OF PARESTHESIA IN THE PHARYNX, LARYNX AND ESOPHAGUS.** *(E.)* Ohmori, K. (Jikei U. Sch. Med., Tokyo, Japan). *Jikei Med J* 14(1):3-4, 1967.

7006 **HIATUS HERNIA.** *(E.)* Walbaum, P. R. (Dept. Clin. Surg., U. Edinburgh, Scotland) and R. J. M. McCormack. *Scot Med J* 13(4):103-109, 1968.

See also: 6696,6724,6838,6847,6848,6905,6920,6941,6942,6943,6945,6946,6951,6960, 7046,7156,7470

STOMACH

(Gastric ulcer is placed with peptic ulcer)

007 THE NATURAL HISTORY OF ADVANCED GASTRIC CANCER. *(E.)* Moertel, C. G. (Mayo lin., Rochester, Minn.). *Surg Gynec Obstet* 126(4):1071-1074, 968.

retrospective study was made of 307 patients (219 males nd 88 females) seen during 1955-1957 with histologically roven inoperable or metastatic adenocarcinoma originating in ne stomach. The mean survival time was 17.4 months (median 1.5) from onset of symptoms, 12.0 months (median 5.0) from iagnosis of the primary lesion, and 7.5 months (median 4.0) om proof of incurability. The length of survival was not atistically different among males and females. The mean surival was 4.6 months (median 4.5) among those who were 20-9 yr old (8 patients), 5.8 months (median 4.5) among those ho were 40-59 yr old (108 patients), 8.9 months (median .0) among those who were 60-79 yr old (180 patients), and .1 months (median 2.0) among those who were 80 yr old or ver. The location of the primary lesion in the stomach had o influence on survival. Those with scirrhous cancer had a nger survival (mean 9.2 months, median 4.5) than did those ith ulcerative (mean 6.7 months, median 4.0) or polypoid sions (mean 5.7 months, median 4.0). Patients with less agressive forms of malignancies, as judged by a longer preceding linical course, seemed to have increased survival periods. The iean survival time of 163 patients who received no form of irgical palliation was 6.9 months (median 3.5); 29 patients ho had a bypass procedure and 39 who had a subtotal reection had mear survival times of 10.0 months (median 4.5 nd 6.0, resp.).

008 CARCINOMA OF THE STOMACH IN INCIPIENT PHASE: ITS HISTOGENESIS AND HISTOOGICAL APPEARANCES. *(E.)* Nakamura, K. (Cancer Inst., okyo, Japan), H. Sugano and K. Takagi. *Gann* 59(3):251-58, 1968.

he resected stomachs from 31 patients (43-79 yr old; 25 males, females) with stomach cancer revealed 33 foci of primary carinoma, all less than 5 mm in diameter. Based on a histological xamination, these lesions could be divided into 3 groups: 1) ne papillotubular type (11 cases), consisting of cylindrical cells orming large tubules and papillary infoldings, with nuclei in ne basal portion of the cell; 2) the tubular type (21 cases), onsisting of flat cylindrical or cuboidal cells forming small ubules, with nuclei located in the cell centers; and 3) the mucoellular type (1 case), consisting of round or ovoid cells with uclei pushed to the cell membrane (signet-ring cells). Twentyight of the foci (84.8%) arose from intestinal-type epithelium, (12.1%) from ordinary mucosa, and 1 (3.0%) represented an lcerogenic cancer. It is concluded that carcinoma of the stomch may originate either in epithelium of the intestinal type or 1 normal mucosa.

009 LOCAL GASTRIC HYPOTHERMIA INDUCED BY DIRECT PERFUSION OF THE GASTRIC UMEN: EXPERIMENTAL BASIS AND CLINICAL APPLICAION. *(E.)* Salmon, P. A. (Dept. Surg., U. Alberta, Edmonton, Canada). *Canad Med Ass J* 97(16):944-949, 1967.

reliminary experiments on the induction of local gastric hypohermia by direct perfusion of the gastric lumen (without the usual balloon) were performed in dogs to determine the competence of the pyloric sphincter and to select a perfusion solution suitable for humans. In the 1st experiment, fluid loss during the 4-hr period was minimal (60-120 ml in 4 anesthetized dogs and 100-160 ml in 3 unanesthetized dogs); the unanesthetized dogs showed little evidence of discomfort. As a result of experiments in which 4 anesthetized and 3 unanesthetized dogs were perfused with either Ringer's solution; 5% glucose, NH_4Cl and KCl; KCl, $NaHCO_3$, 5% glucose and NH_4Cl; 10% glucose; or glucose + NH_4Cl solution (5 or 10%), a perfusion solution containing 5% glucose, 80 mEq NH_4Cl, 10 mEq KCl, and approximately 30 mEq $NaHCO_3$ per liter was selected for human use. The technique was then tried in 4 fasting, locally anesthetized persons having normal gastrointestinal tracts and in 4 patients (25-63 yr old) having upper gastrointestinal or nasogastric hemorrhage. External heating sources were necessary to maintain normal body temperatures. In the normal subjects, serum electrolytes and blood glucose values remained essentially unchanged during the 6 to 8-hr period, fluid loss was 69-245 ml/hr, the plasma space was slightly expanded, and little discomfort was experienced. The technique was successful in 3 of the 4 patients with massive bleeding. The death of the 4th patient, a 63-yr-old man with a hemorrhagic diathesis, cannot fairly be attributed to failure of the technique, since many hemorrhagic sites were outside the stomach and away from the effectively cooled area.

7010 ANTRECTOMY AFTER STAINING WITH CONGO RED AND TRANSILLUMINATION (MOE'S TEST). *(E.)* Hedenstedt, S. (Nacka Hosp., Sweden), A. Lindgren, G. Lundquist and B. Robertsson. *Acta Chir Scand* 133(7):539-543, 1967.

In an attempt to achieve a functionally total antrectomy (elimination of all the gastrin-producing tissue) while preserving as much of the stomach as possible, the boundary between the antrum and corpus was determined preoperatively by means of a modified Moe's test (transillumination of the stomach following staining of the mucosa by insufflation of Congo Red via a sprayer-equipped gastric catheter) in 24 patients (18 with duodenal and 6 with gastric ulcers). Results in these patients were compared with those in 18 patients (10 with duodenal and 8 with gastric ulcers) resected without the benefit of staining techniques by examination of 5 biopsy specimens from each patient. All 42 patients also received jejunal transposition and 37 of 42 (including 27 of the 28 with duodenal ulcers) received selective vagotomy. In patients subjected to the Moe procedure, antrectomy was carried out 2 cm oral to the visible antrum-corpus boundary. The gastrectomies with this procedure tended to be smaller in extent and more oblique, while those in the control group were more abruptly transverse and greater in extent (50% or more). Histological examination of biopsy specimens of the residual stomach showed that the antrectomy was successful along the lesser curvature in 75% and 83% of those operated on with and without Moe staining, resp. Along the greater curvature, antrectomy was complete in all cases at the anterior biopsy site but was complete in only 83% of those stained by Moe's method at the posterior site (100% in controls). Of the 9 failures, 7 were in patients with gastric ulcers. The distinction between antral and duodenal tissue was successful in 88% and 72% of those operated on with and without the benefit of staining, resp. It is felt that the Moe procedure is preferable to that of Bergström and Broomé in that no gastrostomy is required. Proper application of staining techniques for antral delimitation offers the advantage that more of the stomach can be preserved than with the conventional blind technique, resulting in a lower incidence of post-gastrectomy symptoms.

7011 **GASTRIC CARCINOIDS.** *(Rus.)* Markov, I. N. (Central Inst. Postgrad. Med., Moscow, USSR), T. A. Tret'iakova and V. I. Iakushin. *Vestn Khir Grekov* 101(10):11-16, 1968.

Case reports are presented for 4 patients (2 men and 2 women, aged 32-65 yr) with stomach carcinoids. None of these patients had carcinoid syndrome; the only clinical symptom common to all 4 cases was epigastric pain. Severe bleeding occurred in 2 cases and was responsible for the death of 1 patient before surgery. The tumors, which were only diagnosed histologically, were all located in the antrum, in the greater curvature in 3 cases and in the lesser curvature in 1.

7012 **HISTOPATHOLOGICAL AND CLINICAL STUDY OF PYLORIC HYPERTROPHY IN INFANTS.** *(It.)* Francucci, G. (Inst. Gen. Surg. Clin., U. Bologna, Italy). *Gazz Int Med Chir* 72(24-bis, part 1):3156-3172, 1967.

Histological findings in tissue removed from infants, aged 15 days to 4 months, with pyloric hypertrophy included mucosal hypertrophy, numerous glands in the submucosa, and a very thick layer of muscle. The appearance of ganglionic cells in Auerbach's plexus varied from completely developed cells to neuroblast types which were embryonic in nature. Cell changes included granular protoplasm, complete disappearance of some nuclei, and some vacuolated nuclei surrounded by thin circles of cytoplasm. In some regions these nerve cells had degenerated completely and disappeared. Bundles of nerve fibers were found alone or with groups of nerve cells. In some cases the nerve cells were dissociated from broken and discontinuous nerve fibers. These findings indicate that pyloric hypertrophy is not due to underdevelopment of Auerbach's plexus, but to degeneration of nerve cells or fibers.

7013 **MUCOSAL EFFECTS OF ASPIRIN AND ACETAMINOPHEN: REPORT OF A CONTROLLED GASTROSCOPIC STUDY.** *(E.)* Vickers, F. N. (Baptist Hosp., Pensacola, Fla.). *Gastroint Endosc* 14(2):94-99, 1967.

The effects of aspirin and acetaminophen introduced directly into the fasted stomach as a single dose of 650 mg, on the gastric mucosa were studied by means of a Hirschowitz fiberscope in men (33) and women (37) with various gastrointestinal disorders. The observation periods (15-60 min, average 25 min) were separated by a 30-min rest period and, in most cases, acetaminophen was administered first. Positive reactions to aspirin, as measured by erythematous lesions or petechiae on the mucosa or gross bleeding, were found in 12 patients (17%); there were no reactions to acetaminophen. Of 6 patients also given a single i.v. dose (150 mg) of sodium salicylate, two, known to have reacted to orally administered aspirin, showed a positive gastric mucosal reaction.

7014 **VAGOTOMY, GASTROENTEROSTOMY AND EXPERIMENTAL GASTRIC CANCER.** *(E.)* Morgenstern, L. (Cedars-Sinai Med. Ctr., Los Angeles, Calif.). *Arch Surg* 96(6):920-923, 1968.

To determine whether artificially induced hypochlorhydria (b vagotomy and gastroenterostomy) would enhance the effect of known carcinogens in the stomach, young adult rats (150 were divided into 3 groups: group 1 (100 rats) had 20-methy cholanthrene (20-MC)-impregnated cotton threads implanted into the glandular portion of the intact stomach near the mo dependent part of the greater curvature; group 2 (109 rats) had an anterior gastrojejunostomy created near the most dependent portion of the stomach and implantation of 20-MC threads as in group 1; group 3 (133 rats) were treated exactl as in group 2, with the addition of transabdominal vagotomy Animals were observed over an 18-month period. The tumor incidence was 44% in group 1, 64% in group 2, and 75% in group 3. The tumors involved the glandular portion of the stomach, were more frequent in the vicinity of the gastroenterostomy, and were either carcinomas, sarcomas, or mixed The results of these experiments indicate that vagotomy and gastroenterostomy enhance experimental carcinogenesis in the rat stomach.

7015 **GASTRIC NEOPLASIA AND BLOOD GROUPS** *(Sp.)* Zacnich, J. (2nd of May Hosp., Lima, Pe C. Valdivia, L. Sanchez S., M. Fernandez L., V. Gamarra and V. Gonzales. *Acad Peru Cir* 20(1):51-54, 1967.

In a hospital population of 1053 males, the ratio of O to A blood groups was 3.9:1. Among 54 patients with stomach cancer this ratio was 2.83:1 and among 126 patients with pe ulcers, 3.6:1. Thus, the incidence of stomach cancer among patients with blood group A was 1.35 times that among thos with blood group O, and the incidence of peptic ulcer among patients with blood group A was only 1.08 times that of patients with blood group O. These ratios are compared to oth found in the literature, and it is concluded that patients with blood group A are preferentially affected by gastric cancer.

7016 **HISTOLOGICAL FEATURES AND FUNCTIO BEHAVIOR OF THE GASTRIC MUCOSA IN PATIENTS WITH CHRONIC LIVER DISEASE.** *(It.)* Allegr G. C. (Maggiore Hosp., Novara, Italy) and S. Maduli. *Arch I Mal Appar Dig* 34(3):211-245, 1967.

Biopsied mucosa from the gastric fundus was examined histologically and gastric secretion was studied by Lambling's met in 50 patients, aged 23-71 yr, with chronic liver disease; 41 these patients had histories of alcoholism. Changes were fou in the gastric mucosa in 46 cases (92%). They included supe ficial gastritis in 38 patients, preatrophic gastritis in 4, and atrophic gastritis in 4. Lesions were most often found in the neck region of the glands. The severest gastric lesions were seen in patients with severe, rapidly progressing forms of chr liver disease. A study of gastric secretion after histamine stin lation in 32 patients with mild and moderate superficial gastr showed reductions in the total volume of secretion in 28 case the volume of primary acid secretion in 26, and the volume primary alkaline secretion in 23. Pathological values for buff ing capacity and free HCl were found in 25 cases. Free acid flow was reduced in 21 of these patients. Similar, but more pronounced changes in gastric secretion were found in patients with severe superficial, preatrophic, and atrophic gastritis. On the other hand, the volume of primary alkaline secretion was normal in 6 and free acid flow was normal in 4 of 10 patients who had peptic ulcers associated with chronic liver disease. Histological changes in the gastric mucosa of these ulcer patients were essentially the same as those in patients with chronic liver diseases alone.

7017 **MITOCHONDRIAL CHANGES IN GASTRIC MUCOSA AND LIVER UNDER NEUROGENIC DYSTROPHIES.** *(E.)* Anichkov, S. V. (Inst. Exp. Med., Leningrad, USSR), I. S. Zavodskaya, E. V. Moreva and V. V. Korkhov. *Pharmacology* 1(1):2-7, 1968.

Ultrastructural and functional alterations were observed in the liver mitochondria of immobilized rats electrically stimulated for 3 hr via electrodes in their forepaws, and in both gastric and liver mitochondria from rabbits electrically stimulated for 15 min via electrodes implanted in the posterior hypothalamus (to provoke dystrophies of central origin) or traumatized for 15 min by the application of Pean's forceps to the duodenum (inducing reflex dystrophies). The mitochondrial changes in rat liver cells included swelling, destruction of some cristae and decrease in the electron density of the matrix. The optical density of the mitochondria was reduced from a control level of 0.60 ± 0.1 in intact rats to 0.42 ± 0.06 in the irritated rats. The contractile response of the mitochondria to addition of ATP (0.05 ml 0.005 M) was also lower in the experimental animals. Fragmentation of the granular endoplasmic reticulum, decreased numbers of ribosomes and polysomes, and the presence of optically empty vacuoles surrounded by smooth membranes or of electron-dense vesicles surrounded by smooth membranes were also observed in rats 3 hr after irritation. Duodenal traumatization of rabbits produced a lower optical density in the gastric (0.4 ± 0.18) and liver (0.56 ± 0.23) mitochondria than was observed in intact control rabbits (0.67 ± 0.09 and 0.69 ± 0.17, resp.). The contractile response of the mitochondria to ATP was markedly lower in the irritated animals. Similar results were observed in rabbits subjected to stimulation of the posterior hypothalamus, but the decreases in optical density and contractility were less than in animals subjected to duodenal irritation. The observed modifications in the mitochondria are considered to be one of the earliest symptoms of dystrophic processes.

7018 **HISTOPATHOLOGICAL STUDY ON EARLY CARCINOMA OF THE STOMACH: SOME CONSIDERATIONS ON THE ULCER-CANCER BY ANALYSIS OF 144 FOCI OF THE SUPERFICIAL SPREADING CARCINOMAS.** *(E.)* Nakamura, K. (Cancer Inst., Tokyo, Japan), H. Sugano, K. Takagi and A. Fuchigami. *Gann* 58(4):377-387, 1967.

In a series of 1038 patients treated by gastrectomy at the Cancer Institute Hospital during the period Jan. 1964-Dec. 1966, 136 revealed 144 foci of superficial carcinoma (most in patients 40-60 yr old) which were classified into 5 groups on the basis of the largest diameter of the lesion and compared with respect to gross appearance, ulcerative change in the cancerous area, and histological type. The superficial carcinomas exhibited a tendency to undergo erosion or ulceration as their diameter increased; 81.6% of lesions with a largest diameter over 41 mm were accompanied by ulceration. However, since secondary ulceration occurs, histologic criteria may be inadequate to diagnose true ulcer-cancer. The only valid histologic criterion of ulcer-cancer is a minute carcinoma developing within the regenerating epithelium of a healed ulcer; such an ulcer-cancer occurred in only 1 of the 144 superficial carcinomas. With regard to histological type, the undifferentiated carcinomas (mucocellular, scirrhous) were more often accompanied by ulcerative lesions (87.3%) than the differentiated types, represented by tubular and papillotubular forms (26.9%), regardless of the size of the carcinoma. Ulcer-cancer also occurred more frequently in undifferentiated (52.7%) than in differentiated (19.1%) types. It is concluded that the proportion of ulcer-cancer in carcinomas of the stomach is probably only about 1% and maximally about 15%.

7019 **THE CONTENT OF LOW-MOLECULAR-WEIGHT NITROGEN COMPOUNDS IN THE GASTRIC JUICE OF NORMAL SUBJECTS AND PATIENTS WITH GASTRIC DISEASES.** *(Pol.)* Iwańska, J. (2nd Cent. Clin. Hosp., Acad. Milit. Med., Warsaw, Poland), D. Kruze and S. Bober. *Pol Arch Med Wewnet* 39(5):617-622, 1967.

The pH of the gastric juice and the excretion of ammonia, amines,and α-amino-nitrogen were studied in 17 normal subjects, 21 patients with gastric ulcers, 37 with duodenal ulcers, 16 with gastritis + cholelithiasis, 17 with gastric cancer, and 6 with achlorhydric gastritis. The average pH was 6.2 in anacid gastritis compared to 4.8 in stomach cancer or gastritis + cholelithiasis, 2.7-3.0 in peptic ulcer, and 2.2 in controls. The average ammonia excretion (mg% N_2) was 7.7-7.9 in patients with stomach cancer or anacid gastritis, 6.8 in gastritis + cholelithiasis, 3.9-4.1 in peptic ulcer, and 2.6 in controls. The average α-amino-N_2 excretion (mg%) was 8.6 in stomach cancer, 3.6-3.7 in patients with anacid gastritis or gastric ulcers, and 2.2-2.4 in all others. It is concluded that there is a statistically significant increase in α-amino-N_2 excretion in patients with gastric cancer compared to both peptic ulcer patients and controls, and that there is also a statistically significant difference between controls and patients with gastric ulcers.

7020 **RADIOLOGICAL AND CLINICAL CONSIDERATIONS ON GIANT GASTRIC NICHES.** *(Rum.)* Jocu, I. (Dr. I. Cantacuzino Hosp., Bucharest, Romania), V. Sava, M. Lungeanu and R. I. Nitescu. *Med Intern (Bucur)* 19(9):1123-1135, 1967.

Detailed clinical and radiological findings are presented for 28 patients (17 men and 11 women, 42-81 yr old) in whom radiology revealed giant gastric niches 3-6 cm in diameter. The average diameter was 4.5 cm for the 9 lesions eventually shown to be malignant and 3.9 cm for the 19 which were benign. Malignant lesions were found in 8 men and 1 woman. Most of the niches (25 of 28, including 7 of 9 malignancies) were located on the lesser curvature, and the duration of symptoms varied from 2 months to 14 yr. The principal symptom was epigastric pain (27 cases), and only 2 patients (one with cancer and one with a benign ulcer) had demonstrable bleeding. A correct diagnosis was arrived at in all cases by a combination of clinical, radiological and laboratory studies. However, only 4 of the 9 malignant ulcerations were diagnosed as such on the basis of the clinical and radiological findings alone, the key diagnostic information having beeen provided in the other 5 cases by the lack of response to ulcer therapy. Treatment consisting of rest, diet, alkalies, antispasmodics, sedatives and vitamins was given to 17 patients for a trial period of 3-4 weeks; 12 improved and were confirmed as having benign lesions, while 5 with malignancies showed no response. It is emphasized that only biopsy can provide a definite diagnosis of the benign or malignant nature of a giant ulceration in some cases.

7021 **MORPHOLOGICAL CHANGES IN THE LIVER IN PATIENTS WITH CANCER OF THE STOMACH.** *(Pol.)* Smolak, K. (Inst. Oncol., Krakow, Poland), A. Górniak and A. Urban. *Pat Pol* 18(4):517-525, 1967.

Liver biopsy specimens taken from 110 patients (78 men and 32 women, 19-76 yr old, average 55 yr) during surgery for gastric carcinoma revealed nonspecific reactive hepatitis in 63.6% and more severe changes (interstitial inflammation and diffuse fatty degeneration) in 13.6%. The severity of the hepatic changes could not be correlated with age, and seemed to depend mainly on the rate of development of the neoplastic process. Thus, the most severe changes were seen in only 4 of 46 patients with a duration of symptoms of more than 9 months, compared to 11 of 64 patients with symptoms for less than 9 months. Similarly, severe hepatic changes were seen in 12 of the 72 patients with disseminated carcinoma compared to only 3 of 38 patients in whom partial gastrectomy was still possible.

7022 **THE ETIOLOGY OF STOMACH CANCER. I. EXPERIMENTS ON FEEDING AND INJECTING HEATED PORK FAT.** *(Ger.)* Schmidt, F. (Res. Inst. Exp. Oncol., German Acad. Sci., Potsdam-Rehbrücke), J. Proll and W. Fritz. *Mitteilungsdienst* 5(1):6-14, 1968.

Wistar rats were divided into 3 groups and fed 1) a diet containing 15% pork fat which had been heated to 220-230C for 2 hr, 2) the same diet containing unheated pork fat, and 3) a diet without pork fat. A statistically significant increase in gastric papillomas occurred in animals fed heated pork fat. They also developed hemorrhagic gastritis and stomach ulcers. Since no aromatic hydrocarbons were detected in fluorescence spectra of commercial or heated fat and since rats fed the heated fat developed only benign tumors, it is concluded that no carcinogens are present in the fat but that heating produces cocarcinogens. In addition, 100 rats were given 0.1 or 0.5 ml heated pork fat s.c. None of these rats developed tumors at the site of injection.

7023 **THE ETIOLOGY OF STOMACH CANCER. II. THE SIGNIFICANCE OF CARCINOGENIC HYDROCARBONS IN FOODS.** *(Ger.)* Schmidt, F. (Res. Inst. Exp. Oncol., German Acad. Sci., Potsdam-Rehbrücke) and W. Fritz. *Mitteilungsdienst* 5(1):15-30, 1968.

Analysis of foods with UV and fluorescence spectrophotometry showed that almost all foods of plant origin contain 3,4-benzpyrene and a large number of other carcinogenic hydrocarbons while animal products, such as heated and unheated pork fat, do not. Products containing these polycyclic hydrocarbons included rapeseed oil, sunflower seed oil, 4 brands of margarine, mayonnaise, flour and bread, barley, malt, malt coffee, and coffee beans. Heating, roasting, and baking did not appreciably change the content of aromatic hydrocarbons and, in the case of rapeseed oil, reduced it. A large proportion of the carcinogens were eliminated from vegetable oils by refining. Although malt coffee contained a larger amount of benzpyrene than coffee beans, the aqueous extract from coffee beans contained more benzpyrene than that from malt coffee. These findings indicate that most carcinogenic hydrocarbons ingested by man come from foods of plant origin and are more or less independent of the way in which these foods are prepared.

7024 **EXPERIMENTAL STUDY ON THE CLOSURE OF GASTRIC DEFECTS BY A STRIP OF SMALL INTESTINE WITH A MESENTERIC PEDICLE.** *(Rus.)* Syrbu, I. F. (N. I. Pirogov Med. Inst., Odessa, USSR). *Eksp Khir Anest* 13(2):19-22, 1968.

Segments of small intestine 6-10 cm in length with mesenteric pedicles were used to close gastric defects in 35 dogs which were then observed for 1-12 months. The graft adhered to the edges of the gastric wall in all cases, without complications. In time, the edges of the graft became nearly indistinguishable on the serosal side, while the mucosa continued to differ both in color and in surface texture from that of the stomach. No peptic ulcers or other macro- or microscopic changes were note on the surface of the graft. During the first 15 days after the operation the graft received its blood supply from the mesenteric pedicle; later, it was able to survive even if the pedicle wa cut. Graft mucosa removed 5 months after the operation and grafted onto a skin wound, continued to secrete alkaline intestinal fluid. This probably explains the resistance of the graft to the action of gastric juice.

7025 **IS GASTRITIS A DUFFUSE CONDITION?** *(Ger.)* Wolff, G. (Inst. Cancer Res., German Acad Sci., Berlin, Germany) and H. Schwarz. *Gastroenterologia (Bas* 107(6):389-394, 1967.

A Henning-Heinkel tube was used to obtain 2-6 samples of gastric mucosa from different sites in the anterior and posterior walls of the stomach of 1132 normal subjects and patients with gastric disease. The histological diagnosis of these multiple specimens (total of 3882) agreed in 95.8% of the cases, while in 4.2% the findings showed a mixture of normal mucosa, superficial gastritis, and chronic gastritis. From the frequency of agreement it was estimated that 1-2% of the total surface of the stomach has mucosa with a different structure from that of the rest of the stomach. This structurally different mucosa is found in at least half of all stomachs. About 50% or less of all stomachs thus have a completely homogeneous mucosa. The implications of this finding for the biopsy diagnosis of gastric disease are discussed.

7026 **CARCINOMA OF THE GASTRIC STUMP AFTE SURGERY FOR BENIGN STOMACH DISEASES** *(Ger.)* Kootz, F. (Surg. Clin., U. Frankfurt/Main, Germany). *Bruns Beitr Klin Chir* 215(3):275-294, 1967.

Between 1945 and 1964, 25 of 4245 patients with stomach diseases (0.6%) developed carcinomas of the gastric stump after gastrectomy. This group consisted of 22 men and 3 women, aged 19-74 yr, who developed symptoms 5.5-39 yr after surgery. One patient died of peritonitis caused by perforation of an adenocarcinoma in the duodenal stump. Gastrectomies were originally performed for duodenal ulcers in 10 cases, stomach ulcers in 9, pyloric stenosis in 2, erosive gastritis in 1, and erosion produced by ingestion of HCl in 1. Billroth II gastrectomies had been performed in 16 cases. The ratio of

en to women (22:3) was much higher than that among all
rgically treated ulcer patients (11.7). Only 4 of these 25
tients sought treatment within the first 3 months after symp-
ms appeared and 5 waited for 3 yr. Radiological findings
ere positive in 18 cases and gastroscopic findings in 8. The
mor was located in the anastomosis in 7 cases, the entire
ump in 6, the duodenal stump in 1, the antrum in 4, the
rdia-fundus region in 4, the lesser curvature in 2, and the
eater curvature in 1. These tumors consisted of 15 adenocar-
nomas, 2 medullary carcinomas, and 2 solid carcinomas; no
stological studies were made in 6 cases. The carcinoma was
nited to the gastric stump in 6 cases and to the gastric stump
id regional lymph nodes in 7 cases. Partial or total gastrec-
mies were performed in 14 cases and 10 patients were found
be inoperable at laparotomy. One patient died before sur-
ry. Of the 3 patients still alive at the end of 1966, only 1
id survived for more than 5 yr.

)27 THE SIGNIFICANCE OF ABNORMAL BACTE-
RIAL PROLIFERATION IN THE GASTROIN-
ESTINAL TRACT AFTER GASTRIC SURGERY. *(E.)*

ellipiani, A. W. (Royal Infirm., Edinburgh, Scotland) and
. H. Girdwood. *Scand J. Gastroent* 2(3):161-168, 1967.

ne juices of the stomach or stomach remnant and of the jeju-
ım of 21 patients who had undergone gastroenterostomy
id vagotomy and 26 patients who had undergone partial gas-
ectomy with a Billroth II anastomosis were studied for the
esence of fecal type organisms *(Enterobacteriaceae, Strep-
coccus faecalis,* and *Clostridium welchii).* In 1/3 of the gas-
ic aspirates and half the jejunal aspirates of all patients, re-
ırdless of the type of previous surgery, the counts were clas-
fied as abnormal (2×10^4-3×10^9 viable organisms/ml).
oliform organisms were the most common, with *E. coli* the
ost prominent. There was no correlation between the pre-
nce of steatorrhea, diarrhea, and impaired absorption of
tamin B_{12} and the presence of an abnormal number of or-
nisms. Although few patients with free acid in the stomach
ter operation had an abnormal gastrointestinal flora, half of
ıe patients with achlorhydria also had normal bacteriological
ndings. It is felt that mechanisms other than gastric acidity
perate to prevent abnormal bacterial proliferation.

028 THE INCIDENCE OF GASTRITIS IN KOREANS:
A BIOPSY STUDY OF THE STOMACH IN PA-
IENTS WITH DYSPEPSIA AND NEGATIVE UPPER GAS-
ROINTESTINAL RADIOLOGY. *(Kor.)*

Chung, J. M.
Kyungpook Natl. U., Sch. Med., Taegu, Korea), Y. C. Kim,
. K. Yoon, Y. J. Choi, Y. S. Han and Y. K. Park. *Korean
Intern Med* 10(11):719-727, 1967.

astric biopsies (total of 177) were performed in 90 Korean
itients (65 men and 25 women, 11-70 yr old) with dyspepsia
id negative upper gastrointestinal series. The incidence of
ıronic gastritis was 60.0% (superficial gastritis in 40.0%, atro-
ıic gastritis in 18.9%, and borderline forms in 1.1%), while
)% had a normal gastric mucosa. The character and frequency
the dyspeptic symptoms in the normal group and the chro-
c gastritis group were similar; the most frequent complaints
ere epigastric pain, indigestion, and epigastric fullness, noted
73%, 13% and 9% of the cases, resp. Achlorhydria was
und in 12% of histologically normal patients and 41% of
ıose with gastritis. Although the Japanese diet is known to
less irritative than that in Korea, the incidence of chronic
and atrophic gastritis in this study was lower than that reported for Japan. The incidence of chronic gastritis could not be correlated with the intake of pepper or hot drinks, the amount of food eaten at each meal, alcohol consumption or smoking habits. This suggests that dietary habits are not the most significant factor in chronic gastritis.

7029 THE PROBABILITY OF SURVIVAL OF PATIENTS WITH CANCER OF THE STOMACH.

(Pol.) Oszacki, J. (2nd Surg. Clin., Acad. Med. Krakow, Poland) and J. Kulpa. *Pol Przegl Chir* 39(11):1179-1182, 1967.

A statistical analysis of the survival rates in 2 groups of patients gastrectomized for carcinoma of the stomach at least 5 yr before (225 with lymph-node metastases at the time of surgery and 151 without) showed that the probability of surviving from the 4th to the 5th yr is independent of the condition of the lymph nodes, although the presence of metastases has a significant effect on the mortality rates during the first 4 yr. Of an original group of 442 patients (266 with lymph-node metastases and 176 without), 66 (41 with metastases and 25 without) died immediately postoperatively, 134 (104 and 30) died in the 1st yr, 86 (61 and 25) died in the 2nd yr, 38 (27 and 11) died in the 3rd yr, 24 (15 and 9) died in the 4th yr, and 3 (1 with metastases and 2 without) died in the 5th yr; the 5-yr survival (in terms of those surviving the operation itself) was thus 7.6% and 49%, resp., while the 4-yr survival was 8% and 50.5% and the 1-yr survival was 54% and 80%, resp. Mathematical equations are developed for estimating the survival rate of patients with and without lymph-node metastases who survive gastrectomy for stomach cancer: $S = S_o e^{-ct}$ where S and S_o are the number of survivors at time t (0-4 yr) and immediately after the operation, resp., and c is a constant which = 0.6351 for patients with lymph-node metastases and 0.1763 for those without.

7030 EXPERIMENTAL GASTRITIS IN THE DOG, I. PRODUCTION OF ATROPHIC GASTRITIS AND ANTIBODIES TO PARIETAL CELLS. *(E.)*

Krohn, K. (Child. Hosp., U. Helsinki, Finland). *Ann Med Exp Fenn* 46(3):249-258, 1968.

Dogs immunized with autologous and homologous gastric juice or gastric mucosa were observed for pathologic changes in the gastric mucosa and for the occurrence of circulating antibodies to parietal cells. All dogs immunized with any of the different gastric preparations developed inflammatory and degenerative changes in their gastric mucosa during the course of immunization. In the early stages of immunization only slight, superficial inflammation was observed, whereas by 3 weeks -2 months after the 1 st injection of antigen inflammation and degenerative changes (cheifly in parietal cells) occurred. Gastric biopsies taken 2-8 months after the 1st injection revealed total atrophy of the gastric glands in some dogs. Immunofluorescence studies with γ-globulins following fractionation with ammonium sulfate demonstrated circulating parietal cell antibodies simultaneously with or even prior to the appearance of gastric inflammation. All parietal cell antibody activity could be abolished by absorption with dried gastric juice or gastric mucosa homogenate. Immunoelectrophoresis demonstrated that the early antibodies occurred in the IgM fractions, the later ones in fractions containing IgG. These antibodies also reacted against autologous gastric mucosa from the immunized dogs, but no bound IgG could be demonstrated in the gastric mucosa of the immunized dogs. Fourteen of 19 immunized dogs showed a delayed skin reaction to the antigen used in immunization; the occurrence of the positive reaction correlated closely with the appearance of inflammatory lesions in the gastric mucosa. When immunization was discontinued, the parietal cell antibodies disappeared, complete healing of the gastric lesions occurred, and the skin test became negative. These findings support the view that gastric damage in dogs is caused by the action of sensitized cells and not by circulating antibodies.

7031 **EXPERIMENTAL GASTRITIS IN THE DOG, II. PRODUCTION OF ANTIBODIES TO A GASTRIC B_{12}-BINDING AUTO-ANTIGEN.** *(E.)* Krohn, K. (Child. Hosp., U. Helsinki, Finland). *Ann Med Exp Fenn* 46(3):259-272, 1968.

In a study of the antigenic properties of canine gastric B_{12}-binders, gel diffusion techniques combined with autoradiography demonstrated the presence of antibodies to a B_{12}-binding protein in dogs immunized with autologous and homologous gastric juice or gastric mucosa. Inflammatory lesions in the gastric mucosa in the form of diffuse gastritis always preceded the occurrence of antibodies. These antibodies did not react with B_{12}-binders in any dog tissues except the gastric mucosa, duodenum and jejunum. To further characterize the B_{12}-binders in different tissues, fractionations were performed by gel filtration and ion-exchange chromatography. CM cellulose chromatography demonstrated the occurence of a B_{12}-binding fraction, B_{12}-binder I, in all dog tissues examined. Gastric juice, gastric mucosa, duodenum, jejunum, lung, spleen and muscle were also shown to contain another fraction, B_{12}-binder II. Dogs immunized with any of the gastric juice or gastric mucosa preparations produced antibodies to the B_{12}-binder II from gastric mucosa, gastric juice, duodenum and jejunum, but not against the same binder from lung, spleen or muscle, and they did not produce antibodies against B_{12}-binder I from a variety of dog tissues, including gastrointestinal tissues. Rabbit antisera prepared against dog gastric juice reacted against B_{12}-binder I and B_{12}-binder II from both gastrointestinal and non-gastrointestinal tissues. It appears that the gastrointestinal B_{12}-binder II differs from the non-gastrointestinal B_{12}-binder II in at least 1 antigenic site; this site acts as an autoantigen. Schilling tests demonstrated lower absorption of vitamin B_{12} in immunized than in nonimmunized dogs, and in both cases it was lower than in man. In 2 pernicious anemia patients dog gastric juice did not promote absorption of vitamin B_{12}, but autologous antisera to dog gastric juice partly inhibited the effect of human gastric juice on vitamin B_{12} absorption.

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7043 **OBSTRUCTING GIANT TRICHOBEZOAR INVOLVING STOMACH AND DUODENAL BULB.** *(E.)* Small, A. (Jewish Hosp. Med. Ctr., Brooklyn, N. Y.), M. Muehlbauer and S. Kleinhaus. *Amer J Gastroent* 50(4):297-302, 1968.

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7071 **SYNCHRONOUS OCCURRENCE OF GASTRIC CARCINOMA, LEIOMYOSARCOMA, AND DUODENAL ULCER: REPORT OF A CASE.** *(E.)* Fonkalsrud, E. W. (UCLA Sch. Med., Los Angeles, Calif.) and W. F. Barker. *Arch Surg* 96(6):915-919, 1968.

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7073 **FINE STRUCTURE OF INTESTINAL METAPLASIA AND ADENOCARCINOMA OF THE HUMAN STOMACH.** *(E.)* Goldman, H. (Beth Israel Hosp., Boston, Mass.) and S. Ming. *Lab Invest* 18(2):203-210, 1968

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7076 **THE DIAGNOSIS OF MALIGNANT TUMORS OF THE GASTRIC AIR POCKET IN DAILY PRACTICE.** *(Fr.)* De Keersmaeker, J. (65 Ave. Coghen, Brussels, Belgium). *Louvain Med* 87(5):345-352, 1968.

See also: 6673,6740,6755,6766,6838,6843,6846,6849,6850,6859,6860,6891,6904,6912, 6918,6919,6920,6927,6936,6941,6946,6950,6951,6956,6958,7126,7129,7146, 7147,7171,7249,7274.7329

7077 **STUDIES OF INTESTINAL HEALING. I. PRELIMINARY STUDIES OF THE MECHANISM OF HEALING OF THE EVERTING INTESTINAL ANASTOMOSIS.** *(E.)* Ravitch, M. M. (Johns Hopkins U. Sch. Med., Baltimore, Md.), A. Rivarola and J. Vangrov. *John Hopkins Med J* 121(6): 343-348, 1967.

The routine success of through-and-through staple closure of the cut ends of the duodenum and stomach, reported previously, prompted on investigation of mucosa-to-mucosa healing in 3 groups of dogs. In group I (41 dogs), 11 dogs were given end-to-end everting anastomoses of the small bowel, closed by a single row of fine staples parallel to the wound edges (inserted with an ASTS or SK instrument); in 16 dogs, the anastomoses were constructed in the same manner and subsequently wrapped in polyethylene film; in 14 dogs, the omentum was resected and anastomosis performed either as above (6 dogs) or with 2 rows of staples (8 dogs). In group II (15 dogs), everting end-to-end anastomosis was performed with 1 layer of through-and-through silk mattress sutures parallel to the wound edges; 6 animals served as controls and 9 had the anastomosis wrapped with polyethylene film. In group III (9 dogs), inverting anastomoses were constructed with Lembert silk sutures and wrapped with polyethylene film. Of the 11 control animals in group I, none died and none developed peritonitis or abscesses. In 1 dog sacrificed at 15 days, an anastomotic dehiscence was noted, but was firmly sealed by adherence to the abdominal wall. In the 14 other dogs with everting anastomoses, using 1 or 2 rows of staples with omental resection, 13 survived and 1 developed an anastomotic leak and died; 2 other animals had an anastomotic dehiscence completely walled off by adhesions. All 16 animals in which everting anastomoses were wrapped in polyethylene died of peritonitis or abscesses from gross leaks. Of the 6 control dogs in group II with everting mattress sutures, 5 survived and 1 developed peritonitis and died, whereas the 9 subjected to polyethylene wrapping of the anastomosis all died from abscesses or peritonitis. In group III, 3 of the 9 animals with inverting Lembert silk sutures and polyethylene wrapping also died from leaks and peritonitis. These experiments indicate that the bowel will heal satisfactorily mucosa-to-mucosa, but that healing is dependent upon adhesion formation, which is prevented by wrapping the anastomosis in polyethylene.

7078 **EXPERIMENTAL STUDY OF THE FUNCTION AND LONG-TERM DEVELOPMENT OF ANTIPERISTALTIC ILEAL LOOPS (WITH PARTICULAR REFERENCE TO THE PROBLEM OF FECAL CONTINENCE).** *(It.)* Pisano, M. (Inst. Gen. Clin. Surg., U. Cagliari, Italy), R. Milani, A. Revignas, L. Di Martino, G. Peretti and F. Oggianu. *Gazz Int Med Chir* 73(17):1479-1503, 1968.

Ileal segments ranging in length from 30-75 cm were reversed in 16 dogs which had undergone rectocolectomies. Animals in which 65, 70, and 75 cm of the ileum had been reversed died within 3 days after surgery with peritonitis, dehiscence of the proximal suture, and torsion of the reversed segment along with a contiguous portion of the ileum. Dogs in which segments of 45 cm or less had been reversed showed no disturbances in canalization while those with reversed segments of 45-65 cm developed disturbances in canalization between 10 and 18 months after surgery. Intestinal transit was significantly slowed by reversal of an ileal segment. This effect was most apparent with reversal of long segments, and is apparently due to partial retention of the reversed intestine's original polarity, a change in its contractile activity, incomplete obstruction of the passage of the intestinal contents, and the development of a kind of neocolon by dilatation and hypertrophy of the reversed ileum and the part of the ileum above it. Unless they showed signs of partial intestinal obstruction, the dogs' stools and the number of bowel movements were within normal limits. Possible clinical applications of this procedure are discussed.

7079 **THE EFFECTS OF ESSENTIAL FATTY ACID DEFICIENCY ON THE ULTRASTRUCTURE AND FUNCTIONAL CAPACITY OF THE JEJUNAL EPITHELIUM.** *(E.)* Snipes, R. L. (Div. Biol. Med. Sci., Brown U., Providence, R. I.). *Lab Invest* 18(2):179-189, 1968.

The structural and functional characteristics of the jejunum were investigated in 170 rats and 110 BUA mice fed diets deficient in essential fatty acids. No species variations were observed. In both rats and mice, marked alterations occurred in about half the mitochondria and in a few microvilli. Cells on the villi, especially on the proximal half, usually lacked the complex interdigitations between abutting lateral surfaces which are characteristic of normal epithelium, and also showed an increase in the proportion of free ribosomes, particularly in the lower villous cells. The Golgi apparatus was often dilated, but the plasma membranes appeared normal. Determinations of fecal fat revealed that the deficient animals absorbed 83.24% of the available fat in their diet, compared to 90-97% in control animals supplemented with linoleic acid or fed Purina chow. Light- and electron-microscopic examination of a ligated portion of the jejunum which had been injected with chyme from stock chow-fed animals given corn oil confirmed that fat absorption was retarded in deficient animals and that it was restricted to a smaller surface area of the villus (only the upper half) than in control animals. The failure of cells on the lower half of the villus to absorb fat (regardless of mitochondrial structure) seems to be related to the fact that they are incompletely differentiated. The observed alteration in fat absorption may be due to abnormal molecular structure of the endoplasmic reticulum, Golgi apparatus, and the plasma and mitochondrial membranes.

7080 **FINE STRUCTURE OF THE NUCLEI OF THE DUODENAL CRYPT CELLS AFTER X-IRRADIATION.** *(E.)* Hugon, J. (Dept. Radiobiol., Ctr. Stud. Nucl. Energy, Mol, Belgium) and M. Borgers. *Amer J Path* 52(3): 701-723, 1968.

Male mice, BALB/c strain, 12 weeks old, were fasted for 18 hr and then irradiated with 1350 r of X-rays (300 kv, 10 mamp, 100 r/min; filter, 2 mm Cu). Animals were killed 30, 90, and 180 min after irradiation, and the duodenum was prepared for electron microscopic study. Mitosis was completely abolished after 30 min; bridges of chromatin extending between 2 nuclei were occasionally seen, and numerous nuclei showed alterations of the nucleoli, nuclear membranes, or the nuclear sap. An early reaction of some nucleoli with formation of nucleolar caps was noted. Nuclear blebs appeared in the form of large electron-lucent vacuoles which progressively separated from the nuclei; other nuclei showed rapid degeneration.

7081 **PRIMARY CARCINOMA OF THE FREE PORTION OF THE SMALL INTESTINE.** *(Ger.)* Eckart, A. (Rudolf Virchow Munic. Hosp., Berlin, Germany). *Chirurg* 39(1):22-26, 1968.

Of 835 malignant intestinal neoplasms removed surgically during the period 1955-1965, 805 were carcinomas of the large intestine. Only 29 were small intestine carcinomas and only 5 of these were restricted to the free portion between the duodenojejunal flexure and the ileocecal valve. These were found in 4 men and 1 woman, aged 55-77 yr. Three of these adenocarcinomas were found in the jejunum and 2 in the ileum. On the basis of clinical and X-ray findings, a diagnosis of ileus was made in 3 cases and of pyloric stenosis in 1. Secondary anemia was present in 2 cases and elevated erythrocyte sedimentation rates in 3. Laparotomies showed 3 patients to be inoperable; all 3 died a few days after surgery. *En bloc* resections of the tumor and removal of regional metastases were performed successfully in the other 2 cases.

7082 **THE EFFECT OF STAPHYLOCOCCAL ENTEROTOXIN ON THE FINE STRUCTURE OF THE MONKEY JEJUNUM.** *(E.)* Merrill, T. G. (Walter Reed Army Med. Ctr., Washington, D. C.) and H. Sprinz. *Lab Invest* 18(2): 114-123, 1968.

Intragastric administration of 150 μg of purified staphylococcal enterotoxin B to 15 Rhesus monkeys (*Macaca mulatta*) resulted in acute gastroenteritis which was well developed by 2 hr, reached a maximum at 4-8 hr, and rapidly regressed after 12-24 hr. The most prominent structural alterations included shortening and clumping of the microvilli, a decrease in the number of microvilli, loss of a distinct, uniform brush border, elongation of crypts and cytoplasmic vacuolation. The latter was shown by electron microscopy to be due to extensive mitochondrial degeneration, consisting of swelling, distortion and dislocation of cristae, pallor of the mitochondrial matrix and myelin-like configurations. Plasma cells, capillary endothelium, and fibroblasts were especially affected, while polymorphonuclear leukocytes and unmyelinated nerve cells demonstrated few mitochondrial alterations. The mitochondrial degeneration appeared simultaneously in cells of the crypt, villous epithelium and lamina propria. Such structural alterations are associated with a loss of oxidative enzymes (previously shown by histochemical and biochemical studies on the same animals). Although damaged, the mitochondria remain viable and seem to retain the ability to complete the synthesis of the basic units of mitochondrial membranes. This would account for the rapid repair and regeneration of these organelles which occurs within 24 hr of the endotoxin-induced damage.

7083 **EFFECT OF ILEAL BYPASS VERSUS ILEAL EXCISION ON CHOLESTEROL SYNTHESIS AND WHOLE BLOOD CHOLESTEROL CONCENTRATION IN THE RABBIT.** *(E.)* Buchwald, H. (Dept. Surg., U. Minnesota, Minneapolis), I. D. Frantz Jr., R. L. Gebhard and R. B. Moore. *Surgery* 64(1):126-133, 1968.

The effects of ileal bypass or partial ileal excision on total body cholesterol synthesis were investigated in 22 male New Zealand rabbits, and the potential efficacy of each procedure in treating hypercholesterolemia was compared. Before and 3 months afte surgery, the 24-hr incorporation of ^{3}H-water into cholesterol was determined by dividing the specific activity of serum cholesterol by the specific activity of the urine distillate. The incorporation ratio increased from 0.070 in control animals to 0.193 in ileal bypass animals and 0.238 in excised animals; these increases were statistically significant. The ratio of the mean differences in ^{3}H-incorporation for the excised and bypass groups was 1.228, indicating a similarity in cholesterol synthesis for both operated groups. Whole blood cholesterol levels, however, were 88.2 ± 5.8 mg% in the control group, 55.7 ± 2.9 mg% in the ileal bypass group, and 74.5 ± 1.6 mg% in the excised group; ileal bypass thus reduced cholesterol levels 37%, whereas ileal excision caused only a 16% reduction. The reasons for this significant difference are not clear.

7084 **EXPERIMENTAL INTESTINAL ATRESIA.** *(E.)* Abrams, J. S. (Cleveland Metro. Gen. Hosp., Ohio). *Surgery* 64(1):185-191, 1968.

Small intestinal ischemia was produced by mesenteric vascular ligation or mesenteric avulsion in 16 80-100 day-old fetal lambs, *in utero*, in order to bring about the development of jejunoileal atresia during the remaining gestational period. Fetal mortality was high in both groups, but except for 2 fetuses that died at or shortly after surgery, all animals showed some intrauterine growth and development. Complete revascularization of ligated segments was observed in 4 of the 6 lambs undergoing ligation; however, luminal stenosis was evident in the intestine. Mesenteric avulsion produced atresias of the following types: diaphragmatic obstruction or web (Type 1, 2 animals); blind ends connected by a cord with or without a mesenteric defect (Type 2, 4 animals); and disconnected blind ends, usually with a gap in the mesentery (Type 3, 2 animals). It is suggested that all or almost all cases of jejunoileal atresia, in contradistinction to duodenal atresia, are due to mesenteric vascular accidents that may occur at any time during pregnancy.

7085 **SERUM PROTEIN EXCRETION VIA THE GASTROINTESTINAL TRACT.** *(E.)* Hazenberg, B. P. (Acad. Hosp., Groningen, Netherlands). Thesis, U. Groningen Wolters-Noordhoff Med. Publ., 1968, 159 pp.

Studies of gastrointestinal protein excretion in 52 control subjects and 74 patients with various diseases, using ^{131}I-labeled polyvinylpyrrolidone (PVP) as an intravenous marker, indicated that 0.03-1.19% (average 0.37%) of the marker was normally excreted in the feces in 4 days, compared to a urinary excretion of about 40% in 24 hr and 50% in 3 days. The 5th-day stools added only about 7% to the total fecal excretion. Increased fecal excretion (1.4% or more in 4 days) was observed in 2 of 5 patients with congestive heart failure, 3 of 6 with regional enteritis, 5 of 5 with intestinal lymphangiectasis, 6 of 30 with proteinuria, 1 of 6 with amyloidosis, 1 of 6 with cirrhosis, and 3 patients with allergic diarrhea, aphthous gastritis and ulcers, and radiation-induced enteropathy, resp. In another group of 24 control subjects and 30 patients (16 males and 14 females, 7-73 yr old) with primary renal proteinuria, the permeability of the gastrointestinal wall was studied using ^{125}I-labeled low-molecular-weight PVP (LMW-PVP), combined in some cases with ^{131}I-labeled high-molecular-weight PVP (HMW-PVP), the average molecular weight of the 2 markers was 40,000 and 160,000, resp. Increased fecal excretion of LMW-PVP (1.4% or more in 4 days) was observed in 6 of 30 patients, and the average excretion for patients and controls was 0.94% and 0.54%, resp. With HMW-PVP, the average excretion was 0.77% in the patients and 0.54% in controls. The LMW/HMW ratio averaged 1.4-1.5 in both patients and controls, and was less than 1.0 in only 1 patient and 2 controls; however, the ratio tended to decrease with time. On the basis of these findings and a detailed review of the literature, it is concluded that increased gastrointestinal protein loss can be produced by changes in either the mucous membrane, the blood vessel walls, or the lymphatic system. Patients with renal disease also seem to have increased capillary permeability in the digestive tract, with a preferential loss of smaller molecules. The sieving effect, however, is not as great in the intestinal tract as in the kidney. The clinical, biochemical, radiological and histological findings associated with protein-losing enteropathy are considered, and its diagnosis and treatment are discussed. In a supplementary study on 23 normal subjects and 3 patients with protein-losing enteropathy, the protein composition of the jejunal secretions was studied by immunochemical techniques. Plasma/jejunal fluid ratios of prealbumin, albumin, transferrin, α_2-macroglobulin and IgG, IgA and IgM immunoglobulins were measured by double diffusion in agar, and the presence of lipoproteins and ceruloplasmin in the jejunal fluid was established qualitatively. Results were essentially the same in patients and controls, and no evidence for a molecular sieving effect could be discovered. Finally, the origin of the IgA in the jejunal fluid was investigated by immunofluorescence and tracer-incorporation techniques in jejunal biopsy specimens from 6 normal subjects and 3 patients with dysgammaglobulinemia. In all normal subjects and one patient, the results by both methods demonstrated synthesis of IgA, as well as lesser amounts of IgG and IgM, in the jejunal wall. In one patient with no local synthesis of IgA, this immunoglobulin appeared in small amounts in the jejunal fluid after a blood transfusion.

7086 PRIMARY TUMORS OF THE JEJUNUM AND ILEUM: CONSIDERATION OF 40 CASES. *(Sp.)*

Selman-Housein Abdo, E. (Med. Sch., U. Havana, Cuba) and G. Pardo Gómez. *Bull Soc Int Chir* 27(5):367-372, 1968.

Between 1961 and 1966, 40 tumors of the jejunum and ileum were diagnosed. The 16 benign tumors included 9 leiomyomas, 2 neurofibromas, 2 fibromas, 1 lymphangiomyoma, 1 polyp, and 1 hemangiopericytoma. The 24 malignant neoplasms consisted of 7 leiomyosarcomas, 6 adenocarcinomas, 3 carcinomas, 4 carcinoids, 2 reticulosarcomas, 1 lymphoma, and 1 patient with both a jejunal and an ileal melanoma. Ten of the benign tumors were located in the jejunum and 6 in the ileum. Of the malignant tumors, 12 were in the ileum, 6 in the jejunum, and the location of 6 others was not established. Most patients were older than 50 yr, but the proportion of older patients was smaller among those with malignant neoplasms. Benign neoplasms occurred primarily in females (9 of 16) and malignant tumors primarily in males (17 of 24). Both benign and malignant tumors were more common among whites than among Negroes. Diagnoses were established before surgery in 10 cases (3 benign and 7 malignant tumors) and during other operations (3 herniorrhaphies, 1 gastrectomy, and 1 patient with jaundice due to lithiasis) in 5 other cases (3 benign and 2 malignant tumors). One benign and 2 malignant tumors were diagnosed during exploratory laparotomies and 2 benign and 4 malignant tumors at autopsy. Operations were performed in 34 cases (14 benign and 20 malignant tumors). The 5-yr mortality was 7.1% for benign tumors and 42% for malignant tumors.

7087 EXPERIMENTAL EVALUATION OF SOME TECHNIQUES FOR JEJUNOPLICATION. *(Rus.)*

Kalugin, A. S. (Grodny Med. Inst., USSR). *Eksp Khir Anest* 13(3):44-47, 1968.

The results of various types of experimental enteroplication (Noble's and Childs' operations, or transmesenteric enteroplication with the fixed loops placed horizontally, obliquely and fanwise) were studied in 85 dogs, including 14 with healthy intestines and 71 in which adhesions had been produced 7 days to 4 months before. Animals were killed from 6 days to 4 yr after surgery, and none developed recurrent intestinal obstruction. A study of gross specimens revealed the development of extensive adhesions after Noble's juxtamural enteroplication with both vertical and horizontal placement of the fixed loops. After Childs' operation, dense adhesions were seen at the site of the juxtamural fixation. After mesenteric enteroplication, loose adhesions were seen in different areas. In contradistinction to juxtamural enteroplication, suturing of the small intestine to the mesentery permits free disposition of the intestinal loops. It requires only 40-50% of the surgical time required by juxtamural fixation and can be considered the most physiological procedure for the treatment of abdominal adhesions.

7088 SURGERY OF THE INTESTINAL LYMPHATICS. *(Fr.)*

Servelle, M. pp. 138-140 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7089 A STUDY OF THE LYMPHATIC SYSTEM IN DISEASES OF THE SMALL INTESTINE: CLINICAL AND BIOLOGICAL CHARACTERISTICS, METHODS OF DIAGNOSIS AND TREATMENT. *(Fr.)*

Viguié, R., A. Roberti and P. Vesin. pp.105-118 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7090 THE HISTOPATHOLOGY OF THE LYMPHATICS IN THE INTESTINAL WALL. *(Fr.)*

Cerf, M. (Bichat Hosp., Paris, France), C. Marche and C. Debray. pp. 125-133 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7091 CONTRIBUTION TO THE CLINICAL CHARACTERISTICS OF MECONIUM ILEUS. *(Ger.)*

Coerper, H. G. (Surg. Clin., U. Heidelberg, Germany), R. Daum and W. C. Hecker. *Bruns Beitr Klin Chir* 215(4):467-480, 1967.

7092 SPONTANEOUS REGRESSION IN A LEIOMYOSARCOMA OF THE SMALL INTESTINE. *(Hun.)* Czenkár, B. (Heves Co. Counc. Hosp., Hungary). *Magy Sebesz* 20(5):309-311, 1967.

7093 ALIMENTARY ILEUS. *(Hun.)* Rátkai, I. (Janos Balassa 8th Distr. Counc. Hosp., Budapest, Hungary). *Magy Sebesz* 20(4):241-244, 1967.

7094 EFFECTIVENESS OF A GLUTEN-FREE DIET IN THE TREATMENT OF CHRONIC INTESTINAL DISEASES. *(Rus.)* Iekisenina, N. I. (Inst. Nutrit., Acad. Med. Sci USSR, Moscow), G. S. Korobkina, T. N. Zitler and R. I. Chanysheva. *Vop Pitan 26(6):51-57, 1967.*

7095 SMALL BOWEL PERFORATION IN BLUNT TRAUMA. *(E.)* Weiss, M. (Mt. Sinai Hosp., New York, N. Y.) and D. A. Dreiling. *Amer J Gastroent* 50(4): 279-288, 1968.

7096 MULTIPLE TUBERCULOUS PERFORATIONS OF ILEUM. *(E.)* Prout, W. G. (Wolverhampton Group Hosp., England). *Gut* 9(4):381-382, 1968.

7097 PSEUDO-INTESTINAL OBSTRUCTION (FUNCTIONAL INTESTINAL OBSTRUCTION) IN A PREMATURE INFANT. *(E.)* Ramanathan, K. (Mt. Sinai Hosp., Chicago, Ill.) and A. Grossman. *Clin Pediat* 7(2):109-111, 1968.

7098 THE OPERATION OF CHILDS AND PHILLIPS. *(Fr.)* Moreaux, J. (Bichat Hosp., Paris, France) and J. Testart. *Entret Bichat [Chir]* pp. 27-28, 1967.

7099 SOME REFLECTIONS ON THE SURGICAL TECHNIQUES DESIGNED TO PREVENT POSTOPERATIVE OBSTRUCTION OF THE SMALL INTESTINE. *(Fr.)* Toupet, A. *Entret Bichat [Chir]* pp. 27-28, 1967.

7100 MEDICAL ASPECTS OF THE DIVERTICULOSES OF THE SMALL AND LARGE INTESTINE. *(Fr.)* Hofstetter, J. R. (Med. Polyclin., U. Lausanne, Switzerland). *Praxis* 56(41):1378-1381, 1967.

7101 SOLITARY PLASMOCYTOMA OF THE SMALL INTESTINE. *(Fr.)* Jaeger, M. (Med. Clin., U. Lausanne, Switzerland), J. Pettavel, D. Gardiol and J. Fasel. *Schweiz Med Wschr* 98(2):57-59, 1968.

7102 SOME COMMENTS ON BILIARY ILEUS. *(Cz.)* Ružička, J. (OUNZ Hosp., Liberec, Czechoslovakia) and J. Zid. *Cesk Gastroent Vyz* 22(1):38-43, 1968.

7103 EXTENSIVE RESECTION OF THE SMALL INTESTINE IN A SENILE PATIENT FOR GANGRENE DUE TO VOLVULUS FORMATION AND STRANGULATION. *(Ger.)* Schwokowski, C. F. (Polyclin., Weimar, Germany). *Zbl Chir* 93(33):1149-1152, 1968.

7104 DUODENAL DIVERTICULA. *(Ger.)* Sailer, R. (Essen Clin., Ruhr U., Germany) and G. Kuipers. *Zbl Chir* 93(33):1137-1146, 1968.

7105 ACQUIRED ILEUS. *(Ger.)* Tošovský, V. (Pediat. Surg. Clin., Prague, Czechoslovakia) an B. Fišer. *Zbl Chir* 92(26a):1950-1955, 1967.

7106 ILEUS CAUSED BY FOODS. *(Ger.)* Gdanietz, K. (Munic. Clin., Berlin-Buch, Germany). *Zbl Chir* 92(26a):1971-1979, 1967.

7107 TRAUMATIC RUPTURE OF THE SMALL INTESTINE. *(Pol.)* Maliszewski, S. (Distr. Hosp., Monki, Poland) and Z. Latosiewicz. *Pol Przegl Chir* 40(6): 667-669, 1968.

7108 COMPLETE TRAUMATIC SUBCUTANEOUS RUPTURE OF THE RETROPERITONEAL PO TION OF THE DUODENUM. *(Rus.)* Zinchenko, V. Ie. (Med. Clin., Pit No. 18, Snezhniansk Distr., Donetsk Reg., USSR). *Klin Khir* (1):75-76, 1968.

7109 MECKEL'S DIVERTICULUM IN CHILDREN. *(Por.)* Ogawa, K. M. (Darcy Vargas Child. Hosp São Paulo, Brazil), A. Enge and A. L. Mathias. *Matern Infan (S Paulo)* 26(4):387-390, 1967.

7110 THE PATHOLOGY AND CLINICAL CHARACTERISTICS OF DUODENAL DIVERTICULA. *(It.)* Descovich, P. (Inst. Spec. Surg. Path., U. Bologna, Italy *Friuli Med* 22(5):1049-1099, 1967.

7111 PROTEIN IN MECONIUM FROM MECONIUM ILEUS. *(E.)* Schutt, W. H. (Dept. Child Life Health, U. Edinburgh, Scotland) and T. E. Isles. *Arch Dis Child* 43(228):178-181, 1968.

7112 SMALL BOWEL CHANGES IN IRON-DEFICIE CY ANAEMIA OF CHILDHOOD. *(E.)* Guha, D. K. (All-India Inst. Med. Sci., New Delhi), B. N. S. Walia, B. N. Tandon, M. G. Deo, and O. P. Ghai. *Arch Dis Child* 43(228):239-244, 1968.

7113 INTRAMURAL DUODENAL HEMATOMA IN CHILDREN. *(E.)* Babbitt, D. P. (Marquette U. Sch. Med., Milwaukee, Wis.), M. I. Hussain, P. Gavino, S. Sakaguchi and A. R. Pequet. *Amer J Dis Child* 115(1):37-42 1968.

7114 BLIND POUCH SYNDROME, A COMPLICATION OF ILEOCECOSTOMY. [CASE REPOR *(E.)* Watson, M. R. (33d Station Hosp., APO 09069). *Med Bull US Army Europ* 25(10):383-385, 1968.

7115 THE SUPERIOR MESENTERIC ARTERY SYN DROME. *(E.)* Tucker, B. L. (Harper Hosp., Detroit, Mich.). *Harper Hosp Bull* 26(1):2-8, 1968.

7116 *OESOPHAGOSTOMUM COLUMBIANUM:* EXPERIMENTAL INFECTIONS IN LAMBS. EFFECTS OF DIFFERENT TYPES OF EXPOSURE ON THE

INTESTINAL LESIONS. *(E.)* Shelton, G. C. (Dept. Vet. Microbiol., U. Missouri, Columbia) and H. J. Griffiths. *Path Vet (Basel)* 4(5):413-434, 1967.

7117 TRAUMATIC RUPTURE OF INTESTINE ASSOCIATED WITH STRANGULATED INGUINAL HERNIA. [CASE REPORT] *(E.)* Kaushik, S. P. (London Rd. Hosp., Boston, Lincs., England). *Indian Med J* 62(10):207-208, 1968.

7118 TRAUMATIC RUPTURE OF THE DUODENUM. [CASE REPORT] *(E.)* Harris, H. C. (Lewisburg, Tenn.). *J Abdom Surg* 10(1):28-29, 1968.

7119 A CASE OF KAPOSI'S SARCOMA OF THE SMALL INTESTINE. [CASE REPORT] *(E.)* Birch, N. H. (Muhimbili Hosp., Dar es Salaam, Tanzania). *E Afr Med J* 44(8):343-345, 1967.

7120 CARCINOMA OF THE DUODENUM WITH ANEMIA. [CASE REPORT] *(E.)* Updegrove, J. H. (Easton, Pa.), D. Feinberg and W. A. Harlan. *J Abdom Surg* 10(3):92-94, 1968.

7121 INTESTINAL OBSTRUCTION DUE TO A DEFECT IN THE BROAD LIGAMENT. [CASE REPORT] *(E.)* Cava, A. J. (Valley Stream, N. Y.). *J Abdom Surg* 9(11):301-306, 1967.

7122 EFFECT OF ISCHEMIC SHOCK ON THE INTESTINAL MUCOSA. *(Hun.)* Ihász, M. (2nd Surg. Clin., U. Med. Sci., Budapest, Hungary), J. Füsy and E. Kisida. *Kiserl Orvostud* 20(3):310-312, 1968.

7123 HEMOLYTIC SHOCK AND THE INTESTINAL MUCOSA. *(Hun.)* Ihász, M. (2nd Surg. Clin., U. Med. Sci., Budapest, Hungary), G. Ludány and F. Váczi. *Kiserl Orvostud* 20(3):236-239, 1968.

7124 AUTO- AND HETERO-REPLACEMENT OF LIQUIDS AND ELECTROLYTES IN PATIENTS WITH JEJUNO-ILEAL FISTULAS, USING THE TECHNIQUE OF EXTRACORPOREAL INTESTINAL TRANSIT. *(Por.)* Degni, M. (Fac. Med., U. Campinas, São Paulo, Brazil), A. Godoy and P. S. B. Barbanti. *Arq Gastroent* 5(1):15-17, 1968.

See also: 6727,6844,6857,6872,6889,6890,6891,6897,6898,6929,7043,7044,7067,7277,
7496,7505,7537

7125 A NEW APPROACH TO THE TREATMENT OF GASTRODUODENAL ULCERS, USING VIT-U-PEPT INJECTIONS.

(Ger.) Kraheck, G. (Hellweg 238, Dusseldorf-Gerresheim, Germany). *Med Welt* 18(4):283-285, 1968.

"Vit-U-Pept" injections (cabbage juice extract buffered with sodium and potassium phosphates) were used in the ambulatory treatment of 42 patients with radiologically demonstrated gastric and duodenal ulcers of several years duration. All were resistant to other types of medical treatment. At a dosage of 150 mg every 2 days i.m. or i.v., radiological cures were obtained in 26 cases, radiological improvement in 6, subjective improvement in 5, and 5 cases remained unchanged. Symptoms disappeared after an average of 4.6 injections and the average patient received 10.6 injections in 25.6 days. Simultaneous administration of Rauwolfia alkaloids interfered with the therapeutic effect of Vit-U-Pept. Preliminary studies showed that the preparation is nontoxic (guinea pigs) and counteracts the ulcerogenic effect of phenylquinoline carboxylic acid in dogs; in the latter, there was a decrease in mucus secretion and parietal cells and proliferation of chief cells.

7126 THE ROLE OF VASCULAR LESIONS IN THE MALIGNANT TRANSFORMATION OF CHRONIC PEPTIC ULCER.

(E.) Juhász, J. (U. Med. Sci., Budapest, Hungary). *Acta Morph Acad Sci Hung* 16(1):41-52, 1968.

The pathogenesis of ulcerogenic carcinoma was studied on the basis of histologic examination of 443 cases of gastric cancer, 55 (11.5%) of which showed malignant transformation of chronic peptic ulcer. The findings showed that cancer arising from chronic peptic ulcer is a typical scar carcinoma. The carcinomatous transformation begins at the margin of the ulcer with multiple foci arising from the regenerating gastric mucosa. Poor vascularization in and around the scar and the subsequent hypoxia account for the qualitatively and quantitatively insufficient epithelial regeneration which is observed. The altered metabolism of these cells, together with exposure to carcinogenic influences which may act directly on the surface of the freshly formed cell layer, or which may accumulate due to the impaired regional flow of blood, could induce malignant transformation. Thorough examination of many scar cancers revealed that there is almost no proliferation of capillaries at the base of the transformed ulcer, and complete obliteration occurs in a significantly larger number of arteries than in simple ulcer. Complete obstruction of the arteries is also apparent in sections of vessels outside the scar. The ischemic-hypoxic zone is therefore much larger than the scar itself. It is concluded that whether a peptic ulcer heals or undergoes malignant transformation is greatly influenced by scar formation and the vascular changes occurring in it.

7127 VASCULAR FACTOR IN THE ETIOLOGY OF POSTHISTAMINIC GASTRIC ULCER IN GUINEA PIG. PROTECTIVE ACTION OF SYNTHETIC VASOPRESSIN ANALOGUE OCTAPRESSIN.

(E.) Kowalewski K. (Surg.-Med. Res. Inst., U. Alberta, Edmonton, Canada). *Arch Int Pharmacodyn* 170(1):66-73, 1967.

Administration of histamine (75 mg/kg i.m.) to 24 fasting guinea pigs produced large volumes of gastric juice (15.0 ml) and gastric hyperacidity (106 mEq/liter) within 4 hr compared to 12 untreated controls (4.5 ml, 22 mEq/liter, resp.). Acute gastric ulcerations appeared in 100% of the histamine-treated animals and gastric angiograms revealed diffuse congestion and a very prominent vascular network. Octapressin (5 U/kg) did not modify the volume (3.0 ml) or acidity (21 mEq/liter) of gastric secretion and produced no gastric lesions. However, injection of octapressin 10 min prior to administration of histamine in 14 guinea pigs prevented gastric ulceration in 93%, without altering the secretory response of the gastric mucosa to histamine (volume 15.5 ml, acidity 107 mEq/liter). The vascular pattern of animals treated with both histamine and octapressin appeared comparable to that of the control stomachs. It is concluded that octapressin antagonized the effect of histamine on mucosal circulation (prolonged closure of arteriovenous shunts, slowing of mucosal capillary circulation and of venous drainage), preventing the vascular changes considered to be responsible for the posthistaminic ulceration of the mucosa. These findings support the significance of the vascular factor as a primary cause of gastric lesions in histamine-treated animals.

7128 RETROSPECTIVE REVIEW OF 208 PROVED CASES OF ANASTOMOTIC ULCER.

(E.) Condon, J. R. (St. James Hosp., London, England) and N. C. Tanner. *Gut* 9(4):438-441, 1968.

Two-hundred-and-eight proven cases (184 males and 24 females) of anastomotic ulceration following surgical treatment of duodenal (203 cases) and gastric (5 cases) ulcers are reviewed with respect to difficulties in diagnosis, incidence of complications, problems of management and methods of surgical treatment. Symptoms developed an average of 1.7 yr after gastrectomy and 5.9 yr after gastroenterostomy. Pain was completely absent in 7 cases presenting with hemorrhage. More severe and persistent symptoms and a reduced tendency to heal were observed in anastomotic ulcer compared with the original peptic ulcer. Complications of both peptic and anastomotic ulcers were about equal in number (including hemorrhage, perforation, and stenosis). The shorter the interval between the first gastric operation and the development of anastomotic ulcer symptoms, the higher the incidence of complications. Combined radiographic and gastroscopic examination gave a definite diagnosis in 93 of 137 patients examined; results were equivocal in 19 and negative in 25 cases. Anastomotic ulceration after gastroenterostomy (112 cases) was treated by 2/3 to 3/4 gastrectomy followed by gastrojejunostomy (Billroth II) in 93 cases, and by vagotomy in 6 cases. For anastomotic ulceration following Billroth I gastrectomy, higher resection with construction of a gastrojejunal stoma was done in 4 cases, vagotomy alone in 3 cases, and both procedures were used in 2 patients. After Polya gastrectomy (80 cases), the choice lay between a higher gastric resection, vagotomy alone, or both operations combined (17 cases). The overall mortality for 198 operations (including 56 with acute gastrointestinal hemorrhage and 14 with perforated anastomotic ulcer) was 4.5% (9 cases). The authors recommend laparotomy in cases of suspected anastomotic ulcer, even if gastroscopic and radiologic investigations are negative, followed by surgery as soon as possible after diagnosis is made. Only a very small percentage of patients with persistently recurring anastomotic ulcers after vagotomy and partial gastrectomy require total gastrectomy.

7129 **ANTIPERISTALTIC JEJUNAL SEGMENT IN GASTRIC SURGERY.** *(E.)* Hedenstedt, S. (Nacka Hosp., Sweden) and G. Lundquist. *Acta Chir Scand* 133(7):545-554, 1967.

Twelve patients with 6-15 cm antiperistaltic jejunal segments interposed between the stomach and duodenum were followed for from 4 months to more than 3 yr. The group included 9 patients (42-70 yr old) in which jejunal reconstruction was performed during remedial operations for dumping (8 cases) and post-vagotomy diarrhea (1 case) all originally treated for peptic ulcer and 3 patients (39-55 yr old) in which jejunal interposition was done during primary antrectomy and vagotomy for duodenal ulcer. Operative techniques are described in detail. Although the dumping syndrome disappeared in all cases but 1, none of the patients had diarrhea, and all preoperative weights were maintained to within ± 5 kg, only 6 of the patients were entirely symptom-free. The other 6 exhibited signs of motility disturbances and retention, suffered from nausea or slight epigastric pain after large meals, and (in 1 case) suffered from persistent pain. It is concluded that antiperistaltic interposition is inferior to isoperistaltic jejunal transposition for the postgastrectomy syndrome, but superior for post-vagotomy diarrhea.

7130 **PATHOGENESIS OF GASTRIC MICROCIRCULATORY AND MAST CELL CHANGES IN RESTRAINT STRESS.** *(E.)* Guth, P. H. (Orange Co. Med. Ctr., Orange, Calif.) and X. Kozbur. *Amer J Dig Dis* 13(6): 530-535, 1968.

The relative blood content and mast cell concentration in the superficial gastric mucosa were determined in 3 groups of albino rats: 1) vagotomized rats, restrained and unrestrained; 2) adrenalectomized rats, restrained and unrestrained; and 3) rats given i.p. injections of saline (1 ml), urecholine (0.25 mg/100 g), hydrocortisone (10 mg/100 g), and epinephrine (0.04 mg/100 g). Relative blood content was determined by densitometric measurements on sections of hemoglobin-stained tissue and mast cell counts were performed on stained tissue sections. Following vagotomy, 4 hr of restraint resulted in a significant decrease in mucosal mast cells, no change in mucosal blood content, and no gastric ulceration. Following adrenalectomy, restraint resulted in a statistically significant accumulation of blood in mucosal microvessels, no change in mucosal mast cells, and an increased incidence of gastric ulcerations. Urecholine and epinephrine resulted in a statistically significant increase in mucosal blood content, but no change in mast cells; hydrocortisone caused a significant decrease in mucosal mast cells but no change in blood content. No causal relation was found between gastric mucosal mast cell degranulation and gastric mucosal vascular engorgement, which is evidence against a direct role for mast cell degranulation in the pathogenesis of stress ulcers.

7131 **OCCULT GASTROINTESTINAL HEMORRHAGE IN BURNED PATIENTS.** *(E.)* Ousterhout, D. K. (U. Michigan Med. Ctr., Ann Arbor) and I. Feller. *Arch Surg* 96(3):420-422, 1968.

Stool guaiac tests for the presence of occult gastrointestinal bleeding were performed in 50 patients (25 men and 25 women, 6 months-83 yr old) with 1-78% total body burns, and in another group of 43 burned patients the necropsy protocols and microscopic slides were reviewed for gastrointestinal hemorrhage and ulceration. Only 4+ guaiac stools were considered as proof of bleeding. Of the 50 patients given guaiac tests, 29 (58%), had a 4+ result at some time during their hospital stay, most commonly 8 days after burn; 2 (7%) of these 29 patients died. No differences were noted between age groups. Of the 43 cases reviewed at necropsy, 26 (60%) showed evidence of ulceration, hemorrhage, or both, with sites distributed from the esophagus (9 cases) to the large intestine (3 cases). It is concluded that continuous antacid therapy is indicated in all burned patients.

7132 **COMPARATIVE EVALUATION OF THE LONG-TERM RESULTS OF BILLROTH I AND BILLROTH II GASTRECTOMY FOR PEPTIC ULCERS.** *(Rus.)* Gordeiev, V. I. (Kherson Distr. Hosp., USSR). *Khirurgiia (Moskva)* 43(12):42-47, 1967.

In an 11-yr period, 903 gastrectomies were performed for peptic ulcer. About 80% of the patients (810 men, 93 women) were between 20 and 50 yr of age. Ulcers were found in the stomach in 283 cases (31.4%) and in the pyloroduodenal region or duodenum in 620 (68.6%). The Billroth I procedure was used in 261 cases (28.9%). Long-term follow-ups were made on 145 patients with Billroth I gastrectomies (Hofmeister-Finsterer's modification) and 102 with Billroth II. There were 213 men and 34 women in this study group. Subjective complaints were reported by 49 (33.7%) of those with Billroth I gastrectomies and by 52 (50.9%) of those with Billroth II. Typical dumping syndrome was seen in 25 patients (17.1%) with Billroth I gastrectomies and in 66 (64.7%) of those with Billroth II; 4 of those in group 2 later required reconstructive surgery. Glucose tolerance tests run on 85 patients showed that carbohydrate metabolism was almost normal in patients from group 1 while blood sugar levels increased more rapidly during the first 30 min and returned abruptly to values below normal in patients from group 2. Disturbances in liver and pancreatic function and in gastric acid secretion were found in patients from both groups. Recurrent ulcers developed in 1 patient (0.6%) from group 1 and in 2 patients (1.9%) from group 2. Radiological studies on 123 patients showed that stomach evacuation tended to be more normal in patients from group 1 and was accelerated in many patients from group 2.

7133 **SOME DATA ON THE ROLE OF HEREDITY IN GASTRODUODENAL ULCERS.** *(Rum.)* Goia, A. (3rd Med. Clin., IMF, Cluj, Romania). *Med Intern (Bucur)* 19(11):1303-1309, 1967.

A genetic survey of 3000 randomly selected hospital patients revealed a family history of peptic ulcer disease in 12.66% of 2125 controls (patients without ulcers), 17.68% of 147 patients with gastric ulcers (74.9% of whom were males), 32.14% of 728 patients with duodenal ulcers (76.9% of whom were males), and 71.73% of 138 patients with bleeding duodenal ulcers. Among the 234 duodenal ulcer patients with a family history of ulcers, 90 had parents with ulcers (the father in 60 cases and the mother in 30), 82 had siblings with ulcers (18 sisters, 63 brothers, and in 1 case both a brother and a sister), 47 had both parents and siblings, 9 had children with ulcers, and 6 had both children and siblings with ulcers. The masculine preponderance of duodenal ulcer was thus also reflected in the mode of familial transmission. Statistical analysis of the findings in 13 families with bleeding duodenal ulcers indicated recessive transmission (segregation ratio of 27.7% by the method of Weinberg) with a penetrance of 37.7%.

7134 THE INCIDENCE OF DUODENAL ULCERS IN PATIENTS WITH CHRONIC HEPATITIS.

(Rum.) Balta, N. (D. Danielopolu Inst. Norm. Path. Physiol., Acad. Soc. Rep. Romania, Bucharest), S. Schiau, A. Bordeianu, R. Stoenescu and T. Nicolaescu. *Med Intern (Bucur)* 19(10): 1217-1224, 1967.

Repeated gastric secretion studies and serial radiography revealed duodenal ulceration in 41 of 220 patients (18.6%) with confirmed chronic hepatitis who were followed for 6 yr. Hepatitis was diagnosed on the basis of clinical findings, laboratory tests (plasma proteins, prothrombin time) and liver biopsies, and was of the chronic progressive type in 29 of the 41 cases while in the other 12 it had stabilized. Only 7 of the 41 ulcers were actually diagnosed after the establishment of hepatitis, while in 27 cases the diagnoses were simultaneous; 64% of the patients were between 20 and 40 yr of age. Despite the lack of a clear etiological connection, the coexistence of the 2 diseases in these patients is tentatively ascribed to disturbances in biliary and pancreatic alkali secretion, the hormonal control of gastric secretion, and the trophic state of the duodenal mucosa resulting from the effects of hepatitis. The possibility of a direct effect of hepatitis virus on the duodenal mucosa is also mentioned. It is concluded that approximately 8% of all cases of duodenal ulcers in adults are due to hepatic disease, which complicates the therapeutic problem in these patients.

7135 STUDIES ON THE ULCEROGENIC EFFECT OF PARATHYROID HORMONE.

(Ger.) Less, E. (Korvin Hosp., Budapest, Hungary), L. Józsa, Z. Lehóczki and F. Fellner. *Z Ges Inn Med* 23(6):168-174, 1968.

In experiments on dogs, 0.025 g/kg/day i.v. of cinchophen for 2 weeks was the largest dose which did not produce any pathological changes in the stomach or urinary and serum electrolytes. The smallest dose of parathyroid hormone which produced pathological changes in the gastric mucosa was 12 U/kg/day i.m. A group of 6 dogs with Pavlov pouches received 12 U/day of parathyroid hormone for 15 days i.m. while another group of 6 dogs was given 0.025 g/kg cinchophen i.v. and 9 U of parathyroid hormone i.m. Erosion and ulcers of various sizes developed in the stomach, and in some cases in the intestine of dogs in both groups, but lesions were more extensive in animals given both parathyroid hormone and cinchophen. Changes occurred in serum and urinary electrolytes of animals in both groups. Gastric acidity increased more in the group receiving both parathyroid hormone and cinchophen. Parathyroid hormone alone increased mucus production in the stomach, increased the number of chief and parietal cells, and reduced the number of mucus-producing cells. Parathyroid hormone and cinchophen destroyed gastric glands located near erosions and produced inflammatory infiltrates. Production of neutral mucopolysaccharides and periodic acid-Schiff-positive material increased. These findings, which agree with clinical observations, suggest that parathyroid hormone is a factor in the pathogenesis of some kinds of peptic ulcers.

7136 STUDY OF PEPTIC ULCERS FOUND DURING 11,978 CONSECUTIVE AUTOPSIES (FROM 1950 TO 1964).

(Fr.) Weill-Bousson, M. (Inst. Path. Anat., Strasbourg, France) and S. Foussereau. *Arch Franc Mal Appar Dig* 57(3):329-344, 1968.

Peptic ulcers were found in 9.8% of 11,978 adults (61.2% men, 38.7% women) autopsied in the 15 yr from 1950 through 1964. Chronic ulcers (84 healed, 389 active) accounted for 40.2% of all peptic ulcers. Stomach ulcers were found in 55.4%, duodenal ulcers in 42.5%, and both stomach and duodenal ulcers in 2.1%. Stomach ulcers occurred significantly more often in women and duodenal ulcers more often in men. In women, the incidence of chronic ulcers increased progressively with age, while in men the incidence remained stationary after the age of 30. Ulcer complications were the direct cause of death in 169 of the 389 subjects with active ulcers (bleeding in 27%, perforation in 15.9%, stenosis in 0.5%). Perforations occurred significantly more often in women, while bleeding was significantly more frequent in men, probably because many men with ulcers also had cirrhosis and the disorders in blood coagulation which are associated with it. Chronic ulcers accounted for only 1.3% of all deaths, but they caused death significantly more often in men than in women. Cirrhosis was present in 10.3% of the subjects with chronic ulcers and asystole was found in 10.4% of the men. Acute ulcers accounted for 59.6% of all peptic ulcers (241 in women, 462 in men). As with chronic ulcers they occurred most often in the stomach (63.4% gastric, 28.1% duodenal, 8.4% gastric and duodenal), but there were no significant sex differences. Fatal gastrointestinal bleeding occurred in 201 of these 703 subjects (28.5%) and was slightly more common among men than women; perforations only occurred in 2 cases. The diseases most often associated with acute ulcers were cirrhosis (20.7%), cerebral lesions (19.7% and acute infections (17.6%).

7137 STUDIES ON THE MORPHOLOGY OF THE LIVER IN PATIENTS WITH GASTRODUODENAL ULCERS.

(Pol.) Smolak, K. (Inst. Oncol., Krakow, Poland), A. Gorniak and A. Urban. *Pat Pol* 18(3):371-380, 1967.

Preoperative liver biopsies, taken by Menghini needle during surgery for peptic ulcers (47 gastric and 65 duodenal) in 112 patients (89 men and 23 women, average age 45 yr), revealed structural abnormalities in 58.9% of the cases (61.8% of those with gastric and 56.9% of those with duodenal ulcers), with severe changes in 8.9% (10 cases). The duration of illness averaged 5 and 8 yr for gastric and duodenal ulcers, resp., and 51% of the patients had developed complications (bleeding, perforation, pyloric obstruction). The most common hepatic finding was nonspecific reactive hepatitis of weak-moderate intensity. Specific findings included necrobiosis and hepatocellular necrosis (in 51% of gastric-ulcer and 41.5% of duodenal-ulcer patients), nuclear polymorphism (in 44.6% and 38.4%, resp.), proliferation of Kupffer cells (36.1% and 21.5%), nuclear vacuolization (10.4% and 10.7%), bile duct proliferation (12.7% and 23.1%), and fatty infiltration (2.1-7.6%). Normal livers were seen in 51.7% of those under 45 yr of age and only 30.7% of those over 45; normal livers were also found in 54.8% of those with bleeding and 44.4% of those without pyloric obstruction, compared to only 27.2% of those with pyloric obstruction and 1 of 6 patients with perforation. The hepatic lesions are therefore ascribed partially to pyloric obstruction and partially to the effects of age, which probably reduces the regenerative capacity of the liver.

7138 **BEHAVIOR OF THE SERUM ACID MUCOPOLYSACCHARIDES FOLLOWING SURGERY FOR PEPTIC ULCERS.** *(Pol.)* Szulc, H. (3rd Surg. Clin., Acad. Med., Poznan, Poland) and E. Szwabe. *Pol Przegl Chir* 40(1):52-58, 1968.

Serum acid mucopolysaccharide levels were determined before and on the 1st and 7th day after surgery (Rydygier or Hoffmeister-Finsterer gastrectomy) in 20 patients (25-73 yr old) operated on for peptic ulcers (10 gastric and 10 duodenal). The preoperative values in these 2 groups were 132-176 (average 151.3) and 73.5-137 (average 106.0) mg%, resp. There was a definite decrease (17.7-17.8%) in both groups on the 1st postoperative day, to average values of 125.5 and 87.1 mg%, resp.; his decrease was shown by all 20 patients. Between the 1st nd 7th postoperative day, there was a moderate increase in he acid mucopolysaccharide levels in all but 3 patients, reachng average values of 1.36 and 93.6 mg/, resp.; in the gastric lcer group this increase (9.9%) was statistically significant, while n the duodenal ulcer group it was not (7.4% increase). The inal values were still significantly below the preoperative values 9.6-11.7% decrease) in both groups, however, a net decrease eing shown by all but 2 patients.

7139 **THE INFLUENCE OF FLUID AND ELECTROLYTE ADMINISTRATION ON POSTOPERATIVE WEIGHT-LOSS.** *(E.)* Hindmarsh, J. T. (Royal Infirm., Sheffield, England) and R. G. Clark. *Brit J Surg* 54(10):872-874, 1967.

7140 **A CASE OF POSTBULBAR DUODENAL ULCER.** *(Cz.)* Vrubel, F. (Med. Fac., Purkinje U., Brno, Czechoslovakia), A. Novák and J. Vitek. *Cesk Gastroent Vyz* 21(7):425-428, 1967.

7141 **GASTROINTESTINAL ULCERS OF HYPOXIC ORIGIN.** *(Rus.)* Fastovskii, V. L. (1st Munic. Clin. Hosp., Chernovtsy, USSR). *Vrach Delo* (12):8-12, 1967.

7142 **SIGNIFICANCE OF HOSPITALIZATION AND PROPHYLACTIC TREATMENT AGAINST RECURRENCES IN MAINTAINING THE WORKING CAPACITY OF PATIENTS WITH PEPTIC ULCERS.** *(Rus.)* Perveieva, V. G (16th Munic. Hosp., Dnepropetrovsk, USSR). *Vrach Delo* (1):27-29, 1968.

7143 **THE USE OF VITAMIN E IN THE TREATMENT OF GASTRODUODENAL ULCERS.** *(Rus.)* Parkhovnik, M. B. (29th Distr. Hosp., Kharkov, USSR). *Vrach Delo* (1):117-118, 1968.

7144 **THE USE OF ANABOLIC STEROIDS IN THE TREATMENT OF PEPTIC ULCER DISEASE.** *(Rus.)* Epshtein, B. V. (Kiev Milit. Hosp., USSR). *Vrach Delo* (1):124-125, 1968.

7145 **CHRONIC DIGESTIVE DISORDERS FOLLOWING VAGOTOMY.** *(Fr.)* Cornet, A. (Laennec Hosp., Paris, France) and M. Grivaux. *Entret Bichat [Med]* pp. 609-614, 1967.

7146 **STOMACH CANCER FOLLOWING GASTROENTEROSTOMY FOR PEPTIC ULCER.** *(Fr.)* Hillemand, P. (6 Place Mexico, Paris, France) and P. Vayre. *Entret Bichat [Med]* pp. 131-134, 1967.

7147 **CANCER OF THE GASTRIC STUMP FOLLOWING GASTRECTOMY FOR ULCER.** *(Fr.)* Lagache, G. (Gen. Adult Surg. Clin., Hosp. Ctr., Lille, France) and M. Vankemmel. *Entret Bichat [Chir]* pp. 41-46, 1967.

7148 **DRUG-INDUCED GASTRODUODENAL ULCERS.** *(Fr.)* Debray, C. (Bichat Hosp., Paris, France) and P. Duret. *Entret Bichat [Ther]* pp. 9-12, 1967.

7149 **SURGERY FOR INTRACTABLE ULCER.** *(E.)* Wiederanders, R. E. (Harmon Park Clin., Williston, Minn.) and R. J. Olson. *J Lancet* 88(9):229-235, 1968.

7150 **AN EXPERIMENTAL STUDY ON THE EFFECTS OF IMMOBILIZATION AND AGE ON GASTRIC ULCERATION.** *(E.)* Agari, S. (Kyushu U., Fukuoka, Japan). *Igaku Kenkyu* 38(2):333-354, 1968.

7151 **EFFECTS OF DIFFERENT OPERATIONS FOR PEPTIC ULCER ON GASTRIC ACIDITY.** *(E.)* Padhy, K. C. (S. C. B. Med. Coll. Hosp., Cuttack, India) and M. Khadanga. *J Indian Med Ass* 51(3):116-121, 1968.

7152 **A RADIOLOGICAL STUDY OF THE DIGESTIVE TRACT IN ACROMEGALY.** *(It.)* Bruno, G. (Inst. Gen. Med. Clin., U. Turin, Italy), G. M. Molinatti and M. de la Pierre. *Arch Ital Mal Appar Dig* 34(1):99-101, 1967.

7153 **THE SURGICAL TREATMENT OF PEPTIC ULCERS.** *(Sp.)* Markman, I. (Rawson Hosp., Buenos Aires, Argentina). *Arq Gastroent* 5(3):131-140, 1968.

7154 **REVIEW OF THE DISEASES MOST COMMONLY ASSOCIATED WITH PEPTIC ULCERS.** *(Cz.)* Kořenek, J. (Med. Fac., Purkinje U., Olomouc, Czechoslovakia). *Cesk Gastroent Vyz* 21(7):488-492, 1967.

7155 **EVALUATION OF THE INDICATIONS FOR THE SURGICAL TREATMENT OF PEPTIC ULCERS.** *(Cz.)* Veselý, K. T. (Inst. Res. Human Nutrit., Prague, Czechoslovakia). *Cesk Gastroent Vyz* 21(6):415-419, 1967.

7156 **PERSONAL EXPERIENCE WITH THE PREPARATION MUTHESA.** *(Cz.)* Hradský, M. (Med. Fac., Charles U., Hradec Kralove, Czechoslovakia). *Cesk Gastroent Vyz* 21(6):409-411, 1967.

7157 **EVALUATION OF A NEW ANTACID. I. LABORATORY EVALUATION : A PRELIMINARY REPORT.** *(E.)* Samson, E. V. (Mercury Drug Co., Manila, Philippines) and C. B. Asiddao. *Santo Tomas J Med* 22(6): 416-422, 1967.

7158 PEPTIC ULCER OF THE SECOND SEGMENT OF THE DUODENUM. *(Por.)* San Juan, F. (Hosp. State Employees, Rio de Janeiro, Brazil). *Hospital (Rio)* 72(6):1691-1697, 1967.

7159 PEPTIC ULCERS IN CHILDREN AND ADOLESCENTS. *(Por.)* Bettarello, A. (Fac. Med., U. São Paulo, Brazil), O. Borini, S. Zaterka and J. F. Pontes. *Arq Gastroent* 4(3):225-235, 1967.

7160 CLINICAL ASPECTS OF ATYPICAL PEPTIC ULCERS. *(Por.)* De Godoy, A. (Inst. Gastroent., Sao Paulo, Brazil) and A. Bettarello. *Arq Gastroent* 4(3):195-206, 1967.

7161 CLINICAL TRIAL OF THE PREPARATION GLIPIN IN PATIENTS WITH PEPTIC ULCERS. *(Rus.)* Trusov, V. V. (Izhevsk Med. Inst., USSR). *Vrach Delo* (11):133-134, 1967.

7162 DISRUPTION OF SMALL INTESTINAL FUNCTION IN PATIENTS WITH DUODENAL ULCERS. *(Rus.)* Linevskii, Iu. V. (Donetsk Med. Inst., USSR). *Vrach Delo* (11):131-133, 1967.

7163 DIAGNOSTIC AND THERAPEUTIC PROBLEMS IN PEPTIC ULCER. *(Ser.)* Hadžić, N. *Lijecn Vjesn* 90(4):355-357, 1968.

7164 ANASTOMOTIC ULCERS. *(Sp.)* Klinger, J. (Salvador Hosp., Santiago, Chile), E. Ronban and I. Arteaga. *Rev Med Chile* 96(4):229-234, 1968.

7165 CASE REPORT OF A PERFORATED DUODENAL ULCER IN A CHILD. *(Cz.)* Bobek, V. (Dept. Surg., OUNZ, Rokycany, Czechoslovakia). *Rozhl Chir* 46(9):573-574, 1967.

7166 THE TREATMENT OF PYLORIC STENOSIS OF ULCEROUS ETIOLOGY BY DRAGSTEDT'S PROCEDURE: PROBLEMS OF GASTRIC EMPTYING ENCOUNTERED IN 76 CASES. *(Fr.)* Chabal, J. (Fac. Med., Dakar, Senegal), C. Ballon and B. Croizat. *Lyon Chir* 63(5): 680-689, 1967.

7167 VASCULAR FACTORS AS INTEGRATING COMPONENTS IN THE PATHOGENESIS OF GASTRIC ULCERS. *(Ger.)* Wanke, M. (Inst. Gen. Path., U. Heidelberg, Germany). *Med Welt* 18(50):3003-3009, 1967.

7168 CASE REPORT OF A CINGULATE ULCER OF THE STOMACH. *(Rus.)* Sliusarenko, Ie. A. (Yeletsk Railway Hosp., USSR) and V. A. Gerasimchuk. *Klin Khir* (1):78-79, 1968.

7169 PITUITARY FUNCTION IN PEPTIC ULCER DISEASE. *(Rus.)* Mosin, V. I. (Stavropol Med. Inst., USSR). *Vrach Delo* (10):33-36, 1968.

7170 THE ROLE OF TRIOXAZINE IN THE COMPLE TREATMENT OF PEPTIC ULCER DISEASE. *(Rus.)* Kravchenko, A. I. (Kiev Med. Inst., USSR). *Vrach Delo* (10):43-46, 1968.

7171 CLINICAL EVALUATION OF A NEW NON-ANTICHOLINERGIC COMPOUND WITH PROTECTIVE AND ANTISECRETORY ACTIVITY IN THE TREATMENT OF GASTRITIS AND GASTRODUODENAL ULCERS. *(It.)* Fantino, M. (Gen. Surg. Clin., U. Turin, Italy) and L. Roccia. *Minerva Med* 58(86):3677-3685, 1967.

7172 THERAPEUTIC ACTIVITY OF MILID (XYLAM IDE, CR 242) IN THE ANTISECRETORY TREATMENT OF GASTRODUODENAL ULCERS. *(It.)* Giordano, G. (Inst. Spec. Med. Path., U. Naples, Italy) and L. Comi. *Minerva Med* 58(86):3688-3691, 1967.

7173 TREATMENT OF SOME TYPES OF CHRONIC GASTRODUODENITIS AND GASTRODUODENAL ULCERS WITH A DERIVATIVE OF AN ALICYCLIC CARBOXYLIC ACID WHICH CAN EITHER BE ADMINISTERED BY MOUTH OR INJECTED INTRAVENOUSLY OR INTRAMUSCULARLY. *(It.)* Abbona, C. (Gen. Med. Clin., U. Genoa, Italy), P. L. Gatto and G. C. Bezante. *Minerva Me* 58(86):3691-3699, 1967.

7174 THE CLINICAL PHARMACOLOGY, THERAPEUTIC ACTIVITY, AND CLINICAL TOLERANCE OF A NEW NON-ANTICHOLINERGIC AGENT WHIC IS EFFECTIVE IN GASTRODUODENITIS AND GASTRODU DENAL ULCERS (MILID). *(It.)* Cascone, A. (Maggiore Hosp Lodi, Italy), D. Borellini and B. Jezzi. *Minerva Med* 58(86): 3699-3704, 1967.

7175 CLINICAL CONSIDERATIONS ON THE THER PEUTIC EFFECT OF A NEW, NON-ANTICHOLINERGIC, ANTISECRETORY PREPARATION: MILID (XYLAMIDE, CR 242). *(It.)* Castelli, C. A. (Castelli Clin., Bergamo, Italy), L. Locatelli and G. F. Salvioni. *Minerva Med* 58(86):3705-3708, 1967.

7176 PHARMACOLOGICAL AND CLINICAL STUDIES ON A NON-ANTICHOLINERGIC ANTISECRETORY AGENT: MILID (XYLAMIDE, CR 242). *(It.)* Mainoli, S. (Munic. Hosp., Bellano, Italy), C. Milvio and D. Borellini. *Minerva Med* 58(86):3708-3712, 1967.

7177 THE ROLE OF VAGOTOMY. *(E.)* Lehman, J and W. A. D'Alonzo. *J Abdom Surg* 10(1):22-2 1968.

7178 PERFORATED DUODENAL ULCER TREATED WITH PYLOROPLASTY AND VAGOTOMY- A PRELIMINARY STUDY. *(E.)* Chowdhury, A. K. R. (N. R. S. Med. Coll., Calcutta, India). *Indian Med J* 62(10):209-210, 1968.

7179 THE ASSOCIATION OF PEPTIC ULCERATION, CHRONIC RENAL DISEASE AND ANALGESIC ABUSE. *(E.)* Kincaid-Smith, P. (Royal Melbourne Hosp., Australia), J. K. Dawborn and K. F. Fairley. *Clin Med* 75(8): 19-22, 1968.

7180 THE CHALLENGE OF BENIGN GASTRIC ULCER. *(E.)* Aralar, P. M. (Harper Hosp., Detroit, Mich.). *Harper Hosp Bull* 26(1):9-13, 1968.

See also: 6757,6763,6841,6843,6883,7010,7018,7020,7026,7038,7039,7062,7064,7071,
7072,7274,7275,7329,7396

7181 REGIONAL ENTERITIS IN MEXICO. *(Sp.)*

Jinch, H. (Hosp. Nutrit. Dis., Mexico City, Mexico), E. Rojas and R. de Castro. *Gac Med Mex* 97(6):681-689, 1967.

A detailed discussion of the epidemiology, diagnosis and treatment of regional enteritis is presented, based on 22 cases (5 males and 17 females, 9 between the ages of 30 and 40) seen during a 13-yr period. Although patients ranged in age from the 1st to the 7th decade, there was a striking peak in the 4th decade, and a striking preponderance of women; also, 5 of the 22 patients were Jews. There was a high incidence of coexisting psychoneurotic symptoms, and possible partly as a result of this, half of the group were not correctly diagnosed until 5 yr or more after onset of symptoms. The most common complications were intestinal obstruction, malnutrition, anemia, hemorrhage and intestinal fistulas. Radiological examination revealed stenosis, rigid intestinal loops with dilatation of the proximal loop, polyps, a paved aspect, ulceration, irregularities in the lumen and contour, and hypertrophy of the mucosal folds. There were 7 cases of terminal ileitis and 7 with segmental jejunoileitis, with scattered other localizations. The histopathology ranged from lymph stasis and edema via the typical granulomatous lymphangitis up to fibrotic retraction. Medical treatment (rest, diet, antibiotics, antispasmodics and sedatives) produced a cure in 1 of 6 patients and improvement in 2 others, but is generally considered to be ineffective. Radiotherapy and corticosteroids were each tried in 2 cases, without success. Of 15 patients subjected to surgery, 1 died at operation, 3 died later, 1 was lost to follow-up, and 6 of the remaining 10 had recurrences. In view of the high mortality and recurrence rate, surgery is only recommended in the presence of complications.

7182 ACUTE REGIONAL ILEITIS AT IWANAI IN HOKKAIDO—WITH SPECIAL REFERENCE TO INTESTINAL ANISAKIASIS. *(E.)*

Ishikura, H. (Sapporo Med. Coll., Japan), H. Hayasaka and Y. Kikuchi. *Sapporo Med J* 32(4/5):183-196, 1967.

An epidemiologic, clinical and pathocytologic survey of 371 cases of acute regional ileitis occurring in Hokkaido during the period 1955-66 is presented. The high geographic concentration of the disease in the coastal areas of Hokkaido suggests a close relation to ingestion of seafood. Epidemiologic analysis revealed that the peak incidence of the disease coincided with the winter fishing season of Alaska pollack, the intermediate host of the nematode larvae. In a series of 129 cases of acute regional ileitis at Iwanai (west coast of Hokkaido), well-preserved *Anisakis* larvae were obtained from 13 of 50 resected intestinal specimens. It was clearly shown that these larvae were identical to the *Anisakis* larvae found in the pollack fish. X-ray studies revealed stenosis, irregular contours and serrate shadows in the affected small intestine. Extensive ascites was observed in the abdominal cavity (200-500 ml); no mucosal ulcers or granuloma formation was found in intestinal tissue. Eosinophilic infiltration was seen 4 days after the onset of the disease and the intestinal submucosa showed intense edema and fibrinoid swelling of the connective tissue; further infiltration of the various layers of the intestinal wall by eosinophilic leukocytes and lymphocytes suggested an allergic tissue reaction. These change are completely different from those found in Crohn's disease. Experimental studies on rabbits infected and reinfected with *Anisakis* larvae demonstrated an Arthus type allergic reaction almost identical to the reaction observed in human anisakiasis. It is suggested that the disease in humans tends to occur after repetitive ingestion of raw fish containing *Anisakis* larvae.

7183 SURGERY AND CLINICAL PROGRESSION IN PATIENTS WITH CROHN'S DISEASE OF THE SMALL AND LARGE INTESTINE. *(E.)*

Goldstein, M. J. (Dept. Med., U. Chicago, Ill.), H. Schachter and J. B. Kirsner. *Amer J Surg* 115(3):376-384, 1968.

Eight case histories (5 men and 3 women, 17-56 yr old) are presented to illustrate the variety and magnitude of the clinical complications that may follow surgery for Crohn's disease of the small and large intestine. In one patient, recurrent internal fistulas occurred after resection of the large intestine; in another, surgery for recurrent perineal fistulas proved ineffective when extensive inflammatory disease of the small and large intestine was present. Recurrent small intestinal obstruction was observed in a 3rd patient operated on for Crohn's disease of the small intestine; productive transmural inflammation and subsequent fibrosis, as well as external adhesions, may have been responsible for this complication. Other patients with recurrent intestinal bleeding (1 case) or diarrhea and secondary malabsorption (1 case) following resection for Crohn's disease of the small intestine are reported. Small intestinal obstruction ileostomy prolapse and excessive ileostomy output occurred in 1 patient after ileostomy. In 2 other cases, ileitis and obstruction after total colectomy, and recurrent diarrhea, perirectal fistulas and abscesses, malnutrition, obstruction, and severe electrolyte disturbances after colostomy illustrate the relentless, fatal course sometimes followed by Crohn's disease.

7184 REGIONAL ENTERITIS AND ITS SURGICAL TREATMENT. *(Fin.)*

Elfving, G. (2nd Surg. Clin., U. Helsinki, Finland), J. Hästbacka, G. Fock and K. Saikku. *Duodecim* 84(1):32-39, 1968.

7185 AETIOLOGY OF REGIONAL ENTERITIS. *(E.)*

Golde, D. W. (Natl. Inst. Health, Bethesda, Md.) and C. M. McGill. *Lancet* 1(7552):1144-1145, 1968.

7186 ACUTE REGIONAL ENTERITIS. *(Ger.)*

Fahrländer, H. (Inst. Path. Anat., U. Basel, Switzerland) and F. Gloor. *Deutsch Med Wschr* 92(42):1891-1896, 1967.

See also: 7212,7215,7251

7187 **SUBCELLULAR PATHOLOGY OF HEREDITARY FRUCTOSE INTOLERANCE.** *(E.)* Phillips, M. J. (Toronto Gen. Hosp., Ontario, Canada), J. A. Little and T. W. Ptak. *Amer J Med* 44(6):910-921, 1968.

Liver and jejunal biopsy specimens taken from a 27-yr-old male with hereditary fructose intolerance 1 day prior to and 2 hr after the ingestion of 50 g of fructose were examined by light and electron microscopy. No change in the histologic structure of the liver or intestine was noted by light microscopy. However, there were striking fine-structural alterations which coincided with the occurrence of severe clinical manifestations (epigastric pain, dizziness, nausea, sweating and tremor). In the jejunum, the brush border region of the absorptive cells remained unchanged but concentric arrays of smooth membranes were noted in supranuclear regions. In the liver, alterations were more widespread, consisting of concentric and irregularly disposed membranous arrays occurring in the glycogen areas of most hepatocytes, associated with marked rarefaction of the hyaloplasm. Many of the membranous formations resembled cytolysomes. It is concluded that these lesions are a manifestation of focal cytoplasmic degeneration and the consequence of fructose toxicity, and that the formation of the lesions is related to the intracellular accumulation of substrates.

7188 **EFFECT OF THE ADMINISTRATION OF TRIGLYCERIDES CONTAINING MEDIUM-CHAIN FATTY ACIDS ON THE STEATORRHEA OF RATS WITH EXPERIMENTAL BLIND LOOPS.** *(Sp.)* Valdivieso, V. (Clin. Hosp., Cath. U., Santiago, Chile), R. Palma, H. Orrego and L. Coloma. *Rev Med Chile* 95(10):587-594, 1967.

An experimental blind-loop syndrome, with a fecal fat excretion of 18.6% of the dietary intake compared to 3.7% in controls and an increase in fecal weight from 0.87 to 3.11 g/24 hr, was produced in adult AxC rats by constructing an 8-cm jejunal loop 7 cm from the ligament of Treitz (technique of Cameron). Fecal fat excretion could be significantly decreased (to 4.58%), however, with no significant change in fecal weight (3.22 g/24 hr), by administration of a diet containing triglycerides with medium-chain fatty acids (caprylic, capric, caproic and lauric) as the only source of exogenous fat. Sixty days after the operation, the metabolism of glyceryl trioctanoate labeled with ^{14}C in the carboxyl group of octanoic acid was studied by measuring the $^{14}CO_2$ of the expired air during a 90-min period. When the trioctanoin was administered in a fat mixture containing long-chain fatty acids, the respiratory $^{14}CO_2$ accounted for 51% of the administered label in rats with the blind-loop syndrome and 50% in controls. When the trioctanoin was administered in a fat mixture containing medium-chain fatty acids, this difference was eliminated and the metabolism for both blind-loop and control rats was lower (41% and 36.5%, resp.).

7189 **CLINICAL AND MORPHOLOGIC CHANGES FOLLOWING GLUTEN ADMINISTRATION TO PATIENTS WITH TREATED CELIAC DISEASE.** *(E.)* Schenk, E. A. (U. Rochester Sch. Med. Dent., N. Y.) and I. M. Samloff. *Amer J Path* 52(3):579-593, 1968.

Four adult patients (26-66 yr old) with celiac disease were studied before and during treatment with a gluten-free diet and at intervals after readministration of a gluten-containing diet. Clinical studies of intestinal absorption and jejunal biopsies were performed as described in an earlier report. Stool collections between carmine red markers were analyzed for fat content. Pretreatment jejunal biopsies revealed the characteristics of celiac disease in all patients, with a flat mucosa containing no villi, flattening and abnormal staining of the surface epithelial cells, and a dense infiltrate of chronic inflammatory cells in the lamina propria. Prominent lipid accumulations were noted in the surface epithelial cells, with little or no lipid in the lamina propria. During treatment with a gluten-free diet, fecal fat excretion decreased in the 3 patients who had steatorrhea prior to treatment, and jejunal biopsies revealed a progressive decrease of lipid in the surface epithelium, increase of lipid in the lamina propria, and a return toward normal epithelial cells with a villous mucosal pattern. A gluten-containing diet was reintroduced to 3 patients 73, 55, and 40 days after the gluten-free diet. Fecal fat excretion increased significantly within 3-8 days. Jejunal biopsy 24 hr after gluten readministration revealed a prominent accumulation of lipid within the surface epithelium; at 48 hr, a flat mucosa, pseudostratification of the surface epithelium, and a greater increase in the surface epithelium lipid were noted; 12 days later, a pattern similar to the pretreatment stage was seen.

7190 **VITAMIN B_{12} DEFICIENCY IN DOGS FOLLOWING PROLONGED EXCLUSION OF EXOCRINE PANCREATIC SECRETIONS.** *(Rus.)* Lavrova, V. S. (Tomsk Med. Inst., USSR). *Pat Fiziol Eksp Ter* 11(5):64-69, 1967.

Intestinal absorption of ^{60}Co-labeled vitamin B_{12} was studied in dogs whose pancreatic ducts had been ligated and cut and whose omentum had been placed between the pancreas and duodenum. Dogs with ligated pancreatic ducts excreted 65-99% of orally administered vitamin B_{12} in their stools in contrast to 23.7-45.9% excreted by controls. Intestinal absorption of vitamin B_{12} was increased, but not to normal levels, when the vitamin was administered together with 1.5-9 g pancreatin, 80 mg crystalline trypsin, or 10 g $NaHCO_3$. Pancreatin lost its effectiveness when heated in the autoclave at 1.5 atm for 20 min. No atrophic changes were found in the stomach or intestine which might have reduced secretion of intrinsic factor and mucosal absorption. The increase in mucoproteins in gastric juice produced by i.v. injection of insulin was within normal limits in 4 of 5 dogs studied. These findings suggest that reduced intestinal absorption of vitamin B_{12} is due to the absence of pancreatic enzymes, especially trypsin, and a shift in the pH of the intestinal contents towards lower values. Vitamin B_{12} concentrations in the livers of these dogs were reduced and were lowest in animals surviving longest after surgery. Serum vitamin B_{12} concentrations began to decrease 3 months after surgery and fell below normal limits in the fourth month. Among the few symptoms of vitamin B_{12} deficiency in these dogs were macrocytic anemia and a tendency for neutrophils to shift slightly toward the right.

7191 **DIARRHEA AS A FORM OF DIABETIC ENTEROPATHY.** *(Pol.)* Kirchmayer, S. (2nd Clin. Int. Dis., Acad. Med., Krakow, Poland) and K. Cichecka. *Przegl Lek* 24(8):627-633, 1968.

The clinical characteristics, diagnosis and pathogenesis of diabetic diarrhea and steatorrhea are discussed on the basis of 6 personal cases (4 men and 2 women, 22-52 yr old) with a 3-6-yr history of diabetes and a 2-4-yr history of diarrhea. Four of the 6 patients had moderate to severe polyneuropathy and 2 had mild polyneuropathy. Only one patient was continent, and this was also the only patient with an iodophilic intestinal flora. While 2 patients showed normal gastric acidity, 2 were hyperchlorhydric, 1 was hypochlorhydric, and 1 had histamine-resistant achlorhydria. Pancreatic function was normal in all 6 patients. The results of lipid absorption studies, fecal analysis and gastrointestinal radiology indicated that both diarrhea and steatorrhea were caused by accelerated small intestinal transit, resulting in decreased absorption of dietary fat. The differential diagnosis of diabetic diarrhea, gluten enteropathy and other types of steatorrhea is discussed.

7192 **TROPICAL SPRUE IN NORTHERN INDIA.** *(E.)* Misra, R. C. (Maulana Azad Med. Coll., New Delhi, India), N. Krishnan, V. Ramalingaswami and H. K. Chuttani. *Scand J Gastroent* 2(3):192-199, 1967.

The clinical and pathological features are presented for 70 cases (48 males and 22 females) of tropical sprue occurring in the Delhi area, characterized by diarrhea of more than 6 weeks duration, negative stool cultures, and evidence of nutritional failure including loss of weight. The most frequent complaints were diarrhea, loss of weight, soreness of the tongue and mouth, and loose, foul smelling stools. Abdominal pain relieved by the passage of stools was present in 42 patients (60%). Nausea and vomiting were present in 20 cases (37%) and 40 patients (57%) had signs of vitamin B deficiency. The most frequent signs were pallor and cachexia with loss of muscle mass; ankle edema was present in 24 and ascites in 6. A majority of patients (49) were found to excrete 6-15 g of fat daily. In 51 cases, barium meal examination showed dilatation of the jejunum and ileum with thick mucosal folds in the early cases and tubelike patterns in the more advanced cases. In 45 of 66 patients receiving an oral glucose tolerance test, a rise of less than 25 mg/100 ml was seen at 30 min and 1 hr. Jejunal biopsies performed on 41 patients revealed a histologically normal pattern in 5 cases, and varying degrees of abnormalities in the remainder, most being mild to moderate in severity. Although similar to the celiac disease and idiopathic steatorrhea of the West, this disease differs in that it has a short and self-limiting course and the steatorrhea is generally mild, as are the histological abnormalities in the jejunal mucosa. The course of the disease is not influenced by a gluten-free diet. The etiology and pathogenesis remains obscure.

7193 **BILE SALT AND MICELLAR FAT CONCENTRATION IN PROXIMAL SMALL BOWEL CONTENTS OF ILEECTOMY PATIENTS.** *(E.)* Van Deest, B. W. (U. Texas Southwest. Med. Sch., Dallas), J. S. Fordtran, S. G. Morawski and J. D. Wilson. *J Clin Invest* 47(6):1314-1324, 1968.

To determine whether abnormal bile salt metabolism is responsible for the steatorrhea in patients with ileal disease and/or
ileectomy, duodenal bile salt concentrations after a single,
standard meal eaten at 8 a. m. were measured in 8 patients
with ileectomy steatorrhea and compared with those in 11 n
mal subjects and 7 patients without gastrointestinal disease.
Mean bile salt concentration was approximately 50% normal
in the ileectomy group, but some of the patients fell well wi
in the normal range. However, a marked depression of duod
bile salt concentration occurred in ileectomy patients after t
2nd and 3rd meal of the day, suggesting that the 1st meal
flushed out a large fraction of the bile acid pool. Simultane
turnover studies with taurocholate-^{14}C showed a half-life of
3.1 hr in the ilectomy group as compared to 29.5-32 hr in
the control groups, proving interruption of the enterohepatic
circulation in ileectomized patients. Hepatic synthesis, howe
apparently reconstitutes the bile salt pool partially overnight
Below a bile salt concentration of 1.7 mg/ml, less than 0.8
mg/ml of lipid existed in the micellar phase of intestinal con
tents, whereas increasing bile salt concentrations were accom
panied by increasing amounts of fat in the micellar phase. C
1 of 11 samples from 3 ileectomy patients had a micellar fa
concentration greater than 0.8 mg/ml, whereas 33 of 42 sam
ples from the controls were above this level. Abnormally lo
duodenal bile salt concentration during at least a part of the
day, with an associated depression of micellar fat, appears to
be the major cause of decreased fat absorption in patients
with ileectomy steatorrhea.

7194 **MALABSORPTION IN THE TROPICS. 2. T**
ICAL SPRUE VERSUS PRIMARY PROTEIN
MALNUTRITION: VITAMIN B_{12} AND FOLIC ACID ST
(E.) Ghitis, J. (Valle U. Sch. Med., Cali, Colombia), K. Trip
and G. Mayoral. *Amer J Clin Nutr* 20(11):1206-1211, 196

In an effort to define the distinguishing diagnostic features
long-standing tropical sprue, which may result in secondary
tein malnutrition, 5 patients with megaloblastic anemia and
chronic diarrhea, 15 with primary protein malnutrition and
rhea, and 9 with hookworm anemia but no diarrhea were st
Patients with hookworm (previously shown to have normal
testinal biopsies, normal d-xylose absorption, and no steato
had normal (230-900 pg/ml in 6 cases) or high (1000 pg/ml
3 cases) serum vitamin B_{12} levels, normal (5.9-14 ng/ml in
cases) or low (1.7 and 3.2 ng/ml in 2 cases) folic acid levels,
and normal absorption of pteroylglutamic acid (PGA; 4 of 6
patients tested). The bone marrow was normoblastic in all
In subjects with primary protein malnutrition (previously sh
to have abnormal jejunal mucosa, malabsorption of fat and
xylose), absorption of PGA was abnormal in 3 of 4 cases, se
vitamin B_{12} levels were usually normal (402-760 pg/ml in 9
cases) or high (912-1000 pg/ml in 5 cases), being low (173
in only 1 case, and folic acid levels ranged between low (1.2
2.9 ng/ml in 5 cases) and high normal (4.5-19.3 in 10 cases)
Malabsorption of PGA was observed in 3 of 4 patients. The
bone marrow was normoblastic in all but 1 case. Four of th
5 patients with megaloblastic anemia (previously shown to h
steatorrhea, abnormal intestinal biopsy and depressed d-xylo
abosrption) revealed low serum vitamin B_{12} (127-200 pg/ml
and folic acid levels (0.8-4.1 ng/ml), and abnormal PGA abs
tion, while 1 patient had a normal vitamin B_{12} level (417 p
a very low folate level (0.9 ng/ml) and a markedly abnormal
PGA absorption test. In all 5 cases, treatment with PGA resulted in disappearnce of megaloblastosis, improvement of th
anemia and eventual normalization of xylose and fat absorpt

but repeated intestinal biopsies in 3 patients still showed marked abnormalities after several months of PGA and high-protein therapy. In contrast, complete normalization of intestinal histology was observed on high-protein diets in patients with only protein malnutrition.

7195 **FAT ASSIMILATION IN PATIENTS WITH CHRONIC ENTEROCOLITIS FOLLOWING PRESCRIPTION OF A DIET WITH A NORMAL FAT CONTENT.** *(Rus.)* Savoshchenko, I. S. (Inst. Nutrit., Acad. Med. Sci. USSR, Moscow) and L. M. Krums. *Vop Pitan* 26(6):49-51, 1967.

7196 **HISTOLOGICAL AND ELECTRON-MICROSCOPIC STUDY OF THE JEJUNAL MUCOSA IN A CASE OF WHIPPLE'S DISEASE BEFORE AND AFTER ANTIBIOTIC THERAPY.** *(It.)* Sposito, M. (S. Giacomo Hosp., Rome, Italy) and G. Hassan. *Arch Ital Mal Appar Dig* 34(3): 301-314, 1967.

7197 **MALABSORPTION SYNDROMES.** *(E.)* Shingleton, W. W. (Duke U. Med. Ctr., Durham, N.C.) and W. O. Dobbins, III. Springfield, Ill., C. C. Thomas, 1968. 165 pp.

7198 **MALABSORPTION AND MALNUTRITION IN RURAL HAITI.** *(E.)* Klipstein, F. A. (Columbia U., New York, N. Y.), M. Samloff, G. Smarth and E. Schienk. *Amer J Clin Nutr* 21(9):1042-1052, 1968.

7199 **PROTEIN MALNUTRITION-INDUCED MALABSORPTION AND OTHER ENTEROPATHIES IN COLOMBIA.** *(E.)* Mayoral, L. G. (U Hosp., Cali, Colombia.), K. Tripathy, F. T. Garcia, O. Bolanos and J. Ghitis. *Amer J Clin Nutr* 21(9):1053-1065, 1968.

7200 **DESQUAMATIVE ERYTHRODERMA: A CLINICAL STUDY WITH SPECIAL ATTENTION TO THE ASSOCIATED MALABSORPTION SYNDROME.** *(Dut.)* Den Tandt, W. (St. Rafael Acad. Hosp., U. Louvain, Belgium), E. Eggermont and N. Bourgeois. *Acta Paediat Belg* 21(6):433-450, 1967.

7201 **THE SYNDROME OF FAMILIAL MEGALOBLASTIC ANEMIA WITH PROTEINURIA.** *(Ger.)* Jacobi, H. (Pediat. Clin., U. Freiburg i. Br., Germany), H. Heimpel, H. J. Cremer and W. Künzer. *Deutsch Med Wschr* 92(41):1853-1859, 1967.

7202 **THE DIAGNOSIS OF MALABSORPTION DUE TO ENZYME DEFICIENCIES.** *(Ger.)* Drube, H. C. (1st Med. Clin., U. Kiel, Germany). *Deutsch Med Wschr* 93(2):81-82, 1968.

7203 **ENTERIC ENZYMOPATHIES: DISACCHARIDASE DEFICIENCY.** *(Por.)* Pontes, J. F. (Inst. Gastroent., São Paulo, Brazil). *Arq Gastroent* 4(3):251-273, 1967.

7204 **EXPERIMENTAL ENTEROPATHY INVOLVING LOSS OF SERUM ALBUMIN IN THE RAT.** *(Fr.)* Milhaud, G. (Isotope Lab., Pasteur Inst., Paris, France) and P. Guillien. pp. 134-137 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

See also: 6669,6836,6851,6863,6873,7290

LARGE INTESTINE

7205 CHANGING BURSTING STRENGTH AND COLLAGEN CONTENT OF THE HEALING COLON.

(E.) Cronin, K. (U. Oregon Med. Sch., Portland), D. S. Jackson and J. E. Dunphy. *Surg Gynec Obstet* 126(4): 747-753, 1968.

In each of 5 groups of 30 female Sprague-Dawley rats (210-250 g) an anastomosis was made in the lower left colon in 20 animals while the remaining 10 served as controls. Each group was killed at intervals of 3, 5, 7, 10, and 14 days after operation. Two equal, 2.5-cm long segments of bowel were removed (segment 1, containing the anastomosis at its center, and segment 2, immediately proximal to segment 1), the lower end of each segment was tied off, and a small glass nipple connected to a pressurized oxygen source and a sphygmomanometer was tied into the upper end. The preparation was placed in normal saline and the intraluminal pressure was raised by 10 mm Hg every 15 seconds. The rupturing point was usually abrupt; if bubbles appeared, their 1st appearance was taken as the bursting point. In the controls, the average rupturing pressure for segments 1 and 2 were similar (153-158 mm Hg). The rupturing pressures for segment 1 at days 3, 5, 7, 10, and 14 were 36, 176, 194, 218, and 218 mm Hg, resp.; corresponding pressures for segment 2 were 159, 182, 187, 203, and 190 mm Hg, resp. Collagen determinations made on the bowel segments revealed a 40% decrease of collagen in the wound edges and 2.5 cm above the anastomosis and a 10% decrease 1.25 cm below the anastomosis, 3 days after operation. From day 5-10, the collagen concentration increased and was only slightly below normal, but remained so even up to day 14. It is concluded that during the 1st 3 days of the reparative process the collagen of the submucous layer is broken down, both at the anastomosis and above and below it. The loss is made good by the synthesis of new collagen, which is laid down in the adjacent uninjured colon as well as at the anastomosis.

7206 SYNCHRONOUS EXTIRPATION OF THE RECTUM FOR CANCER.

(Rus.) Ryzhikh, A. N. (Sci.-Res. Lab. Proctol., Min. Health RSFSR, Moscow, USSR), S. N. Fain, I. M. Inoiatov and V. B. Aleksandrov. *Khirurgiia (Moskva)* 44(8):122-129, 1968.

In a 2.5-yr period, 102 cancerous rectums were extirpated by either of 2 variants of a procedure involving 2 teams of surgeons, 1 working in the abdominal cavity and the other simultaneously at the peritoneum. In contrast to the conventional procedure of Quénu and Miles, the first variant makes it possible to remove the rectum without opening it so that the danger of implanting metastases and spreading infection is reduced. In the second variant the rectum is only open for 1-1.5 min. The stump of the resected intestine does not remain in contact with the tissues of the true pelvis or the peritoneal incision as long as with the conventional technique, and it is easier to suture the abdominal peritoneum onto the free cavity of the true pelvis. Mobilization of the intestine facilitates removal of tumors from adjacent organs. The average time required to perform this procedure was 1 hr 17 min compared to 2 hr 28 min for the Quénu-Miles procedure. Indications for the synchronous procedure are tumors located low in the rectum (less than 6 cm from the anus), highly malignant tumors, and fatty tissue in the mesentery and intestinal wall. The operative mortality was 5.9% with synchronous extirpation compared to 11.4% with the Quénu-Miles procedure. Death was caused by thromboembolisms of the pulmonary artery in 2 cases, liver failure in 1, sigmoid necrosis and peritonitis in 2, and sepsis in 1.

7207 INTRAPERITONEAL INFECTION AND CANCER OF THE COLON AND RECTUM.

(E.) Liechty, R. D. (U. Iowa Coll. Med., Iowa City), S. E. Vanourny and S. E. Ziffren. *Arch Surg* 96(4):599-603, 1968.

Of 2009 patients treated for cancer of the large bowel in a 20-yr period, 139 (24-91 yr old) had intraperitoneal infections (free perforation with peritonitis or abscesses); 65 of these infections developed prior to and 74 developed after cancer treatment. The overall immediate mortality was 46% (64 patients), including 20 of 24 patients with inoperable perforating cancers. In the group of 65 with preoperative infections, the rectosigmoid was the most common site of perforation (32 of 65); free perforation was more common than abscess formation (40 and 25 cases, resp.). In this group, 38 patients with potentially curable lesions available for follow-up had a 5-yr survival of 47% (18 of 38), with a distinct increase in survival for those patients having primary resection of their tumors as contrasted to those having delayed resections. In the 65 patients with operable cancers among the 74 with postoperative infections, there was a 0-24% immediate mortality with drainage or fecal diversion, while a 95% mortality was noted in those patients treated conservatively. The overall 5-yr survival of patients with large bowel cancer and infection (28%) was not different from the 5-yr survival of all patients with bowel cancer (31%); however, since 46% of patients with infection died immediately, an adjusted 5-yr survival rate of 55% was noted for patients with resectable lesions who survived the infection (39 of 71 patients), while 39% (611 of 1567 patients) of all patients with potentially curable cancers survived 5-yr. It appears that patients with large bowel cancers who sustain, yet survive infection, have an increased 5-yr survival rate.

7208 FAMILIAL POLYPOSIS OF COLON: A TWENTY-FIVE YEAR FOLLOW-UP STUDY.

(E.) Falk, V. S. (Carle Foundation Hosp. Clin., Urbana, Ill.) and J. C. T. Rogers. *Arch Surg* 96(6):967-969, 1968.

A follow-up study of 6 siblings (3 males and 3 females) with familial polyposis of the colon, originally reported 25 yr ago, is presented. The father of the original 7 children (one of whom did not develop polyps) died of rectal carcinoma at 48 yr of age. Four (3 men and 1 woman) of the 6 siblings with polyposis have died of rectocolonic cancer at 28, 29, 30, and 33 yr of age; 3 had had inadequate surgical procedures prior to the study and the 4th deferred surgery until only a palliative procedure was possible. Two of the 6 had adequate surgery (total colectomy, and colectomy and ileosigmoidostomy) at ages 24 and 22 and are still alive and well. These 6 siblings with polyposis have had 7 children, of which 4 (all females) have the familial disease: 1 (30 yr old) had a total colectomy at age 16; 1 (22 yr old) had a colectomy at age 12; 1 (17 yr old) has just recently had a colectomy and carcinoma was found and 1 (16 yr old) has had surgery advised. The remaining 3 children (2 females and 1 male) are free of disease. The 1 male of the original 7 siblings who was free of disease has had 5 children, all of whom are also free of disease. The type of polyposis presented in these cases is a hereditary condition with frequent malignant degeneration. Early radical surgery is the treatment of choice.

7209 CANCER OF THE COLON: THE INFLUENCE OF THE *NO-TOUCH ISOLATION* TECHNIC ON SURVIVAL RATES.

(E.) Turnbull, R. B. Jr. (Cleveland Clin. Found., Ohio), K. Kyle, F. R. Watson and J. Spratt. *CA* 18(2):82-87, 1968.

Follow-up data on 896 patients with cancer of the colon were analyzed to determine the effect of the "no-touch isolation" surgical technique as compared to conventional techniques on survival rates. A total of 664 patients was treated with the "no-touch isolation" technique and 232 patients were treated by a conventional technique when resection was indicated. The operability rates were the same in both groups. Comparison of the survival rates showed that deaths from metastatic cancer were more frequent during the 2 yr after conventional resection. Most of these deaths were due to hepatic metastasis, and it is suggested that this greater incidence of fatal hepatic metastasis is secondary to operative manipulation of the cancer-bearing segment. Of those who underwent resection for cure, 460 patients received the "no-touch isolation" technique and 128 patients received the conventional method. The operative mortality in the first group was only 2.2%, and the age-corrected five-year survival rate was consistently high (81.6%).

7210 EXPERIMENTAL ACUTE COLITIS IN THE RHESUS MONKEY FOLLOWING PERORAL INFECTION WITH *SHIGELLA FLEXNERI*: AN ELECTRON MICROSCOPE STUDY.

(E.) Takeuchi, A. (Walter Reed Army Med. Ctr., Washington, D. C.), S. B. Formal and H. Sprinz. *Amer J Path* 52(3):503-529, 1968.

Male and female rhesus monkeys, free from diarrhea and with stool cultures negative for enteric pathogens, were given 3 x 10^8 *Shigella flexneri* organisms by stomach tube; control animals received sterile broth. Animals were sacrificed 48 hr later, the entire intestine was opened and inspected, and a segment of the descending colon was removed for morphologic study, including fluorescent antibody studies and light and electron microscopy. Infected animals developed the clinical signs and symptoms of acute monkey shigellosis by 24 hr, and a diffuse acute colitis was well established by 48 hr. The small intestine was unaffected, the cellular inflammatory reaction being confined to the mucosa of the colon; the submucosa was involved by edema only. The shigellae were present predominantly in the more luminal portions of the colonic epithelial lining, less frequently in the deeper portion of the crypt gland epithelium and least frequently in the tunica propria. This suggests that dysentery bacilli invade the colonic mucosa from the intestinal lumen with a preference for the cytoplasm of colonic epithelial cells. The bacilli inside the epithelial cells appeared viable, capable of undergoing cell division, lateral spread to adjacent epithelial cells, and penetration into the tunica propria. Disappearance of goblet cells, accumulation of lipid droplets in both surface and crypt epithelial cells, and pyknotic shrinkage of these cells were noted. Extrusion of epithelial cells, leading to the formation of micro-ulcers at the luminal surface and to crypt distortion and blockage, was also seen. A distinct correlation between the quantity of bacilli present and the intensity of the inflammatory response was observed. The endothelial lining of the blood vessels was relatively uninvolved, and abscess formation in the tunica propria was rarely seen.

7211 DIAGNOSIS AND TREATMENT OF CONGENITAL DOLICHOSIGMOID IN CHILDREN.

(Rus.) Trofimova, Z. A. (Astrakhan Med. Inst., USSR) and A. V. Makarov. *Pediatriia* 47(3):64-68, 1968.

During a 5-yr period (1962-1966), dolichosigmoid was diagnosed in 48 children (18 girls, 30 boys). The most common clinical symptoms were abdominal pain and constipation, and the most characteristic radiological sign was great mobility of the intestine in the abdominal cavity. In addition, 23 of these children had functional disturbances of the small intestine. Symptoms were mild in 22 patients, moderately severe in 19, and severe in 7. Conservative treatment, consisting of diet therapy, mineral oil, saline enemas, abdominal massage, exercise, polyvitamins, and fish oil, was used in 37 cases. Eleven others who did not respond to conservative therapy were treated surgically. Hagen and Thorn's mesosigmoidopexy was performed on 2 patients with mild symptoms and rectosigmoid resections on 9 with severe and moderately severe symptoms. Long-term follow-ups of operated patients showed that all were in good health and well developed. Results of conservative treatment were good in 20 cases, satisfactory in 14, and unsatisfactory in 3.

7212 REGIONAL COLITIS OR CROHN'S DISEASE OF THE COLON.

(Fr.) Maratka, Z. (Bulovska Hosp., Prague, Czechoslovakia), D. Kubernatova, V. Capek and J. Kudrmann. *Actualites Hepatogastroent Hotel Dieu* 4(1):A15-A52, 1968.

Of 264 patients with "rectocolitis" seen between 1960 and 1966, 24 (15 females, 9 males) were found to have regional colitis (9.1%). These patients included 5 with ileocolitis, 4 with regional typhlitis, 6 with left-sided regional colitis, and 9 with regional rectitis or colorectitis. Most patients were in the third or fourth decade. Symptoms depended upon the location and severity of the process, but the most common were diarrhea, obstruction, masses, and fistulas. Radiological findings varied but lesions included localized stenosis or obstruction and there was a delay in the transit of barium sulfate. In the initial stages mucosal edema gave a characteristic "cobblestone" image. The only histological finding definitely pathognomic of regional colitis was sarcoids, but their absence did not rule out this diagnosis. Of these 24 patients, 18 were treated surgically by radical removal of the affected part of the intestine. Results were good except in 2 patients who had recurrences in segments higher up, 2 who developed fistulas, and 2 who died. Antibiotics and corticosteroids were used to treat patients with extensive ileocolitis and in whom there were no urgent indications for surgery. One 23-yr-old woman developed polyarthritis, nodular erythema, and episcleritis before signs of intestinal involvement appeared. Papulopustular skin lesions which showed the same sarcoid reaction as lesions of the colon appeared later. A case report is presented for a 56-yr-old woman which shows how lesions develop progressively higher up in the colon and ileum. The only sarcoid reaction was found in pericolonic infiltrates of the small intestine.

7213 SOME GENETIC ASPECTS OF FAMILIAL MULTIPLE POLYPOSIS OF THE COLON IN A KINDRED OF 1,422 MEMBERS.

(E.) Pierce, E. R. (Dept. Biol., U. Louisville, Ky.). *Dis Colon Rectum* 11(5):321-329, 1968.

The genetic basis of polyposis and the manifestation of the polyposis gene were analyzed statistically in the 1421 members of a single kindred. The complete kindred of 7 generations consisted of 706 males, 667 females, and 49 members whose age and sex were unknown; the sex distribution did not differ significantly from the expected ratio of males to females. There were 72 proven polyposis cases (32 males and 40 females), 8 deduced cases, and 14 carrier cases. Deduced cases included those kindred members known to have died from one of the less specific causes listed on death certificates, such as bowel cancer, liver cancer, intestinal cancer, or simply, "cancer"; polyposis carriers were those members free of intestinal symptoms and diseases who produced affected offspring. Descendents with polyposis were produced by 2 of the 3 brothers in the 2nd generation. The data presented do not support the hypothesis that polyposis is more frequent in males than females. The data do show a slight excess of affected sons from affected fathers, and a marked excess of affected daughters from affected mothers ($P < 0.05$). Further investigations are needed, however, to determine whether these tendencies are biologic or genetic phenomena. The polyposis gene seems to be transmitted as a simple Mendelian dominant. The incidence of individuals heterozygous for polyposis at birth was calculated to be 1 in 6850 in the Louisville, Kentucky area. Relative biological fitness (the measure of the ability to maintain personal genes in the population by passing them on to the next generation) was calculated to be 84.85% in this group when compared with the general population. The penetrance of the polyposis gene (the regularity with which the gene expressed itself) was calculated to be 49.43%. It was also estimated that 12 mutations occurred per million loci per generation.

7214 PROCIDENTIA OF THE RECTUM STUDIED WITH CINERADIOGRAPHY: A CONTRIBUTION TO THE DISCUSSION OF CAUSATIVE MECHANISM.

(E.) Brodén, B. (St. Görans Hosp., Stockholm, Sweden) and B. Snellman. *Dis Colon Rectum* 11(5):330-347, 1968.

Cineradiography of the rectum, performed in 54 patients (6 men and 48 women) with anal symptoms while straining at stool, revealed 16 cases of internal intussusception without enterocele, 4 cases of internal intussusception with enterocele, 17 cases of prolapse without enterocele, where the intussusceptum prolapsed through the anal canal, and 11 cases of prolapse with enterocele, including 6 cases where the small intestine descended into the procidentia. In 3 additional cases, the procidentia protruded rapidly, without straining, when the patient sat on the examination seat; rectal prolapse and enterocele formation occurred simultaneously. In 2 final cases, prolapse began in the anal canal itself; the groove around the procidentia was absent and enterocele was present in only 1 patient. Intussusception beginning at the rectosigmoid junction was never followed by rectal prolapse. Thus, in patients with procidentia, straining at stool produced rectal intussusception followed by the prolapse of the intussusceptum through the anus. The intussusceptum and procidentia contain a peritoneal pouch formed by the anterior rectal wall proximal to the peritoneal reflection. At different stages in the development of the intussusceptum and procidentia, small intestine may enter this pouch, but this does not cause prolapse.

7215 CROHN'S DISEASE OF THE COLON.

(Fr.) Mialaret, A. Buson, M. Julien and J. Chomé. *Mem Acad Chir (Paris)* 93(26/27):829-835, 1967.

Case reports are presented for 5 patients with granulomatous colitis, 2 of whom were adolescents. In 3 cases Crohn's disease simulated ulcerative colitis or hemorrhagic rectocolitis. An 18-yr-old boy with histologically confirmed Crohn's disease of the ileum later developed lesions in the sigmoid. A 15-yr-old girl, who underwent a total colectomy, had lesions of the colon, cecum, and appendix. Gross examination of lesions in the large intestine showed small islets of pseudopolyps similar to those found in ulcerative colitis. However, histological studies demonstrated the presence of giant polynuclear cells and granulomatous features typically found in Crohn's disease. A 44-yr-old man apparently developed acute Crohn's ileitis after an episode of ulcerative colitis. The relative rarity of Crohn's disease of the colon in France is attributed to the strict criteria used by French physicians in its diagnosis. All of the following features must be present: 1) mucosal ulcerations with or without fistulas, 2) sclerotic thickening, particularly of the submucosa, 3) hyperplasia of colonic lymphatic structures, and 4) characteristic histological findings. Most French reports of this disease have been published in the past 2 or 3 yr and might be accounted for by autosuggestion from British publications.

7216 ALTERATIONS IN THE MOUSE CECUM AND ITS FLORA PRODUCED BY ANTIBACTERIAL DRUGS.

(E.) Savage, D. C. (Rockefeller U., New York, N. Y.) and R. Dubos. *J Exp Med* 128(1):97-110, 1968.

Administration of penicillin (0.1-0.6 g/liter in drinking water) to NCS and NCS-D mice produced enlargement of the cecum and a 2- to 3-fold increase in cecal wet weight within 12 hr. The increase in weight was due chiefly to the accumulation of water in the lumen, and not in the wall. Terramycin (1.7 g/liter) and kanamycin (1.0 g/liter) resulted in similar but less spectacular changes in the cecums. With all drugs the cecums remained enlarged and generally tended to increase in weight as long as the antibiotics were administered. During the 12-24 hr after drug administration, there was no detectable evidence of histologic abnormality in the cecal epithelium. In contrast, profound changes occurred in the microflora of both the mucous layer and the lumen: in animals given penicillin the cecums were free of culturable bacteria; fusiform-shaped bacteria enterococci, and bacteroides were present in mice receiving Terramycin; and lactobacilli and bacteroids were found in those given kanamycin. After the 1st 48 hr, each drug induced a characteristic proliferation of microflora qualitatively different from those found in control mice. In mice given penicillin for only 24 hr, the cecal weight and bacterial flora spontaneously returned to normal levels in 7-8 days after stopping the drug administration; this period was not significantly shortened by contaminating their food with cecal homogenates from control animals. When penicillin was given for 14 days, it took more than 2 weeks for cecal weights and bacterial populations to turn to normal after discontinuation of the drug, but only 0 days when the mice were given the contaminated food. hese observations, together with similar results in germ-free dents, suggest that some particular types of bacteria (probably siform) play an essential role in maintaining the integrity of e water-transport mechanism in the intestinal epithelium.

217 **RESULTS OF THE SURGICAL TREATMENT OF APPENDICITIS.** *(Pol.)* Korfel, Z. (J. niadecki Distr. Hosp., Bialystok, Poland), H. Buluk and K. jejska. *Pol Przegl Chir* 39(11):1198-1204, 1967.

uring the period 1963-66, 1595 patients were operated on for ppendicitis at the Bialystok Provincial Hospital, representing 0.8-23.5% of the 7320 patients operated on during this period. he largest age group was between 12 and 20 yr for both males nd females, and 70-75% of all patients were under 40. Duraon of symptoms at time of surgery was longer than 3 days in 2.9-27.0% and was 1 day or less in 37.4-45.0%. The main ymptoms were pain (spontaneous in 100% and in response to ressure in 93.6%), nausea and vomiting (88.7%), muscular gidity (79.5%), fever (76.2%) and leukocytosis (above 9000 in 1.1%). There were 1290 cases of simple acute appendicitis ncreasing from 67% to 92.9% of the cases between 1963 and 966), 31 cases with local peritonitis, 44 cases with diffuse perionitis, and 230 cases of chronic appendicitis. Postoperative omplications (mainly suppuration and abscesses) developed 10.8-16.2% of all cases, and the mortality rate was 0.2-0.7%. he average duration of hospitalization was 6.9-8.3 days for hronic appendicitis, 8.0-10.2 days for simple acute appendicitis, 1.3-15.4 days for local peritonitis, and 19.8-22.2 days for iffuse peritonitis. Both the percentage of complications and verage hospital stay have decreased during the period under iscussion. It is concluded that the diagnosis of acute appendiitis remains difficult, especially in young women, and that here is a dangerous tendency to neglect abdominal pain, reulting in delays in treatment and the development of compliations. Surgery is not recommended for chronic appendicitis, ince there is always a danger that the operation might be ndertaken during an acute phase.

218 **FISTULA OF THE TRANSVERSE COLON DUE TO CHRONIC ABSCESSING PARANEPHRITIS.** *(It.)* Pomidori, A. (Inst. Surg. Symptomatol., U. Naples, taly) and A. Balice. *Gazz Int Med Chir* 72(24, bis):3207-216, 1967.

219 **A NEW TECHNIQUE FOR REDUCING THE COLON IN THE TREATMENT OF ACQUIRED MEGACOLON.** *(Por.)* Mendonca, T. (Fac. Med., Fed. U. Goias, Goiania, Brazil). *Rev Goiana Med* 13(3/4):177-180, 967.

220 **FIFTEEN YEARS' EXPERIENCE WITH APPENDICITIS (ANALYSIS OF 9485 CASES).** *Hun.)* Lőke, M. (Kaposvar Hosp., Hungary), G. Nagy, B. Szabó and M. Vőlgyi. *Magy Sebesz* 20(6):359-364, 1967.

7221 **MULTIPLE MALIGNANT TUMORS OF THE LARGE INTESTINE.** *(Cz.)* Mohl, H. L. KUNZ South. Bohemia, Ceske Budejovice, Czechoslovakia). *Cesk Gastroent Vyz* 21(7):441-445, 1967.

7222 **COLITIS CYSTICA PROFUNDA.** *(E.)* Scruggs, F. L. (Methodist Hosp., Memphis, Tenn.) and J. K. Duckworth. *Southern Med J* 61(6):618-620, 1968.

7223 **ADENOMAS OF THE COLON.** *(E.)* Paradny, R. (Mt. Sinai Med. Sch., New York, N. Y.) and A. E. Kark. *Amer J Gastroent* 50(3):186-194, 1968.

7224 **THE PATHOLOGICAL ANATOMY OF HIRSCHSPRUNG'S DISEASE.** *(Rus.)* Bakhtiiarov, V. A. (Kemerovo Med. Inst., USSR) and V. D. Levin. *Vrach Delo* (12):12-15, 1967.

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7226 **THE EARLY DIAGNOSIS OF CANCER OF THE RIGHT COLON.** *(Fr.)* Debray, C. (Bichat Hosp., Paris, France) and F. Coste. *Entret Bichat [Med]* pp. 123-130, 1967.

7227 **DIVERTICULOSIS OF THE COLON AND SIGMOIDITIS.** *(Fr.)* de la Vaissiere, G. (Rothschild Hosp., Paris, France). *Entret Bichat [Chir]* pp. 57-62, 1967.

7228 **HYPERTROPHIC TUBERCULOSIS OF THE RECTUM.** *(E.)* Hawley, P. R. (St. Mark's Hosp., London, England), H. R. I. Wolfe and J. M. Fullerton. *Gut* 9(4):461-465,1968.

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7230 **FAMILIAL POLYPOSIS OF COLON: A TWENTY-FIVE YEAR FOLLOW-UP STUDY.** *(E.)* Falk, V. S. (Dept. Surg., Carle Clinic, Urbana, Ill.) and J. C. T. Rogers. *Selected Papers Carle Clin* 21(2):34-38, 1968.

7231 **MUCUS-SECRETING ADENOMATOUS POLYP AT THE SITE OF URETEROSIGMOIDOSTOMY: A CASE REPORT AND REVIEW OF THE LITERATURE.** *(E.)* MacGregor, A. M. C. (Maryfield Hosp., Dundee, England). *Brit J Surg* 55(8):591-594, 1968.

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7235 **FAMILIAL POLYPOSIS OF THE COLON.** *(Sp.)* Jimenez Ibañez, J. (T. de Alvear Hosp., Buenos Aires, Argentina), C. A. Macagno and J. H. Guillamondegui. *Prensa Med Argent* 55(22):1045-1051, 1968.

7236 **THE BIOPSY DIAGNOSIS AND PATHOLOGY OF HIRSCHSPRUNG'S DISEASE IN RELATION TO ACQUIRED AND FUNCTIONAL MEGACOLON.** *(Ger.)* Meier-Ruge, W. (Inst. Path., U. Basel, Switzerland). *Verh Deutsch Ges Path* 51:323-329, 1967.

7237 **THE POSSIBILITY OF MALIGNANT DEGENERATION IN ADENOMA OF THE COLON.** *(Ger.)* Reifferscheid, M. (Med. Fac., Rhine-Westphalia Tech. Inst., Aachen, Germany). *Deutsch Med Wschr* 92(42):1896-1900, 1967.

7238 **MULTIPLE PATHOLOGY OF THE COLON: REPORT OF A CASE.** *(Por.)* De Souza, V. C. T. (Tradesmen's Hosp., Rio de Janeiro, Brazil), E. M. de Miranda, H. Carrapatoso and E. M. de Freitas. *Hospital (Rio)* 72(6): 1851-1862, 1967.

7239 **INFLAMMATORY TUMORS FOLLOWING APPENDECTOMY.** *(Bul.)* Atanasov, A. (Higher Med. Inst., Sofia, Bulgaria) and Kh. Mikhov. *Khirurgiia (Sofiia)* 21(2):133-139, 1968.

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7241 **INTESTINAL OBSTRUCTIONS DUE TO VOLVULUS OF THE RIGHT COLON: REPORT OF TWO CASES.** *(It.)* Barile, E. (Munic. Hosp., Genoa, Italy) and G. Grosso. *Rass Arch Chir* 6(4):247-261, 1968.

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7245 **IMPERFORATE ANUS IN THE CHILD: ITS SIGNIFICANCE AND TREATMENT.** *(Por.)* Lopes Filho, O. C. (Darcy Vargas Child. Hosp., São Paulo, Brazil). *Matern Infanc (S Paulo)* 26(4):357-370, 1967.

7246 **THE SURGICAL TREATMENT OF HIRSCHSPRUNG'S DISEASE.** *(Por.)* Pinto, V. C. (Darc Vargas Child. Hosp., São Paulo, Brazil). *Matern Infanc (S Paulo)* 26(4):395-398, 1967.

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7248 **INTUSSUSCEPTION OF THE VERMIFORM APPENDIX: REPORT OF A CASE.** *(It.)* Bergnach, A. (Munic. Hosp., Gorizia, Italy). *Friuli Med* 22(5): 1161-1168, 1967.

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7250 **REPORT OF A CASE IN WHICH THE SIGMOI COLON WAS INTERPOSED BETWEEN THE RIGHT HEMIDIAPHRAGM AND THE LIVER.** *(Fr.)* Ionesc A. (Botosani Hosp., Romania) and P. Mendelsohn. *Lyon Chir* 63(6):855-857, 1967.

7251 **ANOTHER CASE OF CROHN'S DISEASE OF THE COLON.** *(Fr.)* Jouanneau, P. (Fac. Med., Rouen, France), Y. Geffroy, R. Laumonier, A. Fondimare, J. Larcher, F. Cottard and U. Marel. *Mem Acad Chir (Paris)* 93(28):843-848, 1967.

7252 **A CASE OF CROHN'S DISEASE WITH DOUBLE COLONIC LOCALIZATION.** *(Fr.)* Edelmann, G., May and P. Etienne. *Mem Acad Chir (Paris)* 93(28):895-898, 1967.

7253 **APPENDICITIS WITH PERITONITIS DUE TO *PASTEURELLA MULTOCIDA*. [CASE REPOR** *(E.)* Controni, G. (Child. Hosp., Washington, D. C.) and V. Stephens. *Amer J Med Techn* 34(8):467-469, 1968.

7254 **VASCULAR OCCLUSION OF THE COLON AND ORAL CONTRACEPTIVES: POSSIBLE RELATION.** *(E.)* Kilpatrick, Z. M. (Hosp. St. Raphael, New Haven, Conn.), J. F. Silverman, E. Betancourt, J. Farman and J. P. Lawson. *New Eng J Med* 278(8):438-440, 1968.

7255 **ACUTE APPENDICITIS AT BROOKE GENERA HOPSITAL: A REVIEW OF 500 CONSECUTIV CASES (1959-1965).** *(E.)* Peterson, H. D. (Walter Reed Gen. Hosp., Washington, D. C.). *Milit Med* 133(1):33-38, 1968.

7256 **EMERGENCY AND NONPLANNED REMOVAL OF THE LEFT COLON.** *(E.)* Herrington, J. L. (Vanderbilt U. Med. Ctr., Nashville, Tenn.) and H. A. Graves J *Surg Gynec Obstet* 126(4):1045-1052, 1968.

7257 **URINARY COMPLICATIONS AFTER ABDOMI PERINEAL RESECTION OF THE RECTUM IN**

EN. *(E.)* Glass, R. L. (U. Missouri Med. Sch., Columbia) d J. S. Spratt. *Amer Surg* 34(3):238-241, 1968.

258 **PROGRESS IN CANCER DETECTION. II. CANCER PREVENTION AND DETECTION IN HE ANUS, RECTUM AND COLON.** *(E.)* Hertz, R. E. Mem. Ctr. Cancer Allied Dis., New York, N. Y.) and M. R. oddish. *S Afr Cancer Bull* 11(4):174-178, 1967.

7259 **RECTAL AND COLONIC SENSATION IN PATIENTS WITH VOLVULUS IN UGANDA.** *(E.)* Shepard, J. J. (Dept. Surg., Makerere U. Coll., Kampala, Uganda) and P. G. Wright. *E Afr Med J* 44(11):451-454, 1967.

7260 **THE SURGICAL TREATMENT OF CANCER OF THE ANUS.** *(Fr.)* Pettavel, J. (Cantonal Hosp., Lausanne, Switzerland). *Praxis* 57(29):1029-1031, 1968.

See also: 6680,6862,6882,6888,6889,6921,6949,7100,7183

7261 THE PRODUCTION OF COLONIC AUTO-ANTIBODIES IN RABBITS BY IMMUNIZATION WITH *ESCHERICHIA COLI.* *(E.)* Cooke, E. M. (Westminister Med. Sch., London, England), M. I. Filipe and I. M. P. Dawson. *J Path Bact* 96(1):125-130, 1968.

Immunofluorescence studies of colonic tissue demonstrated colonic autoantibody production in 11 of 19 rabbits immunized with various strains of *E. coli* isolated from the feces of patients with ulcerative colitis. No morphological changes were observed in the mucosa of the large intestine. However, histochemical studies revealed increased alkaline phosphatase and glucose-6-phosphate dehydrogenase (G6PD) activity in the surface epithelium. This distribution of these enzymes remained normal (alkaline phosphatase in the brush border and supranuclear zone of the epithelial cytoplasm, and G6PD uniformly distributed in the cytoplasm). In the crypts, G6PD activity was especially marked in certain cells which are probably argentaffin cells. Acid and alkaline phosphatase continued to be absent in the crypts after immunization; the reaction for G6PD was weak and that for succinic dehydrogenase was moderate; the nonspecific esterases showed weak to moderate activity in some cells. Histiocytes in the lamina propria showed a slight decrease from normally high alkaline phosphatase activity, while succinic dehydrogenase and nonspecific esterases increased slightly and G6PD increased markedly. No significant changes in muscular or autonomic plexi were observed. These findings cast doubt on the significance of autoantibodies in the pathogenesis of ulcerative colitis. The observed enzyme alterations may indicate early colonic damage.

7262 PROPERTIES OF STRAINS OF *ESCHERICHIA COLI* ISOLATED FROM THE FAECES OF PATIENTS WITH ULCERATIVE COLITIS, PATIENTS WITH ACUTE DIARRHOEA AND NORMAL PERSONS. *(E.)* Cooke, E. M. (Westminster Hosp. Med. Sch., London, England). *J Path Bact* 95(1):101-113, 1968.

The properties of strains of *E. coli* isolated from the feces of 47 patients with ulcerative colitis, 44 patients with acute diarrhea, and 49 normal patients without gastrointestinal disease were compared; feces from 3 other patients with ulcerative colitis failed to yield *E. coli.* Eighteen of the 47 strains from ulcerative colitis patients, 21 of 44 strains from patients with acute diarrhea, and 31 of 49 strains from normal subjects were sensitive to 6 antibiotics tested (ampicillin, colomycin, kanamycin, streptomycin, sulfafurazole and tetracycline). There were 14 necrotoxin-producing strains and 8 hemolysin producing strains from the patients with ulcerative colitis; in the patients with diarrhea, there were 3 and 5 strains, resp.; and in the normal subjects there was 1 and 1 strain, resp. Five of 15 strains from patients with ulcerative colitis and 1 of 15 strains from the normal controls gave severe reactions in ligated segments of rabbit ileum. The strains of *E. coli* from the ulcerative colitis patients also included a larger proportion belonging to a smaller number of 0-groups than the strains from the 2 other sources. Further work is required to clarify the role of pathogenic strains of *E. coli* in ulcerative colitis.

7263 SPECIFIC INHIBITION OF THE *IN VITRO* MIGRATION OF LEUCOCYTES IN ULCERATIVE COLITIS AND CROHN'S DISEASE. *(E.)* Bendixen, G. (State Hosp., Copenhagen, Denmark). *Scand J Gastroent* 2(3 214-221, 1967.

The influence of tissue homogenates prepared from fetal colo mucosa, jejunoileal mucosa, liver and kidney on the *in vitro* gration of peripheral leukocytes was studied in 20 normal co trols, 19 patients with ulcerative colitis or hemorrhagic procti tis, and 14 patients with Crohn's disease. Migration indices (MI) were calculated, based on the average 24-hr migration area in a series of control cultures without antigen and a seri of cultures containing the fetal antigen. An MI of less than 1.0 indicates inhibition and greater than 1.0 indicates stimula tion. In the 20 controls, cell migration was unaffected by colonic or jejunoileal homogenates, with MI = 0.82-1.14 and 0.81-1.09, resp. In the 19 cases with ulcerative colitis or her orrhagic proctitis, subnormal MI values with either colonic o jejunoileal homogenates were seen in 13 cases, all of whom were in active stages of the disease. A normal MI was found in 6 patients, of whom 1 had active disease. In 2 patients w had undergone colectomy, the subnormal MI values returned to normal at 10 and 22 days postoperatively. In the 14 patients with Crohn's disease, migration of leukocytes was unaf fected by colonic and jejunoileal homogenates, as in the controls. The MI was unaffected by preparations from liver and kidney in all cases. These results indicate the presence of cel lular (delayed-type) hypersensitivity to some constituent of normal colonic or jejunoileal mucosa in leukocytes from patients with ulcerative colitis.

7264 THE REACTIVITY OF MESENTERIC VESSEL OF RATS WITH THE SERA OF ULCERATIVE COLITIS PATIENTS. *(E.)* Polcak, J. (Med. Dept., J. E. Purkinje U., Brno, Czechoslovakia), L. Vacek and M. Skalova *Amer J Proctol* 18(5):402-405, 1967.

Application of 0.1 ml of serum from 42 patients with ulcerative colitis to the Wistar rat mesentery adjacent to the ileum produced varying degrees of vasocontriction in the capillaries, precapillaries and arterioles. The reaction was strongest with sera from active forms of the disease (rapid effect lasting several minutes, often with thrombosis in arterial and sometime even in the venous systems) and weakest with sera from patients in remission (slowing of blood flow without vasoconstr tion). An intermediate reaction was provoked by sera from mild forms of the disease. In contrast, the sera of 23 healthy consanguineous relatives produced strongly positive reactions in only 2, poor reactions in 16, and no effect in 5 cases. Th reaction was consistently negative in sera from 26 healthy sub jects. No effect was observed with sera from 134 of 156 patients with various diseases, but slightly positive reactions occurred in lupus erythematosus (5 of 5 cases), rheumatoid arth tis (6 of 7 cases), and diabetes mellitus (11 of 15 cases). Onl Divascol, among several vasoactive drugs tested (atropine, Des Analergin, papaverine), interfered with the constrictive influen of the colitis sera. Electrophoresis of sera from patients with ulcerative colitis yielded 15 fractions; the γ-globulin fractions (No. 12-15) produced a fairly strong vasoconstrictive reaction, while the α_2-globulins had a weak effect. Cohn's fraction I (obtained from citrated plasma using Cohn's method "6") con taining fibrinogen did not produce any change in the vessels but fractions II and III, containing γ-globulin, prothrombin, isoagglutinin and plasminogen, showed fairly strong reactions.

The IV-1 fraction (α-globulins and lipoid substances) did not have any effect. Fraction VI-4, containing α- and β- globulins, yielded a weakly positive reaction.

7265 **TUMORS OF BILIARY EPITHELIUM AND ULCERATIVE COLITIS.** *(E.)* Ham, J. M. (Middlesex Hosp., London, England). *Ann Surg* 168(6):1088-1093, 1968.

7266 **ANAEMIA IN ULCERATIVE COLITIS.** *(E.)* Ormerod, T. P. (Roy Infirm., Sheffield, England). *Proc Roy Soc Med* 61(9):931, 1968.

7267 **NONSPECIFIC ULCERATIVE COLITIS.** *(E.)* Turnbull, R. B. Jr (Cleveland Clin. Found., Ohio), P. F. Schofield and W. A. Hawk. *Advances Surg* 3:161-225, 1968.

7268 **ULCERATIVE COLITIS IN AN AFRICAN.** *(E.)* Sealey, B. J. (U. Rhodesia, Salisbury) and M. Gelfand. *Cent Afr J Med* 14(8):173-175, 1968.

7269 **CLINICAL ASPECTS OF THE PATHOLOGICAL ANATOMY AND PATHOPHYSIOLOGY OF IDIOPATHIC COLITIS.** *(Ger.)* Wittig, G. (Inst. Cancer Res., German Acad. Sci., Berlin) and G. P. Wildner. *Arch Geschwulstforsch* 32(3):249-261, 1968.

7270 **ULCERATIVE COLITIS AND DIVERTICULOSIS OF THE COLON.** *(Cz.)* Kojecky, Z. (Med. Fac., Purkinje U., Olomouc, Czechoslovakia) and L. Benyšek. *Cesk Gastroent Vyz* 21(7):433-435, 1967.

7271 **CURRENT OPINIONS ON ULCERATIVE COLITIS AND REGIONAL ENTERITIS.** *(Cz.)* Mařatka, Z. (Na Bulovce Hosp., Prague, Czechoslovakia). *Cesk Gastroent Vyz* 21(8):499-502, 1967.

7272 **HISTOLOGICAL FINDINGS IN THE RECTUM DURING TREATMENT OF ULCERATIVE COLITIS.** *(Cz.)* Cheli, R. (Inst. Med. Path., U. Genoa, Italy). *Cesk Gastroent Vyz* 21(6):353-355, 1967.

7273 **ULCERATIVE COLITIS FROM THE SURGICAL POINT OF VIEW.** *(Ger.)* Mörl, F. K. (Surg. Clin., U. Tubingen, Germany) and P. Matis. *Med Welt* 18(47):2844-2852, 1967.

PANCREAS

7274 **THE EXOCRINE AND ENDOCRINE FUNCTIONS OF THE PANCREAS IN PATIENTS WITH PEPTIC ULCERS AND CHRONIC GASTRITIS.** *(Rus.)* Borin, Ia. V. (Ivano-Frankovsk Med. Inst., USSR), A. S. Liashkevich and R. P. Makos'. *Vrach Delo* (1):23-27, 1968.

Pancreas enzyme secretion, carbonate alkalinity of the duodenal contents, and glucose tolerance were studied in 38 patients with peptic ulcers (14 with stomach ulcers, 24 with duodenal ulcers), 78 patients with chronic gastritis (48 with gastric hyposecretion and 30 with normal or hypersecretion), and 16 controls. Pancreatic enzyme secretion was pathological in more than half of the patients with peptic ulcers, particularly in those with duodenal ulcers, those with long histories of ulcers, and those with clinical signs of pancreatic insufficiency. Glucose tolerance curves were abnormal in 12 of the 31 ulcer patients studied. Urinary diastase values were not considered a good criterion for relatively minor disturbances in exocrine pancreatic function. Exocrine pancreatic function was reduced in 60% of the patients with chronic gastritis and gastric hyposecretion and was very abnormal in 57% of those with chronic gastritis and gastric hypersecretion. Glucose tolerance curves were abnormal in 29 of the 78 patients with chronic gastritis.

7275 **GASTRIC ACID SECRETION AFTER PANCREATICODUODENECTOMY.** *(E.)* Waddell, W. R. (U. Colorado Med. Sch., Denver) and R. W. Loughry. *Arch Surg* 96(4):574-585, 1968.

Gastric acid secretion tests (betazole hydrochloride, 1.7 mg/kg s. c.) were performed on 14 patients (6 women and 8 men, 33-74 yr old) 6 weeks to 16 yr after pancreaticoduodenectomy. The indications for this procedure were gastric carcinoma with pancreatic extension (1), trauma (1), pancreatic adenocarcinoma (4), common bile duct carcinoma (1), duodenal or ampullary carcinoma (3), nonfunctioning islet cell carcinoma (1), and Zollinger-Ellison syndrome (3). The basal secretion of acid for the 1 patient in whom the antrum was not removed was 54.0 mEq/liter (4.3 mEq/hr), compared to 10.0 mEq/liter (0.5 mEq/hr) in the 5 patients with partial antrectomy and 11.5 mEq/liter (0.6 mEq/hr) in the 8 patients with total antrectomy. After maximal stimulation, the corresponding values were 86, 59 and 36 mEq/liter (16.8, 8.2 and 3.7 mEq/hr), resp. These data indicate that in those patients that had subtotal gastrectomy (with or without vagotomy), basal secretory activity was minimal, although they could respond to stimulation. Limited gastrectomy resulted in higher rates of secretion; in this group there was 1 patient with marginal ulcers, 1 with a gastric ulcer, and 1 who had a bleeding marginal ulcer 8 yr after surgery, but was asymptomatic 8 yr later. It is concluded that pancreaticoduodenectomy with inadequate gastric resection to control secretion is potentially ulcerogenic; however, with either subtotal gastrectomy or antrectomy with vagotomy, the potential for postoperative ulceration is minimal.

7276 **THE EXOCRINE PANCREAS AND PROTEIN-CALORIE MALNUTRITION.** *(E.)* Barbezat, G. O. (Dept. Child Health, U. Cape Town, S. Africa) and J. D. L. Hansen. *Pediatrics* 42(1):77-92, 1968.

Pancreatic function tests, using a dual-tube technique and stimulating the pancreas with secretin and pancreozymin, were performed on 14 children (mean age 25 months) with kwashiorkor (before and after treatment), 7 children with marasmus (mean age 20 months), 10 chronically malnourished children (mean age 102 months) and 8 normal controls (mean age 33 months). Volume output of the pancreas, the pH of the juice and the amylase, lipase, trypsin, chymotrypsin, and ribonuclease output were studied, and the color index of the juice was measured. All patients with kwashiorkor and marasmus received penicillin, sulphadiazine, and potassium chloride for 1 week. Iron, vitamins, and other drugs were given if indicated. Although there was an increase in volume and pH after hormonal stimulation in all groups, there was no difference in these 2 parameters among the groups studied. There was a statistically significant correlation between volume output and body weight. Patients with kwashiorkor and marasmus had grossly deficient enzyme outputs; however, normal outputs were observed after initiation of dietary therapy. The older, chronically malnourished children demonstrated a range of results varying from normal to abnormal. A significant positive correlation was noted between enzyme output and serum albumin concentration. No deficit in bile production was noted among the children with protein-calorie malnutrition.

7277 **EXPERIMENTAL PANCREATICODUODENAL PRESERVATION AND TRANSPLANTATION.** *(E.)* Idezuki, Y. (U. Minnesota Med. Sch., Minneapolis), J. A. Feemster, R. H. Dietzman and R. C. Lillehei. *Surg Gynec Obstet* 126(5):1002-1014, 1968.

A new technique for pancreaticoduodenal transplantation has been developed which preserves both the exocrine and the endocrine function of the graft by allowing the exocrine secretions to drain into the recipient's jejunum. Long-term survival following this type of pancreaticoduodenal allograft was studied in 5 groups of dogs: 1) 19 dogs receiving orthotopic transplants with venous return directed into the inferior vena cava; 2) 4 dogs receiving heterotopic transplants and inferior vena caval return; 3) 3 dogs receiving orthotopic transplants with venous return directed into the portal vein; 4) 6 dogs receiving heterotopic transplants and portal venous return; and 5) 15 dogs receiving heterotopic transplants following *in vitro* preservation of the graft (4C, 3 atmospheres O_2) for 6-24 hr. Postoperative treatment consisted of parenteral chlortetracycline or chloramphenicol for the first 5 postoperative days; azathioprine, 4-8 mg/kg day, was given to some dogs in groups 1, 2, 4 and 5, and methylprednisolone was given when rising blood sugar indicated pancreatic rejection. All dogs in groups 3 and 4 and all those in group 5 receiving grafts which had been preserved for 24 hr died within 10 days. A total of 6 dogs has so far survived 20 days or longer, with and without immunosuppressive therapy. The longest survivals were 98 days in a dog without immunosuppression and more than 150 days in a dog receiving immunosuppression. Normal functioning of the transplant was confirmed by fasting blood sugar levels, glucose tolerance tests, and plasma insulin levels. No significant difference was observed between dogs in which the venous return

rom the pancreas was directed into the inferior vena cava and
hose in which it flowed into the portal venous system. Two
f the grafts were successfully preserved *in vitro* for 22 hr, and
ne dog has survived for more than 5 months with one such
llotransplant. It is felt that pancreatic allotransplantation may
e considered for clinical use.

7278 EXOCRINE FUNCTION OF THE PANCREAS IN PATIENTS WITH VIRAL HEPATITIS. *(Rus.)*

'otekhina, K. L. (L'vov Med. Inst., USSR). *Vrach Delo* (11):
05-108, 1967.

nzyme studies were run on 117 children, 2 yr old or older,
vith viral hepatitis (53 with mild, 48 with moderately severe,
nd 16 with severe forms). During the acute stage, blood dia-
tase values were elevated in 27 cases and reduced in 23; urinary
evels were reduced in 95 cases. There was no strict correlation
etween blood and urinary diastase values, but reduced urinary
evels tended to be associated with normal blood levels and nor-
nal urinary values with high blood levels. No significant changes
ccurred in serum diastase values during recovery. During the
cute period, pancreatic amylolytic activity was studied by de-
ermining blood diastase levels on 34 children before and after
doses of honey taken 1 hr apart. In 15 children, blood dia-
tase levels increased after the first dose and decreased consid-
rably after the second, usually below their initial level. This
s attributed to increased production of the enzyme, which
nters the blood in large quantities because of mechanical com-
ression of the pancreatic ducts and increased vascular perme-
bility. In another group of 17 patients, diastase levels de-
reased after the first dose and increased greatly after the sec-
nd. A lack of response to the first dose and a moderate in-
rease after the second occurred in 1 patient with severe hepa-
itis. Only 1 patient had a normal diastase curve. These devia-
ions in the diastase curves persisted during recovery in all
4 patients. Pancreatic lipolytic activity, studied with the io-
lolipol test on 115 patients, was reduced in 111 cases (96.6%).
During recovery these values increased gradually in all but
9.2% of the patients. These results suggest that the use of
ancreatic enzymes might be beneficial in the treatment of
iral hepatitis in children.

7279 THE ARTERIAL SYNDROME OF CANCER OF THE BODY OF THE PANCREAS. *(Fr.)*

Warter,
. (Hosp. Ctr., U. Strasbourg, France), A. Sacrez, M. O. Spach
nd D. Storck. *Arch Franc Mal Appar Dig* 57(3):289-306,
968.

linical examination of 13 patients with cancer of the head of
he pancreas revealed that 12 had arterial systolic murmurs in
region above the umbilicus, in the left epigastric region, and
n the left hypochondriac region. The diagnosis was confirmed
t surgery or autopsy in 12 cases. In 11 of these cases phono-
ngiograms were obtained which had spindle-shaped or rhom-
oid tracings characteristic of arterial compression. In 11 cases
eliac arteriograms or autopsy showed the presence of several
rterial stenoses produced by tumors. These were located in
he aorta in 1 case, the celiac trunk in 5, at the bifurcation of
he celiac artery in 2, in the hepatic aretery in 5, the splenic
rtery in 9, the gastric coronary in 1, the upper mesenteric
rtery in 5, and the left renal artery in 1. These stenoses ac-
ount for the murmurs heard on auscultation.

7280 PANCREAS TRANSPLANTS IN DOGS: A STUDY OF THE SURVIVAL AND FUNCTIONAL CAPACITY OF THE TRANSPLANT. *(Fr.)*

Grenier, J. F. (U. Hosp. Ctr., Strasbourg, France), M. Gillet, G. Santizo Lepe, A. Klein, A. M. Barth and A. G. Weiss. *Ann Chir* 21(25/26):1523-1537, 1967.

Autologous and homologous transplants of the pancreas to the neck were performed in 13 and 37 pancreatectomized dogs, resp. With regard to survival time and function, homologous transplantation of a complete duodenopancreatic segment was preferable to a partial transplantation of the organ, as required in all cases of autologous transplantation. The mean survival time of autologous transplants was 11 days, compared to 7.5 days for the homografts. In most cases, death resulted from local infection, thrombosis or the effects of pancreatectomy. The transplants continued to function throughout the survival period, with a tendency to hypoglycemia revealed by means of the glucose tolerance test. Rejection was first indicated by an increase in blood amylase and lipase levels, especially marked in secretin-induced pancreatic activity tests. This was followed by a decrease in the output of pancreatic juice and in pancreatic enzyme activity. Signs of rejection were seen in the acini before the islets of Langerhans. In cases of long survival, the pancreas often showed pronounced signs of fibrosis.

7281 PANCREATIC FUNCTION AND THE ZINC CONTENT OF THE BLOOD, URINE AND FECES IN PATIENTS WITH ENDEMIC GOITER. *(Rus.)*

Gerasimenko, N. I. (Ivano-Frankovsk Med. Inst., USSR) and N. D. Iegorushkina. *Vrach Delo* (1):127-128, 1968.

7282 DIAGNOSIS AND TREATMENT OF MUCOVISCIDOSIS. *(Fr.)*

Hennequet, A. (Hosp. Sick Child., Paris, France). *Entret Bichat [Med]* pp. 455-458, 1967.

7283 PANCREATIC PSEUDOCYSTS WITH ABSCESS FORMATION. *(E.)*

Nassar, J. A. (VA Hosp., San Juan, Puerto Rico), L. Ehrlich and C. G. Acosta. *Bol Asoc Med P Rico* 60(9):443-446, 1968.

7284 PSEUDOCYSTS OF THE PANCREAS. *(E.)*

Venkataraman, M. S. (Govt. Gen. Hosp., Madras, India), B. Bhaskaracharya, K. Chandrasekaran, S. Sundararaman and M. S. Ramkrishna. *J Indian Med Ass* 51(2):71-76, 1968.

7285 PANCREATIC PSEUDOCYST ENCAPSULATING SPLEEN AND PRESENTING AS SUBPHRENIC ABSCESS. *(E.)*

Laughlin, E. H. (Huntsville Hosp., Alabama). *J Med Ass Alabama* 38(4):331-333, 1968.

7286 DIAGNOSTIC STUDIES IN CARCINOMA OF THE PANCREAS: A REVIEW OF 100 PATIENTS. *(E.)*

Brown, C. H. (Cleveland Clin. Found., Ohio), H. Rastogi, R. Alfidi and A. Rodriguez-Antunez. *J Louisiana Med Soc* 120(9):375-385, 1968.

7287 PARANEOPLASTIC ENDOCARDITIS CAUSED BY CARCINOMA OF THE PANCREAS [CASE

REPORT]. *(Fin.)* Toivanen, A. (Turku U. Med. Sch., Finland), U. Wegelius and M. Möttönen. *Duodecim* 83(22):1294-1297, 1967.

7288 **EFFECT OF NUTRITION ON THE FUNCTION AND MORPHOLOGY OF THE RAT PANCREAS. 1. EFFECT OF STARVATION.** *(Sl.)* Bučko, A. (Res. Inst. Human Nutrit., Bratislava, Czechoslovakia), Z. Kopec and J. Babala. *Cesk Gastroent Vyz* 21(6):369-375, 1967.

7289 **EFFECTS OF EXPERIMENTAL HYPERCALCEMIA ON THE PANCREAS IN DOGS.** *(Sp.)* Milanese, J. C. (Ramos Mejia Hosp., Buenos Aires, Argentina) and C. J. Morel. *Rev Argent Cir* 13(4/5/6):113-116, 1967.

7290 **PANCREATIC INSUFFICIENCY AND STEATORRHEA.** *(Fr.)* Kiekens, R. (Surg. Clin., Brugmann Hosp., Brussels, Belgium), J. P. Govaerts and P. Wissocq. *Acta Gastroent Belg* 30(11):711-720, 1967.

7291 **BASIC SCIENCE AND PANCREATIC SURGERY. A STUDY IN ANATOMY, PHYSIOLOGY AND PATHOLOGY.** *(E.)* Smith, R. *Trans Med Soc London* 84: 110-147, 1968.

7292 **SERUM ANTISTAPHYLOCOCCAL ALPHA-HEMOLYSIN TITERS IN CYSTIC FIBROSIS.** *(E.)* Iacocca, V. F. (Child. Hosp., Philadelphia, Pa.) and G. J. Barbero. *Arch Dis Child* 43(228):220-222, 1968.

7293 **MASSIVE PANCREATIC ASCITES.** *(E.)* Parrish, R. A. (Med. Coll. Georgia, Augusta), A. L. Humphries and W. H. Moretz. *Arch Surg* 96(6):887-891, 1968.

7294 **PANCREATIC FISTULAS RESULTING FROM SURGICAL PROCEDURES.** *(Ger.)* Eichelter, P. (2nd Surg. Clin., U. Vienna, Austria) and L. Howanietz. *Bull Soc Int Chir* 27(2):139-142, 1968.

7295 **PERSONAL TECHNIQUES FOR THE LOCAL USE OF TRASYLOL IN PANCREATIC INJURIES EXPERIMENTAL AND CLINICAL RESULTS.** *(Ger.)* Dorobisz, T. (1st Surg. Clin., Med. Acad. Wroclaw, Poland) and M. Grzebska. *Bull Soc Int Chir* 27(2):125-132, 1968.

7296 **AN EXPERIMENTAL STUDY ON THE SIGNIFICANCE OF THE EFFERENT LYMPHATIC PATHWAY FROM THE PANCREAS IN PATHOPHYSIOLOGY.** *(Fr.)* Murat, J. (Ed. Herriot Hosp., Lyon, France), M. Barbier and P. Mallet-Guy. *Bull Soc Int Chir* 27(2):94-100, 1968.

7297 **SURGICAL DISEASES OF THE PANCREAS.** *(Fr.)* Moreaux, J. (Bichat Hosp., Paris, France), H. Bismuth and C. Hernandez. pp. 210-226 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

See also: 6840,6866,6872,6875,6880,6935,7091,7190,7416

7298 THE TREATMENT OF ACUTE HEMORRHAGIC PANCREATITIS BY SUMP DRAINAGE. *(E.)*

Waterman, N. G. (U. Louisville Sch. Med., Ky.), R. Walsky, M. L. Kasdan and B. L. Abrams. *Surg Gynec Obstet* 126(5): 963-971, 1968.

Case reports are presented for 10 patients (6 men and 4 women, 18-78 yr old) with acute hemorrhagic pancreatitis treated by surgical drainage of the pancreas and the lesser sac with a triple-lumen apparatus (double-lumen sump or 2 side-by-side, rubber urethral catheters, placed inside the lumen of a large Penrose drain with multiple perforations), continuous gastric suction, fluid replacement, antibiotics, and steroids (when necessary). There was only 1 death among the 10 patients with this form of treatment (10% mortality), in contrast to much higher mortality rates (35-75%) in earlier reports in which either nonsurgical treatment or double-lumen sump drainage was used. The more favorable results in this series were due to the fact that the triple-lumen sump apparatus remained patent and functional, whereas double-lumen sump drains often become occluded with debris, necrotic tissue, and blood clots. The effectiveness of triple-lumen sump drainage was demonstrated by the results of experimentally induced pancreatitis in 40 guinea pigs (pancreatic injection of bile salts and trypsin). Sump drainage was used in 32 animals, and 18 served as untreated controls. All the controls, except 1, were dead within 2 days; 26 of the 32 treated animals were alive on the 3rd day, and 18 were alive on the 7th day. Five of the 6 treated animals that died within 3 days had intraabdominal displacement of the drainage apparatus; the remaining deaths were due to infection. Improvement in the results of treatment of acute hemorrhagic pancreatitis can be expected with the use of triple-lumen sump drainage.

7299 THE COURSE OF CHRONIC RECURRENT PANCREATITIS IN ELDERLY PATIENTS.

(Rus.) Volkova, L. P. (Leningrad Sanit. Hyg. Med. Inst., USSR). *Vestn Khir Grekov* 100(6):51-55, 1968.

A clinical study was made of 113 patients (30 men and 83 women, aged 50-94 yr), including 96 with chronic recurrent, 10 with pseudocancerous, 5 with pseudocystic, and 2 with calcareous pancreatitis. Cardiovascular disorders present in 34 cases included generalized atherosclerosis and atherosclerotic cardiosclerosis. Metabolic disorders included liver function disturbances in 59 cases, hypoglycemia in 16, hyperglycemia in 19 (diabetic glucose tolerance curves in 29), and hypochlorhydria in 36. Trypsin activity was very low in 96 patients and amylolytic activity was low in 47; lipolytic activity was reduced, but not to such a great extent, in 21 cases. Blood diastase levels were elevated in 35 cases and urinary diastase levels in 23, but there was no correlation between the blood and urinary values. Morphological abnormalities of the gastrointestinal tract, found in 47 cases, included perigastritis, periduodenitis, uncoiling of a loop of duodenum, duodenal malformations, and papillitis. Functional changes, present in 62 cases, included hypo- and hypertonic duodenal stasis, bulbar stasis, and duodenal dyskinesia. Thirty-four operations were performed on patients with chronic recurrent pancreatitis and pain syndrome, including laparotomies and removal of adhesions in 17 cases, gastric resections on 3 patients with peptic ulcers, postceliac neurotomies in 8 cases, and anastomoses between the biliary tract and intestine in 2 cases. Cholecystoduodenostomies were performed on 4 patients with pseudocancerous pancreatitis and a choledochoduodenostomy on 1. Cysts were drained in 3 patients with pseudocystic pancreatitis and a cystogastrostomy performed on 1. One patient with calcareous pancreatitis underwent a pancreatectomy and the other, a cholecystoduodenal anastomosis. Three-year follow-ups showed that good to satisfactory results were obtained in 31 operated patients. Some patients developed recurrences of pain and dyspepsia and patients with calcareous pancreatitis showed signs of progressive pancreatic fibrosis and sclerosis.

7300 SHUNT OPERATIONS IN CHRONIC PANCREATITIS: REPORT OF 33 CASES. *(Fr.)*

Doutre, L. P. and J. Périssat. *Gaz Med France* 75(17):3557-3561, 1968.

In 31 of the 33 cases, pancreatitis was localized primarily or exclusively to the right side. In 2 cases, the tail was involved, and these 2 patients underwent left pancreatectomies. The others underwent pancreaticojejunostomies (29 cases) or anastomoses between a pseudocyst and the jejunum (2 cases). There were 7 operative deaths: 1 from bleeding in a patient with cirrhosis and a splenic thrombus, 2 in patients with cachectic malnutrition, and 4 in patients with symptoms of encephalopathy 3-7 days after surgery. At the end of 1 yr, 21 of 23 patients followed were in good condition, but at the end of 3 yr only 12 of 18 had regained their weight, had undertaken their previous activities, and no longer had any symptoms.

7301 A CLINICAL TRIAL OF AN ENZYME INHIBITOR IN PANCREATITIS. *(E.)*

Kneisel, J. J. (Yale U. Sch. Med., New Haven, Conn.). *Arch Surg* 96(3): 442-449, 1968.

Of 23 patients with moderately severe to severe acute pancreatitis, 9 received Trasylol i.v. (2 received 100,000 KIU daily and 7 received 400,000 KIU daily) and 14 were treated conventionally (controls). A total of 4 patients died: 2 of the controls and 2 that received the lower dosage of Trasylol. No statistically significant differences were noted in the daily temperature or the changes in temperature among the 2 groups. The mean change in pulse rate over the 1st 5 days of treatment was -11 beats/min for the Trasylol-treated group and +0.8 beats/min for the controls (statistically significant, $P < 0.001$). The mean changes in blood pressure over the 1st 5 days were the same for both groups. There was a faster fall in the serum amylase levels in the Trasylol treated patients (6 days) than in the controls (9-14 days); however, similar prompt falls (4-6 days) were seen in the patients who died. It is felt that the continued testing of high enzyme inhibitor dosages early in the course of severe acute pancreatitis is warranted.

7302 THE PROGNOSIS FOR PATIENTS OPERATED ON FOR CHRONIC PANCREATITIS: THE MEDICAL POINT OF VIEW. *(Fr.)*

Debray, C. (Bichat Hosp., Paris, France), J. P. Hardouin, B. Gouin and J. M. Bouillier. *Arch Franc Mal Appar Dig* 56(7/8):621-644, 1967.

A prospective study was carried out on 46 patients (43 men, 3 women) with chronic pancreatitis, including 36 with calcareous pancreatitis (33 men, 3 women) and 10 with noncalcareous pancreatitis. Thirty of these 46 patients had a history of alcoholism and 1 a history of familial pancreatitis; 3 had associated cirrhosis and 1, hyperparathyroidism. Their age at onset of symptoms ranged from 12-62 yr. Surgery was performed on 26 of the 36 patients with calcareous pancreatitis and on all 10 of those with noncalcareous pancreatitis. Symptoms disappeared or improved in 6 of the 10 patients with calcareous pancreatitis who did not undergo surgery and remained stationary in 4. Glucose tolerance tests were abnormal in 8 of these 10 patients. Cholangiography showed compression of the common bile duct in 5, but none ever developed biliary tract complications. There were 2 operative deaths among the 36 operated patients: 1 was a patient with severe pancreatitis and 2 pseudocysts treated by sphincterotomy and cholecystectomy, and the other a cachectic reoperated patient subjected to left hemipancreatectomy with splenectomy. Three deaths occurred 1-3 yr after surgery due to a thrombosis of the vena cava, progressive cirrhogenous posttransfusional hepatitis, and associated decompensated cirrhosis. There were 15 good results and 15 failures among the 30 surviving patients. Glucose tolerance tests were abnormal in 23 of the 25 tested and diabetic in 12. No improvements in glucose regulation occurred after surgery, but digestive disorders were not aggravated. None of the surgically treated patients developed tuberculosis or signs of bone calcification. Good results were obtained in 2 of 5 splanchnicectomies, 2 of 3 Oddi sphincterotomies, 6 of 12 cases in which the pancreatic duct was drained, 3 of 8 with pancreaticojejunal anastomoses, 4 of 11 pancreatectomies, 0 of 3 cases in which a pseudocyst was drained, and 1 of 1 with a cholecystectomy. Disorders completely disappeared for 3 yr in a patient who underwent a palliative gastroenterostomy for duodenal compression and a choledochoantrostomy. These results suggest that chronic pancreatitis should be treated medically since surgery can relieve only the results of pancreatitis and not its cause.

7303 **LUMBAR RETROPERITONEAL BLOCK OF THE PANCREAS WITH ANTIBIOTICS AND PROCAINE.** *(Rus.)* Roman, L. I. (Chernovtsy Med. Inst., USSR). *Khirurgiia (Moskva)* 44(3):124-129, 1968.

When 100-250 ml of dye or contrast medium were injected into the perirenal adipose capsule of cadavers, as procaine is in Vishnevskii's paranephric block, none of it passed beyond the adrenals to the pancreas. Solutions injected by the presacral retroperitoneal route reached the pancreas only after injection of massive amounts (up to 500 ml and more). However, when 100-150 ml were injected retroperitoneally in the lumbar region of 110 cadavers, dye or contrast medium reached the fascial sheaths of the pancreas in 101 cases. Failure of these solutions to reach the pancreas was attributed to errors in technique in 3 cases and to gross anatomical changes in the retroperitoneal tissue in 6. Bilateral lumbar retroperitoneal blocks of the pancreas were then performed in 65 patients with chronic and acute pancreatitis and pancreatitis associated with cholecystitis by injecting 150 ml of 0.25% procaine containing 500,000 units of penicillin and streptomycin on each side. Blocks were performed once in 27 cases, twice in 16, and 3-5 times in 22. Within 15-20 min abdominal pain and vomiting stopped. Cardiovascular function improved, temperatures and peripheral blood pictures returned to normal, and urinary diastase values decreased. Repeated blocks combined with conventional therapy for pancreatitis produced rapid recoveries in some patients. This technique gave better results than Vishnevskii's paranephric block. However, it can only be regarded as symptomatic therapy for patients who have constant or periodically exacerbated pain.

7304 **A COMPARISON OF THE CLINICAL AND HISTOPATHOLOGICAL DIAGNOSIS OF CHRONIC PANCREATITIS DEVELOPING DURING THE COURSE OF DISEASES OF THE BILIARY TRACT, GALLBLADDER, STOMACH AND DUODENUM.** *(Pol.)* Torzecka, W. (1st Clin. Int. Dis., Acad. Med., Lodz, Poland). *Ann Acad Med Lodz* 9:63-126, 1967.

Clinical studies on 54 patients (22 men and 32 women, 27-81 yr old) who were operated on for diseases of the gallbladder (17 cases), peptic ulcers (20 cases) or other diseases (17 cases) revealed signs of pancreatic insufficiency and/or chronic pancreatitis in about 80%. Chronic pancreatitis was tentatively diagnosed in 14 of the 1st group, 17 of the 2nd and 10 of the 3rd (although 13 of these diagnoses were borderline). Howeve microscopic examination of pancreatic tissue obtained by biops during surgery revealed actual inflammation in only 21 cases (2 of the 1st group, 8 of the 2nd and 11 of the 3rd), includin 5 which had shown no clinical signs of pancreatitis. In 20 othe cases (13 of the 1st group, 6 of the 2nd and 1 of the 3rd), microscopic examination revealed pancreatic dysfunction ("dyschylia") which presumably accounted for the clinical symptom Signs of pancreatic inflammation wre also noted during autops in 46 of 60 patients (33 men and 27 women, 21-80 yr old) who died of hepatobiliary disease, 22 of 34 patients (24 men and 10 women, 27-86 yr old) who died of gastroduodenal disease, and 4 of 29 controls (15 men and 14 women, 3-83 yr old) who died of diseases not affecting either the gastroduodenal or the hepatobiliary tract. Histopathological signs of pancreatic dysfunction were noted in 4, 2 and 0 cases in these 3 groups. It is concluded that chronic pancreatitis cannot be differentiated from pancreatic "dyschylia" on the basis of clinical symptoms. The literature on the etiology, pathogenesis, and clinical manifestations of chronic pancreatitis is reviewed at length.

7305 **MARGINAL NEURECTOMY IN THE TREATMENT OF CHRONIC RECURRENT PANCREATITIS WITH PAIN SYNDROME.** *(Fr.)* Napalkov, P. N. (Inst. Med. Hyg., Leningrad, USSR), M. A. Trunin and I. F. Krutikova. *Lyon Chir* 63(6):801-811, 1967.

In a study of 20 human cadavers and 25 unspecified laborator animals, it was demonstrated that the pancreas is innervated b branches of the solar plexus, by the posterior gastric plexus, and by perivascular nerves of the hepatic, splenic, and mesenteric arteries. Most of the nerves reaching the pancreas are associated with branches of autonomic nerves innervating the walls of arteries which form a closed frontal plexus around the pancreas. Nerves enter the pancreas through its periphery. Thus, the effects of postganglionic neurotomies are limited to the pancreas. On the basis of these findings, a surgical procedure was developed in which nerves located between the trunk of the hepatic and splenic arteries along the upper edge of the pancreas are resected. In some cases it becomes necessary o expose the roots of the hepatic and splenic arteries although his could disturb the innervation of the liver, biliary tract, and spleen. Nerves along the upper and lower edges of the pancreatic head are dissected when lesions are limited to the head of he pancreas. Neurotomies are performed along the splenic artery when lesions are located in the body and tail. This procedure is useful for lesions located in the head and the right half of the pancreas when none of the adjacent organs are involved. Based on short-term follow-ups, encouraging results were obtained in 18 patients (15 women and 3 men, aged 26-1 yr) on whom this procedure was performed.

7306 DIFFUSION OF ENZYMES IN ACUTE PANCREATITIS: A CLINICAL AND EXPERIMENTAL STUDY.

(Fr.) Grenier, J. F. (Surg. Clin. B, Strasbourg, France), M. Gillet, M. Hatano and A. G. Weiss. *Arch Franc Mal Appar Dig* 56(9):773-791, 1967.

Lipase and amylase levels in various body fluids were measured n 170 dogs with pancreatitis induced by injection of bile into he pancreatic ducts and in 37 humans with clinical pancreatitis. In humans, hyperamylasemia and hyperlipasemia were not found consistently, and were unrelated to prognosis. Variation appeared in only 10 of 30 cases, reaching a peak at 48 hr, then declining to normal values. In dogs, amylasemia appeared 8 min after initiation of pancreatitis, reached a peak in from 15 min-1 hr, and declined after 4 hr; lipasemia appeared at 9-15 min, reached a maximum at 4 hr, and did not begin to decline until after 6 hr. The concentration of each enzyme was the same in the portal vein and in the inferior vena cava within the first 6 hr. Simultaneous enzyme measurements in the thoracic duct, pleural fluids and pericardial fluids indicated rapid direct passage from the pancreas to the thoracic duct. This occurred n addition to rapid local diffusion into the retroperitoneal space and peritoneal cavity (15 min). The results suggest that enzyme diffusion through the blood in acute pancreatitis is secondary to lymphatic and direct local diffusion.

7307 ENZYME TOXICITY IN ACUTE PANCREATITIS: A CLINICAL AND EXPERIMENTAL STUDY.

(Fr.) Grenier, J. F. (Surg. Clin. B, Strasbourg, France), M. Gillet, F. Oberling and A. G. Weiss. *Arch Franc Mal Appar Dig* 56(9):793-819, 1967.

The correlation between blood amylase and lipase levels and the symptoms and mortality rate of induced pancreatitis was studied in 111 dogs treated by various means. Animals treated with i.v. Trasylol (10), thromboclase (12) or pancreatectomy (9) showed 92-100% mortality, as did untreated controls. Animals treated with i.v. corticoids (12) showed 66% mortality, and those treated with i.p. Trasylol (11), i.v. or i.p. chlortetracycline (22), or heparin (12) showed 42-45% mortality. Treatment with a combination of agents reduced mortality to 8%, but these responses were unrelated to blood amylase levels (highest levels were found in the group treated by combined therapy). Moreover, the severity of pancreatic lesions did not correlate with high blood amylase levels. A study of 30 cases of acute pancreatitis in humans revealed 6 cases in which patients showed normal enzyme levels but mental or renal disturbances usually attributed to enzyme toxicity and 4 cases in which enzyme levels rose in the absence of pancreatic lesions and toxemia. Thus, despite previous views that enzyme toxicity may be an important element in the pathology of acute pancreatitis, there is strong experimental and clinical evidence that it is not.

7308 PHOSPHOLIPASE ACTIVITY IN PANCREATIC EXUDATE IN EXPERIMENTAL ACUTE PANCREATITIS.

(E.) Gjone, E. (U. Hosp., Oslo, Norway), E. Ofstad, P. F. Marton and E. Amundsen. *Scand J Gastroent* 2(3):181-185, 1967.

After acute pancreatitis was induced in dogs by injecting a mixture of taurocholate and trypsin into the pancreatic duct, the pancreatic exudate was collected outside the body wall by a special technique over separate 1-hr intervals. All exudates were able to break down lecithin by phospholipase and lysophospholipase action. Calcium, bile, and taurocholate stimulated while EDTA inhibited the degradation of lecithin. Phospholipase activity showed a maximum in the 1st hourly collection with a leveling off during the 4-7 hr periods. Amylase activity paralleled the phospholipase activity of the exudate. The demonstration of an early release of lecithin-hydrolyzing enzyme systems in acute pancreatitis may indicate an important role of these enzymes in the pathogenesis of acute pancreatitis by cytolysis and the liberation of cellular toxins.

7309 PHYSIOPATHOLOGICAL CORRELATIONS BETWEEN PANCREATITIS AND HYPERPARATHYROIDISM: OBSERVATIONS ON 15 CASES.

(It.) Cariati, E. (Inst. Surg. Path., U. Genoa, Italy) and U. Valente. *Rass Arch Chir* 5(6,bis):482-487, 1967.

Blood calcium levels were usually increased, phosphorus levels were low or unchanged and urinary phosphate and calcium levels were increased in 15 patients with chronic pancreatitis, confirmed histologically in 6 cases. These changes are attributed to parathyroid hyperfunction secondary to the pancreatitis. Low blood calcium levels found in 2 patients in the early stages of pancreatitis were probably due to the absence of parathyroid hyperfunction and those found in 3 other patients are attributed to malabsorption. In a 47-yr-old woman with a parathyroid adenoma, pancreatitis was believed to be secondary to hyperparathyroidism. She had osteoporosis, joint pains, hypercalcemia, cholelithiasis, and pancreatic insufficiency due to chronic pancreatitis. After removal of the 2 upper parathyroids, symptoms of hyperparathyroidism gradually and progressively improved, blood calcium levels decreased, and pancreatic insufficiency improved.

7310 RENAL INVOLVEMENT DURING ACUTE EXPERIMENTAL PANCREATITIS.

(It.) Pasquini, F. (Inst. Spec. Surg. Path., U. Perugia, Italy), P. Furiosi and A. Gerardi. *Gazz Int Med Chir* 72(24,bis):2978-2998, 1967.

7311 ACUTE PANCREATITIS: CONSIDERATION OF THE CLINICAL PICTURE IN 63 CASES.

(Sp.) Introzzi, A. Jr. (San Martin Polyclin., Argentina) and C. Dallorso. *Rev Argent Cir* 13(4/5/6):121-122, 1967.

7312 RENAL AND HEPATIC PATHOLOGY IN HEMORRHAGIC PANCREATITIS.

(E.) Sussman, H. M. (Polyclin. Hosp., New York, N. Y.), E. Reyes, A. Van

Caneghem, W. E. Finkelstein and A. M. Gelb. *Amer J Gastroent* 50(4):315-321, 1968.

7313 **CHYLOUS ASCITES COMPLICATING CHRONIC PANCREATITIS.** *(Fr.)* Blanchon, P. (Bicetre Hosp., Kremlin Bicetre, France) and J. Dupuis. *Entret Bichat [Med]* pp. 479-484, 1967.

7314 **THE COURSE OF CHRONIC PANCREATITIS AFTER SURGERY.** *(Fr.)* Debray, C. (Bichat Hosp., Paris, France), J. P. Hardouin and B. Gouin. *Entret Bichat [Med]* pp. 485-490, 1967.

7315 **ENZYME INHIBITION IN THE TREATMENT OF ACUTE HEMORRHAGIC PANCREATITIS.** *(Turk.)* Degerli, U. (Haydarpasa Milit. Hosp., Istanbul, Turkey), I. Ünlü and N. Özden. *Turk Tip Cem Mec* 34(6):377-384, 1968.

7316 **DIAGNOSTIC DIFFICULTIES ENCOUNTERED IN FOUR CASES OF ACUTE PANCREATITIS.** *(Turk.)* Konuralp, H. Z. (Med. Fac., U. Istanbul, Turkey), S. Belgerden, B. Görgün and O. Arioglu. *Turk Tip Cem Mec* 34(6):361-368, 1968.

7317 **A CASE OF ACUTE TYPHOID PANCREATITIS VERIFIED BY SURGERY AND SUBSEQUENTLY CURED.** *(Fr.)* Monod-Broca, P., P. Auzépy, G. Lesourd and C. Goumet. *Mem Acad Chir (Paris)* 93(24/25):779-783, 1967.

7318 **SPONTANEOUS HEMOPERITONEUM APPEARING DURING SEGMENTAL PORTAL HYPERTENSION CAUSED BY CHRONIC PANCREATITIS.** *(Fr.)* Guillemin (Lyon, France), H. Viard, P. Mikaeloff, J. Cuilleret and Y. Sauvage. *Mem Acad Chir (Paris)* 93(24/25):737-742, 1967.

7319 **THE PATHOGENESIS AND TREATMENT OF ACUTE PANCREATITIS.** *(Rus.)* Kozlov, I. Z. (I. M. Sechenov 1st Moscow Med. Inst., USSR) and G. N. Akzhigitov. *Sovet Med* 30(10):50-55, 1967.

7320 **PROPHYLACTIC AND LATE CORTICOTHERAPY AND ANTI-ENZYME THERAPY IN ACUTE EXPERIMENTAL PANCREATITIS.** *(Sp.)* Cumsille, E. (J. J. Aguirre Hosp., Santiago, Chile), R. Agar, E. Waisberg, A. Ubilla and F. Solis. *Rev Med Chile* 96(4):239-245, 1968.

7321 **ANABOLIC STEROIDS IN THE TREATMENT OF PATIENTS WITH CHRONIC PANCREATITIS.** *(Rus.)* Tuzhilin, S. A. (Moscow Clin. Hosp., MPS, USSR), V. A. Shaternikov, A. A. Petrova and T. A. Tsagikian. *Vrach Delo* (10):36-39, 1968.

7322 **THE RADIOLOGICAL FINDINGS IN PANCREATITIS: AN ANALYSIS OF 120 CASES.** *(Sp.)* Pitol, C., A. (Hosp. Nutrit. Dis., Mexico City, Mexico). *Gac Med Mex* 97(1):52-68, 1967.

7323 **THE SEARCH FOR INHIBITORS OF THE PANCREAS: PRELIMINARY REPORT.** *(Sp.)* Alvizouri, M. (Munic. Hosp., Morelia, Mich., Mexico). *Gac Med Mex* 97(6):719-726, 1967.

7324 **A CASE OF ACUTE PANCREATITIS WITH GANGRENE OF THE DUCT OF WIRSUNG.** *(Fr.)* Lahaut, J. and R. Dargent. *Mem Acad Chir (Paris)* 93(22):669-674, 1967.

7325 **REESTABLISHMENT OF PANCREATICODUODENAL FLOW VIA THE DUCT OF WIRSUNG IN CERTAIN FORMS OF CHRONIC PANCREATITIS.** *(Fr.)* Lortat-Jacob, J. L. (Paris, France) and C. A. Richard. *Bull Soc Int Chir* 27(2):101-109, 1968.

7326 **A COMPARATIVE STUDY OF PANCREATIC ENZYME SECRETION AND THE RESULTS OF PANCREATOGRAPHY FOLLOWING DRAINAGE OF THE DUCT OF WIRSUNG.** *(Sp.)* Artigas, V. (Santa Cruz y San Pablo Hosp., Barcelona, Spain), M. T. Ribera and J. Puig-Lacalle. *Bull Soc Int Chir* 27(2):110-113, 1968.

7327 **SURGERY IN ACUTE PANCREATITIS.** *(Ger.)* Asp, K. (2nd Surg. Clin., U. Helsinki, Finland). *Bull Soc Int Chir* 27(2):133-138, 1968.

7328 **SIDE-TO-SIDE PANCREATICOJEJUNOSTOMY WITH CAUDAL PANCREATECTOMY FOR TREATMENT OF CHRONIC PANCREATITIS WITH CALCULI.** *(E.)* Sato, T. (Tohoku U. Sch. Med., Sendai, Japan), Y. Saitoh and T. Maki. *Arch Surg* 96(6):892-898, 1968.

7329 **PANCREATITIS FOLLOWING OPERATIONS ON THE STOMACH.** *(E.)* Abasov, I. T. (Azerbaijan Inst. Roentgen., Baku, USSR). *Arch Surg* 96(6):909-914, 1968.

See also: 6871,6992,7513

LIVER AND BILIARY TRACT

GENERAL

30 **EARLY CHANGES IN DIET-INDUCED FATTY**
LIVERS OF MICE. *(E.)* Wilson, J. L. (U.
ssissippi Sch. Med., Jackson), A. D. Ashburn and W. L.
lliams. *Anat Rec* 161(1):23-35, 1968.

high-fat (28% lard), low-protein (8% casein), lipotropic-defi-
nt diet was fed to Taconic Swiss mice (50 males, 104 nullipar-
s females; 30-48 days old) for 1-90 days. Animals were sac-
ced at various times up to 90 days and the livers were ex-
ined under the microscope. Up to 21 days, parenchymal
osis was greatest in the peripheral and outer middle zones of
lobule; after 21 days, fat increased in the central and mid-
zones and decreased in peripheral zones. Within 12 days
oid pigment developed in the Kupffer cells; within 31-45
ys these cells coalesced and formed large ceroid globules
ich increased in size and number up to 90 days. Reticular
rosis surrounded the masses of ceroid. Stromal distortion
nsisting chiefly of compression of reticulum by fat-laden
patocytes was prominent in peripheral lobular zones within
lays. Sinusoids were obstructed and dilated by this process;
ne became nonfunctional and were transformed into fibrous
rds. With progressive liposis (after 30 days), irregular stromal
anges developed in all zones of the lobules. There were no
nificant lesions in the kidneys, adrenal glands, smooth mus-
or reproductive organs. The changes seen were unrelated
the sex or age of the mice.

31 **EXPERIMENTAL ALTERATIONS IN THE POR-**
TAL CIRCULATION. *(Dut.)* Klopper, P. J. (Lab.
p. Surg., U. Amsterdam, Netherland). *T Gastroent* 11(5):512-
3, 1968.

experiments on anesthetized rabbits, acute occlusion of the
rtal vein was consistently followed by a rapid increase in por-
pressure and a drop in arterial pressure, terminating in death
an average of 37.7 ± 19.9 min. The survival time could be
reased significantly (to 666 ± 516.4 min) by prophylactic
nstruction of a splenoportal shunt. In both rabbits and dogs,
dual ligation of the portal vein was well tolerated, even
ough accompanied by a progressive rise in portal pressure. In
gs, complete occlusion could be achieved in an average of 23
ys, resulting in an increase in portal pressure of 14.3 cm H_2O.
rtal hypertension produced in this way could be alleviated by
nstructing a parenchymal shunt between the liver and spleen,
d could be prevented by a prophylactic splenorenal shunt. In
gs, construction of an anastomosis between the renal vein and
portal vein was followed by an increase in the volume of
ood in the portal system and a consequent slight rise in por-
pressure; however, neither this technique nor occlusion of
portal vein by cellophane are considered to be effective ways
producing experimental portal hypertension. A splenoportal
unt may be an effective approach to the treatment of portal
pertension in patients in whom the splenal vein is too small
permit a splenorenal shunt.

32 **HEPATIC MORPHOLOGIC CHANGES FOLLOW-**
ING END-TO-SIDE PORTACAVAL SHUNT IN
OGS. *(E.)* Lanier, V. C. Jr. (Vanderbilt U. Med. Ctr.,
shville, Tenn.), R. D. Buchanan and J. H. Foster. *Amer*
rg 34(3):185-195, 1968.

Liver biopsies were made before at 10-day intervals for 60 days and at 90 days after end-to-side portacaval shunt in 10 dogs. Microscopic examination revealed centrolobular fatty degeneration at 10 days post-shunt which progressed throughout the liver by 30 days. During the first 20 days there was a collapse of the larger portal veins associated with an increase in periportal vascular channels communicating between the hepatic arterioles, sinusoids and portal veins. In addition, the connective tissue around the portal triads gradually increased during this period. At 30-40 days post-shunt, individual hepatocytes appeared very atrophic. A prominent finding was the continued increase in periportal vascular channels with associated hyperplasia of the arterioles. Vascular changes were less marked in sections showing the greatest degree of fatty change. Glissom's capsules appeared thickened and there were areas of subcapsular vascularization which often extended into the hepatic parenchyma terminating at portal triads. Reticulum studies showed evidence of arterial hypertrophy. From 50-90 days, the degree of further morphologic change was minimal. The portal triads continued to show thickened hyperplastic arterioles, prominent periportal vascular channels, periportal fibrosis and atrophic hepatocytes. Intrahepatic bile ducts remained patent, there were no variations in glycogen content, and a progressive increase of hemosiderin deposition occurred in the Kupffer cells over the 90-day observation period. Neither gross nor microscopic alterations were observed in 3 sham-operated dogs similarly studied for 90 days. These observations indicate that arteriovenous and arteriosinusoidal shunting is a factor leading to increased hepatic artery flow following complete portal diversion.

7333 **EFFECT OF AN END-TO-SIDE PORTACAVAL SHUNT ON LIVER REGENERATION IN THE MACACA MULATTA MONKEY.** *(E.)* Donahoo, J. S. (Johns Hopkins U. Sch. Med., Baltimore, Md.), R. R. Baker, J. Sebor and G. D. Zuidema. *Amer Surg* 34(3):196-199, 1968.

Eighteen days after an estimated 40% hepatectomy in 8 monkeys, the animals were killed, the precise extent of hepatectomy was determined by a previously described method employing radiogold (*J. Surg. Res.*, in press), and the degree of liver regeneration was calculated. An average regeneration of 66% (10.7-169.3%) of the residual liver mass occurred after 39-46% hepatectomy. In an additional 7 monkeys, subjected to end-to-side portacaval shunts and then 5 days later to 29-44% hepatectomy, an average regeneration of 65% (11.5-153%) occurred at 18 days. These data suggest that the ability of the liver to regenerate following a 40% hepatectomy is not impaired by end-to-side portacaval shunt. In both shunted and control animals, there was wide variation in the extent of regeneration. The overall changes in body weight following hepatectomy appeared to be related in part to this wide variation in the degree of regeneration.

7334 **ELECTRON MICROSCOPIC STUDIES IN BILIARY ATRESIA. I. BILE DUCTULAR PROLIFERATION.** *(E.)* Hollander, M. (Mount Sinai Sch. Med., City U. New York, N. Y.) and F. Schaffner. *Amer J Dis Child* 116(1): 49-56, 1968.

Electron microscopy of 8 liver biopsy specimens obtained with the Menghini needle from 7 children with extrahepatic biliary atresia revealed prominent accumulations of electron-dense intracellular material, presumably components of bile, in the ductular cells. The largest masses appeared flocculent and homogeneous, lying free in the cytoplasm. Increased magnification revealed remnants of double membranes and vescicles within the masses. The rest of the electron-dense material was scattered within large vacuoles bounded by a single membrane. Nuclei were displaced by these granular accumulations and the mitochondria were increased in number in some ductular cells; in most they were irregularly shaped and enlarged, with the mitochondrial membranes often being indented or serrated and some cristae being curled. Polyribosomes and endoplasmic reticulum were increased, and the rough endoplasmic reticulum appeared dilated. The Golgi zone was least prominent in the ductular cells with the most electron-dense material. Pinocytotic vacuoles were prominent in the basilar portion of the cell. The basement membrane surrounding the ductules sometimes appeared frayed, at times, duplicated. Changes observed in the ductular cells of the preductules were similar to those found in the cells that formed ductules. The hepatocytes of the preductule differed only slightly (mitochondrial cristae often curled, rough endoplasmic reticulum occasionally dilated) from hepatocytes away from the ductule. It is suggested that excretion of bile by hepatocytes in biliary atresia is associated with resorption by the ductular cells. The presence of segmented leukocytes, lymphocytes, macrophages and plasma cells close to ductules supports the hypothesis that an irritating material has been reabsorbed by the ductules. Leakage of bile constitutents into the surrounding tissues may provide the growth stimulus for ductular proliferation and for the fibrogenesis which leads to cirrhosis.

7335 **ELECTRON MICROSCOPIC STUDIES IN BILIARY ATRESIA. II. HEPATOCELLULAR ALTERATIONS.** *(E.)* Hollander, M. (Mount Sinai Sch. Med., City U. New York, N. Y.) and F. Schaffner. *Amer J Dis Child* 116(1): 57-65, 1968.

Electron microscopy of liver biopsies obtained with the Menghini needle from 6 children with congenital biliary atresia (4 at 7-9 weeks of age and 2 at 12-13 months) revealed essentially normal hepatocytes early in the disease and organelle alterations associated with hepatic insufficiency at a later stage. In the 7-9 week-old patients, most hepatic parenchymal cells showed a normal amount and distribution of glycogen. Mitochondria were normal in number, size and shape, but some enlarged and bizarre forms were sometimes found (curled cristae, detached, ring-shaped cristae). Most bile canaliculi appeared normal and were formed by 2-3 hepatocytes; however, dilated ones (about 10%) were surrounded by 4, 5 or 6 cells. Endoplasmic reticulum appeared normal, with little focal cytoplasmic degeneration. The parenchymal cells of children over 1 yr old showed more glycogen depletion than the younger patients. The rough endoplasmic reticulum was moderately to greatly dilated and ribosomes were irregularly absent. Smooth endoplasmic reticulum was increased and disrupted. Polyribosomes and free ribosomes were fewer in number. Many mitochondria showed serrated membranes and curved or detached cristae. Sinusoidal microvilli were reduced and uninterrupted basement membranes below the sinusoidal epithelium in the portal and midzonal regions were more common than at the early stage of the disease. The canaliculi were formed by 2 or 3 cells exclusively. Luminal size and the number and shape of microvilli were usually normal. As in the younger patients, the bile secretory apparatus appeared normal in most cells. Cytoplasmic accumulations of bile components were not a prominent feature of hepatocyte in either age group, suggesting that parenchymal cells in biliar atresia excrete bile. Regurgitation of bile probably does not occur at the hepatocellular level, but rather at a point distal t the canaliculi, in the ductules.

7336 **ORTHOTOPIC LIVER HOMOTRANSPLANTATION: COAGULATION AND HEMATOLOGI CHANGES IN THE PIG.** *(E.)* Blecher, T. E. (Royal Infirm., Bristol, England), J. Terblanche and J. H. Peacock. *Arch Sur* 96(3):331-339, 1968.

The results of hematologic, coagulation and fibrinolysis studi on 16 pigs receiving orthotopic liver homotransplants are presented. All animals showed increased fibrinolytic activity, m imal 4 hr after the operation, which returned to normal and then to mildly depressed levels on the 2nd and 3rd postoperative days. Fibrinogen levels followed the same pattern, being reduced shortly after the phase of increased fibrinolytic activity and then rising to high normal or above normal during th following 2-3 days. Prolongation of thrombin clotting times and prothrombin times occurred during the 1st 7 hr when th animals were heparinized, with rapid return to normal; however, half of the animals showed 2nd peaks of prolonged thro bin clotting times and often of prothrombin times as well. These peaks were considered to be due partly to the presence of fibrinogen breakdown products. No significant disturbanc in thromboplastin generation were detected. Early functionin of the transplanted liver was demonstrated by the rapid retur to normal of the fibrinogen levels and prothrombin times; th latter test provided the earliest index of failing function in 2 long-term survivors prior to death. Polycythemia induced by transfusion during the operations was spontaneously correcte to preoperative values within 24 hr after surgery. Mild postoperative neutrophilia was usually noted, and thrombocytope was evident in only 3 animals. The use of antifibrinolytic th apy is not advised as a routine procedure in such experiments

7337 **STUDY OF BILE ACIDS IN THE BLOOD OF 195 PATIENTS WITH CIRRHOSIS, SEVERE JAUNDICE, HEPATITIS, AND CHOLESTATIC SYNDROME** *(Fr.)* Rautureau, M. (St. Antoine Hosp., Paris, France), B. Chevrel and J. Caroli. *Rev Medicochir Mal Foie* 42(4/5):167-236, 1967.

A total of 557 dihydrocholanic and trihydrocholanic acid det minations were made spectrophotometrically with Minibeck's reagent on blood from 195 patients with liver and biliary tra diseases. These included 50 with cirrhosis, 21 with severe jau dice, 49 with hepatitis, 54 with obstructions of the common bile duct, and 21 with primary biliary cirrhosis. Marked increases in trihydroxycholanic acid levels were consistently fou in patients with neoplastic obstructions of the common bile d and in 50% of those with cholestatic hepatitis. Moderate incre in trihydroxycholanic acid levels occurred when histological or biological cholestasis was present but was not the most outstan ing feature of the clinical picture. These were found in the earl stages of viral hepatitis, in a few cases of cirrhosis complicated b cholestasis, in most cases of primary biliary cirrhosis before the ıset of portal cirrhosis, and in atrophy of the liver. Dihydroxy- olanic acid levels were very high in severe hepatocellular in- fficiency resulting from severe jaundice. Moderately high levels ere found in all hepatocellular damage, regardless of the etiology, some patients with mild viral hepatitis and in all recurrences of epatitis, in some with biliary obstructions or apoplexy affecting e hepatic parenchyma, in all with cirrhosis complicated by in- ctions, and in many with neoplastic obstructions of the common le duct which were associated with large increases in the tri- ydroxycholanic acid level. Preferential increases in trihydroxy- olanic acid levels can be attributed to a break in the enterohe- atic circulation, so that they flow backward from the bile into e blood, or to prolonged biliary stasis which favors synthesis trihydroxycholanic acid from cholesterol by increasing the ctivity of 12α-hydroxylating enzymes. Preferential increases dihydroxycholanic acid levels may be due to deficiencies the activity of 12α-hydroxylating enzymes associated with ver failure. Intestinal infections occurring in patients with ver damage may result in selective formation of free intestinal ihydroxycholanic acid, which cannot be conjugated by the ver and passes into the blood, or in liver insufficiencies char- cterized by failure to synthesize trihydroxycholanic acids.

338 A CLINICAL STUDY ON 100 CASES OF PRIMARY CARCINOMA OF THE LIVER. *(E.)*

hoi, H. K. (Presbyterian Med. Ctr., Chonju, Korea). *Korean J tern Med* 11(2):117-123, 1968.

he 100 cases of histopathologically proven primary carcinoma the liver encountered during the period 1964-67 represented 77% of all admissions and 5.78% of all malignant neoplasms. he sex ratio of males to females was 5.7:1 and ages ranged from 5 to 72 yr, with males in the 5th decade and females in the 6th ecade showing the highest incidences. There were 85 cases of epatocellular carcinoma, 6 of cholangiocarcinoma, 2 of hepato- astoma, 1 of mixed-cell carcinoma, and 6 of poorly differentia- d carcinoma of hepatic origin. Complaints leading to diagnosis ere, in order of frequency, epigastric fullness, discomfort, in- igestion, weight loss, and anorexia. Liver carcinoma was mis- iagnosed as liver abscess, infectious hepatitis, cirrhosis or other onditions in 16%. Metastasis occurred in 11% of the cases, with ng involvement being most frequent (6 cases). Hepatomegaly nd splenomegaly were observed in 98% and 7%, resp. No spe- fic laboratory findings leading to diagnosis were observed, al- ough BSP retention was seen in 82%, and SGOT elevation in 1.4%. *Clonorchis sinensis* infestation was found in 29% of the ases, a history of hepatitis in 8%, and severe alcoholism in 24%. he average duration from onset to diagnosis was 60 days, while e average survival time after liver biopsy was 50.2 days (range om 24 hr to 223 days).

339 BILIARY COMPLICATIONS OF HYDATID CYSTS OF THE LIVER. *(Sp.)* Belgrano Statti,

I., J. A. Bouzas, E. A. Mettler and T. Barry. *Prensa Med Argent* 9(55):1383-1385, 1968.

mong 378 patients subjected to biliary tract surgery between 957 and 1967, 75 had involvement of the common bile duct nd in 19 cases this was due to hydatid cysts of the liver. A cor- ect preliminary diagnosis was made in 6 cases, while the other 3 were initially believed to have choledocholithiasis. Choledocho- omy, exploration of the biliary tree, transduodenal papillosphinc- erocholedochotomy, insertion of a Kehr tube, and treatment of the cyst were used in 12 cases. Classical bipolar treatment with dilatation of the papilla by a No. 48 Béniqué sound was used in 2 cases. In 2 more patients a transduodenal papillosphinctero-choledochotomy was performed and the cyst treated by washing and aspiration via the biliary canaliculi without a Kehr tube. Among 3 cases in which only the cystic pole was treated, the biliary pole had to be treated after 90 days in 1 case. Complications included 1 case each of hemobilia, jejunoileal obstruction in a reoperated patient, obstruction of the common bile duct despite use of a Kehr tube, and cachexia and mental imbalance in a patient with severe bilirrhagia. Surgery was successfully repeated in the last 2 cases.

7340 PROBLEMS IN THE SURGICAL TREATMENT OF PORTAL HYPERTENSION. *(Rus.)* Makhov,

N. I. (M. F. Vladimirskii Surg. Clin., Moscow, USSR), L. A. Ender, V. I. Zhigileva and O. E. Koloss. *Khirurgiia (Moskva)* 44(3):8-14, 1968.

The results of 4 surgical procedures are compared in 155 patients, including 51 (64.7% cirrhotics) who underwent omentohepatodiaphragmopexy alone, 29 (65.2% cirrhotics) who underwent omentohepatodiaphragmopexy combined with splenectomy, 50 (61.2% cirrhotics) who underwent splenectomy alone, and 25 (84% cirrhotics) who were given splenorenal anastomoses. Of the 42 patients who died, 40 had cirrhosis. Good or satisfactory results were obtained in only 14 of the 51 patients undergoing omentohepatodiaphragmopexies. A reduction in portal circulation occurred in only 1 case. There were 8 operative deaths and 10 long-term deaths. Patients with severe portal hypertension (390 mm H_2O or more) developed recurrent, and often fatal, bleeding soon after surgery. Three patients who survived episodes of bleeding were given splenorenal anastomoses with good results. Poor results with omentohepatodiaphragmopexies are attributed to the failure of anastomoses to form between blood vessels of the omentum and those of other organs. Good and satisfactory results were obtained in 16 of the 29 patients undergoing omentohepatodiaphragmopexies and splenectomies. However, there were 5 operative deaths and 4 long-term deaths. Good or satisfactory results were obtained in 27 of the 50 patients undergoing splenectomy alone. There were 8 operative deaths but only 3 long-term deaths. Good results were obtained in 21 of the 25 patients receiving splenorenal anastomoses. There were 3 operative deaths and 1 long-term death. The observation period for patients with splenorenal anastomoses was only 2.5 yr, but this is believed to be adequate for establishing long-term results in the treatment of portal hypertension.

7341 SPLENECTOMY AND SPLENORENAL ANASTOMOSIS IN THE TREATMENT OF PORTAL HYPERTENSION. *(Rus.)* Lytkin, M. I. (S. M. Kirov Acad. Milit. Med., Leningrad, USSR) and V. S. Bykov. *Khirurgiia (Moskva)* 44(3):24-31, 1968.

Of 107 patients with portal hypertension (71 cirrhotics, 36 noncirrhotics), 35 underwent splenectomy, 39 splenectomy combined with organ anastomoses, and 33 splenectomy with splenorenal shunts. There were 13 operative deaths among 85 patients treated surgically in the authors' clinic; 10 of these were cirrhotics. Those who died included 7 with splenectomies, 5 with splenectomies and organ anastomoses, and 1 with a splenectomy and splenorenal anastomosis. The cause of death was liver failure in 6 cases, bleeding varices in 3 cases, acute thrombosis of the portal and mesenteric veins in 2 cases, ascites and peritonitis in 1 case, and acute fibrinolysis in 1 case. Eighty patients were followed for 6 months to 6 yr after surgery and information was obtained from questionnaires sent to 14 others. The best results were obtained with splenectomy and splenorenal shunts, both in cirrhotics and in noncirrhotics.

7342

PRIMARY HEPATOCELLULAR CARCINOMA IN THREE MALE SIBLINGS. *(E.)* Hagstrom, R. M. (Vanderbilt U. Sch. Med., Nashville, Tenn.) and T. D. Baker. *Cancer* 22(1):142-150, 1968.

A rare case of familial primary hepatocellular carcinoma is described in 3 male siblings who died shortly after the appearance of symptoms at the ages of 11, 22 and 31 yr. Metastatic infiltration to both lung fields occurred in the youngest case and a possible metastatic lesion in the right acetabulum of the 22-yr-old was suspected 6 yr prior to the onset of the final illness. Factors known to be associated with the occurrence of hepatoma, such as cirrhosis, hepatitis and genetic influences, were not apparent in any of the brothers. There was no history of jaundice and no evidence of associated hepatic disease in any of the cases, except for a questionable area of cirrhosis of undetermined type in the biopsy sections of the oldest brother. The biopsies otherwise revealed almost identical histologic patterns in all 3 brothers; autopsies were not performed. An apparently healthy 30-yr-old sister and 26-yr-old brother (the latter underwent an extensive physical examination at the NIH Clinical Center) are surviving. No other family members had primary hepatocellular carcinoma. A detailed analysis of residential and occupational history failed to reveal definite exposure to known hepatic carcinogens. It is of interest that the patients lived in the vicinity of several large feed and grain mills and a cotton oil company from 1940-50, but no definite connection to the development of the cancers could be made.

7343

THE PATHOLOGY OF PRIMARY LIVER CARCINOMA: AUTOPSY FINDINGS IN 124 CASES. *(Ger.)* Voigt, K. G. (Inst. Path., Karl Marx U., Leipzig, Germany) and W. Helbig. *Arch Geschwulstforsch* 21(1):39-71, 1968.

Primary liver carcinoma was diagnosed in 124 patients and cirrhosis in 1166 patients among the 26,235 autopsied between 1951 and 1963 at Leipzig University. Of the patients with liver carcinoma, 78 were males and 46 females; 106 were 50-80 yr old. The tumors included 75 hepatocellular carcinomas (60.5%), 42 cholangiocellular carcinomas (33.9%), and 7 mixed forms (5.6%). Hepatocellular forms were most common among men and cholangiocellular forms among women. Cirrhosis and liver carcinoma were associated in 53 cases (42.7%). This association occurred more than twice as often with hepatocellular as with cholangiocellular forms. Lymph node metastases were found in 64.5% of these patients and organ metastases, primarily into the lungs, skeleton, serous membranes, and adrenals, in 46.8%. Jaundice and symptoms of portal hypertension occurred most often in patients with hepatocellular carcinoma and cirrhosis, while cholelithiasis and cholangiolithiasis occurred more often in patients with cholangiocellular carcinoma. Two patients had other malignancies (Hodgkin's disease, a lymphoblastoma in the process of becoming a retothel sarcoma) and 2 patients were pregnant. The tumor, its metastases, and general cachexia were established as the cause of death in 58 cases; bronchial pneumonia, in 36; bleeding esophageal varices, in 10; and pulmonary embolism, in 9.

7344

ARTERIAL OXYGEN DESATURATION IN CHRONIC HEPATIC DISEASE AND ITS EFFEC ON THE CENTRAL NERVOUS SYSTEM. *(Rum.)* Strat, C. (2nd Med. Clin., IMF, Iasi, Romania), G. Mardare, S. Iancu, V. Strat, A. Dorneanu, A. Dumitrescu and S. Jauca. *Med Intern (Bucur)* 19(11):1293-1302, 1967.

In 53 of 57 patients (32-69 yr old) with chronic liver disease (4 with chronic hepatitis, 16 with compensated cirrhosis and 37 with decompensated cirrhosis, including 11 in precoma), oxyme ric studies of blood from the femoral artery, using a photoelectric method, revealed an abnormally low oxygen saturation (average 88% compared to 95% in 10 controls). The average pO_2 was 70.8 mm Hg compared to 95-100 mm Hg in controls, and the arteriovenous difference was decreased by an average o 20.2% in 38 cases. Respiratory function was essentially normal in all 16 patients with compensated cirrhosis and in 17 of 28 with decompensated cirrhosis in which respiratory function test were done, even though half of these patients showed significan decreases in O_2 saturation. In 10 cases with hepatic cirrhosis an portal hypertension, splenoportography and splenomanometry demonstrated that the drop in O_2 saturation was the combined result of portamediastinal shunts (6 cases) and pulmonary hypo ventilation produced by the presence of ascites. There was no direct correlation between blood O_2 levels and the neuropsychia tric or electroencephalographic findings. However, cerebral hypoxia, together with hypokalemia, is known to increase the tox city of ammonia for the nerve cell.

7345

LOSS OF BASOPHILIC GRANULATION IN EXPERIMENTAL HEPATITIS. *(Rum.)* Roman, E. (Dept. Physiopath., IMF, Bucharest, Romania) and T. Negru. *Stud Cercet Fiziol* 13(3):261-269, 1968.

The Shelley test (degranulation of rabbit basophils in contact with liver homogenates from previously sensitized guinea pigs; *Nature* 195:1181, 1962) was given to 57 adult guinea pigs (14 males and 43 females, average weight 326 g) in which experime tal hepatitis had been produced 6-92 days earlier by an autoimmune technique (injection of rabbit liver mitochondria or mitochondrial supernatants in complete Freund's adjuvant) Each guinea pig received 2 injections at an 8-day interval and then a 3rd injection after 21 days, at mitochondrial protein concentrations of 40, 60 and 100 mg/ml, resp. The Shelley test was clear positive in 64.9% of the animals, with no significant difference between those receiving intact mitochondria and those given mitochondrial supernatant; doubtful results were obtained in 4 of 29 and 5 of 28 cases, resp. There was no significant sex difference, since 64.3% of all males were positive. No positive results were obtained in 10 controls inoculated only with Freud's adjuvant or 13 given no injections at all, although doubt ful results were obtained in 2 of the 1st group. The highest proportion of positive tests (reaching 100% in some groups) was attained in guinea pigs sacrificed 17-25 and 89-92 days after the 3rd injection. These results tend to support the autoimmune hypothesis of the etiology of chronic hepatic disease.

7346

EFFECTS OF DISEASES OF THE GALLBLADDER AND BILIARY TRACT ON THE LIVER. *(Sl.)* Bucko, A. (Res. Inst. Human Nutrit., Bratislava, Czechoslovakia) and J. Babala. *Cesk Gastroent Vyz* 22(1):1-5, 1968.

Liver biopsy specimens obtained during surgery from 72 patients (14 men and 58 women, 22-76 yr old) with a 1-40 yr history (average 6 yr) of lithiasis of the gallbladder (53 cases) or common bile duct (19 cases) revealed pathological changes in 29% and 50%, resp. These included moderate steatosis in 5.8-7.7% and 12.5%, resp., moderate-severe cholestasis in 1.9-5.8% and 25-37.5%, moderate-severe bile duct dilatation in 9.6% and 37.5%, bile duct proliferation in 7.7-26.9% and 50%, moderate regressive parenchymal changes in 9.6% and 37.5%, moderate-severe inflammatory mesenchymal infiltration in 13.45-19.2% and 12.5-37.5%, moderate-severe mesenchymal fibrosis in 11.5-13.45% and 25-37.5%, and hyperplasia of the Kupffer cells in 36.5% and 12.5%, resp. The average duration of illness in the 19 patients with choledocholithiasis was 16 yr (1-30). Early surgery for cholelithiasis is recommended in order to avoid permanent liver damage.

7347 **REGIONAL INFUSION THERAPY FOR SOME LIVER DISEASES IN CHILDREN.** *(Rus.)* Akopian, V. G. (Cent. Inst. Postgrad. Med., Moscow, USSR), V. V. Gavriushov, T. D. Kuznetsova, M. N. Krutikov and V. A. Lavrov. *Khirurgiia (Moskva)* 44(7):82-88, 1968.

A technique was developed for infusing drugs into the portal vein by introducing a catheter into the umbilical vein in such a way that bleeding from the vein does not occur after completion of the infusion. Between each infusion, 1500-2000 units of heparin in 2-3 ml of 0.25% procaine are injected into the catheter to prevent formation of thrombi. The catheter is then sealed by heating and clamped shut. Promising results were obtained by using this technique to infuse antibiotics into 25 children, aged 2 months to 15 yr, with various suppurative diseases of the liver. To reduce hepatic hypoxia, which was shown to be present in children with cirrhosis and portal hypertension, this technique was used for infusing 150-450 ml of oxygenated blood into 5 children with liver failure and hepatic coma. Two recovered, but 3 others died of massive gastrointestinal bleeding after regaining consciousness. Portal hypertension decreased periodically in 2 of these children, apparently due to reflex closing of arteriovenous anastomoses in the portal system because of increased oxygenation of liver tissue.

7348 **PERIARTERIAL HEPATIC NEURECTOMY IN CHRONIC AND RECURRENT JAUNDICE.** *(Fr.)* Marinaccio, G. (Inst. Surg. Symptomatol., U. Bari, Italy) and G. M. Bonomo. *Lyon Chir* 63(6):825-838, 1967.

Eighteen patients (12 women and 6 men, aged 17-70 yr) with chronic or recurrent jaundice underwent dissection and resection of nerve plexi along the hepatic artery and its main branches between the celiac plexus and the hilum. This procedure was combined with splenectomy in a patient with hemolytic splenomegaly, the creation of a portacaval shunt in a patient with cirrhosis, cholecystostomies in 2 patients, a cholecystectomy in 1, ligature of the splenic artery in 1, and removal of adhesions in 2. The hepatic artery was ligated in an unspecified number of cases. Liver biopsies, made in 14 cases, showed that cholestatic hepatitis was present in 5 cases, biliary stasis in 2, hepatic necrosis in 4, acute hepatitis in 2, cirrhosis in 1, steatosis in 1, and hepatic fibrosis in 1. Long-term follow-ups of 13 patients showed that the results of surgery were good or satisfactory in 11 cases and poor in 1 patient with cholestatic hepatitis and progressive sclerosis. One patient with cirrhogenous liver disease died in a hepatic coma 3 months after surgery and 2 had recurrences of jaundice.

7349 **DIFFERENTIAL DIAGNOSIS OF CLINICAL JAUNDICE IN KOREA (A STATISTICAL STUDY OF 223 CASES).** *(Kor.)* Choi, H. J. (Wallace Mem. Baptist Hosp., Pusan, Korea) and C. B. Kim. *Korean J Intern Med* 10(12):795-801, 1967.

A statistical analysis of the causes of jaundice (serum bilirubin of 2.0 mg% or higher) was made in 223 patients (155 males and 68 females, most 30-60 yr old) seen over a 43-month period in Pusan, Korea, representing 1% of the 22,718 patients seen during that period. Hepatitis accounted for 30%, hepatoma 11.5%, cirrhosis 28%, and biliary tract disease 21.5% of all cases. Among patients below 30 yr old (excluding congenital biliary anomalies), 90% had hepatitis, while among those below 40, 75% had either hepatitis or cirrhosis. Above the age of 40, 50% had surgical types of jaundice, including hepatoma, biliary tract inflammation or biliary tract malignancy. No patient under 30 yr of age had jaundice due to a malignancy. Congenital biliary tract anomalies were present in 3 males and 6 females (congenital biliary atresia in 8 and congenital choledochal cyst in 1). Parasites were incriminated in 6 cases (ascariasis in the common bile duct in 1, and clonorchiasis causing portal cirrhosis in 5). The total bilirubin range was 2-5 mg% in 50% of all cases, with the highest value of 33.75 mg% being seen in a case of cholangioma.

7350 **THE COAGULATION DEFECT AFTER EXTENSIVE LIVER RESECTION IN MAN.** *(E.)* Almersjö, O. (Sahlgren Hosp., Göteborg, Sweden), S. Bergmark, L. Engevik, K. Korsan-Bengsten and J. Ygge. *Scand J Gastroent* 2(3):204-208, 1967.

The acitivity of clotting factors I, V, II, VII, and X, platelet counts, and plasminogen levels were studied in the postoperative period of 4 patients subjected to 40-80% liver resection; a case of major surgery (gastrectomy) selected from a study of 22 cases in which the same factors were studied was used for comparison. The plasminogen concentration in 3 of the hepatectomy cases was reduced to the same extent as in the gastrectomy case 2 days postoperatively, and reached preoperative levels by the 5th-10th postoperative day, but the maximal peak was reached later in the hepatectomy patients than in the gastrectomy case. The drop in platelet count in the first few postoperative days as well as the increase above the initial values later in the postoperative course was the same for both the liver resection and gastrectomy cases. In the hepatectomy cases, there was a marked decrease in fibrinogen and factors V, II, VII, and X. In the cases subjected to 40% resection, the clotting factors were normalized after 3-10 days; in those in whom 60% and 80% of the liver was resected, factors II, VII, and X reached the normal level after several months. This was in contrast to the gastrectomy case, in which an increase in fibrinogen and no decrease in factors II, V, VII, and X were noted. The most likely cause for the decreased clotting factor activity following partial hepatectomy is a decreased synthesis of these factors.

7351 **NON-SUPPURATIVE AMOEBIC HEPATITIS - A CLINICAL ENTITY AND ITS RESPONSE TO METRONIDAZOLE THERAPY.** *(E.)* Banerji, R. N. (Safdarjang Hosp., New Delhi, India), N. Lakshmipathy, A. K. Nag, S. K. Khanna and V. Behari. *Indian Pract* 21(10):749-757, 1968.

The clinical features and results of treatment of 36 patients with nonsuppurative amebic hepatitis are presented. In all cases the onset was insidious and afebrile; mild to moderate hepatic enlargement, mucus in stools with periodic diarrhea, and normocytic anemia were present. Predominantly epigastric or upper right quadrant pain, elevated erythrocyte sedimentation rates, increased serum transaminase levels and abnormal excretion values for ^{131}I-Rose Bengal occurred in 83.3%, 83.3%, 91.3% and 83.0%, resp. Significantly, leukocytosis and radiological evidence of a raised diaphragm were absent in all cases. Serum protein studies showed no hypoproteinemia, but mild hypergammaglobulinemia, mild hypoalbuminemia and a reduction in the α_1-globulins with a mild rise in the α_2-globulins were observed in many patients. Such alterations differ from the serum protein pattern found in liver abscess or portal cirrhosis. None of 12 patients with nonamebic hepatic disorders responded to 20 mg/kg body weight metronidazole for 20 days, but 10 of 11 and 21 of 21 patients with amebic hepatitis treated with the same dose of metronidazole for 20 or 10 days, resp. were clinically improved or cured. A 60% failure rate was noted when the drug was given for only 5 days. Regression of the hypergammaglobulinemia and the elevation in serum albumin levels was closely correlated with the clinical response to therapy. The course of clinical improvement (subjective symptoms almost gone by 10th day of treatment, hepatic tenderness almost absent by 15th day, significant regression in liver size in from 15-30 days was much slower than that observed for liver abscess. It is concluded that the nonsuppurative clinical condition described in this study differs from amebic liver abscess.

7352 **EXPERIMENTAL EFFECTS OF THE LIGATION OF THE EFFERENT HEPATIC LYMPHATICS.** *(Fr.)* Mandairon, Y. (Ed. Herriot Hosp., Lyon, France) and G. Yoshitomi. pp. 37-41 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7353 **PORTAL DECOMPRESSION BY MEANS OF SPLENOHEPATIC PARENCHYMAL ANASTOMOSIS IN THE RABBIT.** *(It.)* Delbue, V. (Inst. Surg. Path., U. Padua, Italy), D. Oselladore, F. Sartori and I. Fantin. *Gazz Int Med Chir* 72(24-bis):3037-3042, 1967.

7354 **BEHAVIOR OF THE IMMUNOGLOBULINS AND OF THE RHEUMATOID FACTOR AGGLUTINATION TEST IN THE CHRONIC LIVER DISEASES AND HEPATIC CIRRHOSIS.** *(It.)* Liotta, S. (Inst. Spec. Med. Path., U. Rome, Italy), A. de Laurenzi, L. Fontana, G. Papa and G. Isacchi. *Gazz Int Med Chir* 72(24-bis): 2999-3012, 1967.

7355 **EXTRACORPOREAL PERFUSION OF AN ISOLATED AUXILIARY LIVER. PART I. THE HEPATECTOMIZED ANIMAL. PART II. CHANGES PRODUCED BY PARENTERAL THERAPY IN A HEPATECTOMIZED ANIMAL.** *(It.)* Romeo, S. (Inst. Surg. Symptomatol., U. Catania, Italy), G. Gruttadauria, G. Vagnoni and A. Bordi. *Gazz Int Med Chir* 72(24-bis):3289-3314, 1967.

7356 **EXPERIMENTAL ANALYSIS OF THE CONDITIONS FOR BILIOLYMPHATIC REFLUX.** *(Fr.)* Michoulier, J. (Ed. Herriot Hosp., Lyon, France). pp. 26-32 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7357 **HEPATIC LYMPH FLOW DURING BILIARY OBSTRUCTION.** *(Fr.)* Ritchie, H. D. (London Hosp., England). pp. 22-25 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7358 **THE LYMPHATICS OF THE LIVER DURING THE FIRST FEW HOURS OF CHOLEDOCHAL OBSTRUCTION.** *(Fr.)* Ahualli, D. (Ed. Herriot Hosp., Lyon France), J. Michoulier and R. Lodi. pp. 17-21 in: Circulatio Lymphatique et Arterielle en Pathologie Digestive Abdominal [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7359 **A PHYSIOPATHOLOGICAL EVALUATION O THE VARIOUS TECHNIQUES FOR SURGER ON THE EFFERENT LYMPHATICS OF THE LIVER.** *(Fr.)* Mallet-Guy, P. (Ed. Herriot Hosp., Lyon, France), Y. Mandairon, G. Yoshitomi and J. Foray. pp. 72-80 in: Circul tion Lymphatique et Arterielle en Pathologie Digestive Abdom nale [Lymphatic and arterial circulation in abdominal digestiv pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7360 **DRAINAGE OF THE THORACIC DUCT IN LIVER DISEASE.** *(Fr.)* Dumont, A. E. pp. 61-63 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominalc [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7361 **ENTERIC OXYGEN THERAPY IN THE TREAT MENT OF CHRONIC DISEASES OF THE LIVE AND BILIARY TRACT.** *(Rus.)* Nezhdanova, O. V. (Sanat. No. 1, Morshin, USSR) and M. A. Vashchenko. *Vrach Delo* (1):120-121, 1968.

7362 **THE COMPLEX TREATMENT OF PATIENTS WITH CHRONIC HEPATOCHOLECYSTITIS AT THE "POLIANA" HEALTH RESORT.** *(Rus.)* Stakhiv, I. V. (Poliana Sanat., Trans-Carpath. Distr., USSR). *Vrach Delo* (1):121-122, 1968.

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7364 **JAUNDICE AND PREGNANCY.** *(Fr.)* Laroche, C. (Fac. Med., Paris, France), R. Caquet and J. Grégoire. *Entret Bichat [Med]* pp. 381-388, 1967.

7365 **CHRONIC HEPATITIS.** *(Fr.)* Schwarzmann, V. (St. Antoine Hosp., Paris, France). *Entret Bichat [Med]* pp. 327-332, 1967.

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7367 **A CLINICAL AND BIOLOGICAL STUDY OF THE EFFECT OF A NEW DRUG ON JAUNDICE.** *(Fr.)* Ribet, A. (Purpan Hosp., Toulouse, France) and M. Hardy. *Entret Bichat [Ther]* pp. 121-123, 1967.

7368 **AGNOGENIC MYELOID METAPLASIA WITH PORTAL HYPERTENSION.** *(E.)* Colker, J. L. (Cornell U. Med. Coll., Ithaca, N. Y.), W. G. Baker and M. Kolodny. *Gastroint Endosc* 15(2):70-75, 1968.

7369 **SERUM ENZYME ACTIVITIES IN NEONATAL JAUNDICE OF FULL-TERM AND PREMATURE INFANTS.** *(It.)* Pallucchini, F. (Inst. Pediat. Clin., U. Pavia, Italy), A. Marchi and L. Polito. *Pediatria (Napoli)* 76(4):557-570, 1968.

7370 **THE CLASSIFICATION OF CHRONIC HEPATITIS WITH SPECIAL REFERENCE TO ACTIVE OR AGGRESSIVE CHRONIC HEPATITIS.** *(It.)* Gentilini, P. (Inst. Spec. Med. Path., U. Florence, Italy). *Recent Progr Med (Roma)* 45(3):201-229, 1968.

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7372 **INCIDENCE OF PRIMARY CARCINOMA OF THE LIVER IN THE WEST OF SCOTLAND BETWEEN 1949 AND 1965.** *(E.)* Manderson, W. G. (Royal Infirm., Glasgow, Scotland), R. S. Patrick and E. E. Peters. *Gut* 9(4):480-484, 1968.

7373 **ACUTE FATTY LIVER OF PREGNANCY.** *(E.)* Joske, R. A. (Dept. Med., U. Western Australia), D. J. McCully and F. L. Mastaglia. *Gut* 9(4):489-493, 1968.

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7377 **CLINICAL AND HISTOLOGICAL OBSERVATIONS IN EIGHT CASES OF IDIOPATHIC JAUNDICE OF PREGNANCY SUBJECTED TO BIOPSY OF THE LIVER.** *(It.)* Sposito, M. (S. Giacomo Hosp., Rome, Italy), G. Hassan, L. Cancrini and G. Marinucci. *Arch Ital Mal Appar Dig* 34(3):315-327, 1967.

7378 **CASE REPORT OF A PRIMARY NEUROGENIC TUMOR OF THE LIVER (GANCLIONEUROMA).** *(It.)* Guernelli, N. (Inst. Surg. Symptomatol., U. Bologna, Italy) and B. Tonelli. *Arch Ital Mal Appar Dig* 34(2):175-192, 1967.

7379 **METABOLIC FACTORS AFFECTING THE DURATION OF ELECTRICAL SYSTOLE IN LIVER DISEASES.** *(Cz.)* Válek, J. (Inst. Res. Human Nutrit., Prague, Czechoslovakia), V. Brodan, E. Kuhn and L. Blahniková. *Cesk Gastroent Vyz* 21(7):462-468, 1967.

7380 **EFFECT OF 4-METHYL-2,6-DI-(t-BUTYL)-PHENOL (IONOL) ON HEPATOMA INDUCTION IN RATS.** *(Rus.)* Frankfurt, O. S. (Inst. Chem. Phys., Acad. Sci. USSR, Moscow), L. P. Lipchina, T. V. Bunto and N. M. Emmanuel'. *Biull Eksp Biol Med* 64(8):86-88, 1967.

7381 **INTRAHEPATIC CHOLESTASIS IN A CASE OF PRIMARY HYPERPARATHYROIDISM.** *(Cz.)* Petera, V. (Med. Fac., Charles U., Plzen, Czechoslovakia) and V. Krauz. *Cesk Gastroent Vyz* 21(8):514-516, 1967.

7382 **LONG-ACTING PYRIDINE-3-CARBOXYLIC ACID HYDROXYMETHYLAMIDE IN THE TREATMENT OF HEPATOBILIARY CHOLESTASIS AND CHOLANGITIS.** *(Sp.)* Cerullo, E. (Ramos Mejia Hosp., Buenos Aires, Argentina), J. Satriano and E. F. Valdes. *Prensa Med Argent* 55(22):1052-1055, 1968.

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7384 **TECHNICAL AND SURGICAL REQUIREMENTS FOR AN AUXILIARY LIVER TRANSPLANT IN THE RAT.** *(It.)* Serio, G. (Inst. Surg. Symptomatol., U. Padua, Italy) and P. L. Sussi. *Chir Ital* 19(5):614-631, 1967.

7385 **ACUTE CHOLESTASIS INDUCED BY ALPHA-NAPHTHYLISOTHIOCYANATE AND LIGATION OF THE COMMON BILE DUCT IN THE RAT.** *(Sp.)* Garay, E. R. (Inst. Gastroent., NIH, Haedo, Argentina), A. Piccaluga, V. Perez and M. Royer. *Rev Esp Enferm Apar Dig* 26(9):1079-1085, 1967.

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7388 **EXPERIMENTAL STUDIES ON THE LYMPHATIC CIRCULATION OF THE LIVER. XII. SURGICAL TECHNIQUE FOR DRAINAGE OF THE CISTERN OF PECQUET.** *(Fr.)* Mallet-Guy, P. (Surg. Clin. A, Fac. Med., Lyon, France), Y. Mandairon and J. Murat. *Lyon Chir* 63(5): 667-673, 1967.

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7414 ASCARIASIS OF THE LIVER AND BILIARY TRACT. *(Por.)* Ogawa, K. M. (Darcy Vargas Child. Hosp., São Paulo, Brazil) and A. L. Mathias. *Matern Infanc (S Paulo)* 26(4):381-386, 1967.

7415 ANTIGLOMERULAR AND ANTI-SMOOTH-MUSCLE-FIBER ANTIBODIES IN HEPATIC DISEASE. *(Sp.)* Velasco, M. (Salvador Hosp., Santiago, Chile). *Rev Med Chile* 96(4):217-220, 1968.

7416 SERUM TRYPSIN ACTIVITY IN PATIENTS WITH CHRONIC HEPATITIS AND CIRRHOSIS AS AN INDICATION OF THE EXOCRINE ACTIVITY OF THE PANCREAS. *(Rus.)* Lukanev, G. D. (Chernovtsy Med. Inst., USSR). *Vrach Delo* (10):30-33, 1968.

7417 A CAUSE OF OBSTRUCTIVE JAUNDICE WHICH IS DIFFICULT TO DIAGNOSE: "ALVEOLAR ECHINOCOCCOSIS OF THE LIVER". *(Fr.)* Demirleau, J., J. Caroli and M. Noureddine. *Mem Acad Chir (Paris)* 93(29): 887-894, 1967.

See also: 6781,6787,6788,6837,6839,6840,6842,6845,6853,6855,6857,6861,6864,6876, 6885,6893,6894,6900,6901,6908,6914,6917,6923,6928,6930,6931,6932,6937, 6938,6939,6940,6947,6957,6959,6960,6962,6965,6970,7021,7134,7137,7312, 7318

7418 SERIAL STUDIES OF THE LIVER IN WOMEN TAKING PROGESTATIONAL AGENTS. *(Sp.)*

Reyes, H. (Salvador Hosp., U. Chile, Santiago), S. Donoso, M. Velasco and R. Katz. *Rev Med Chile* 96(6):399-404, 1968.

Fifteen women were followed for 8-18 months and 6 for 3-8 months after they began taking oral contraceptives. An estrogen-progestagen combination was given to all but 1 patient who received a sequential formula. Blood bilirubin and SGPT values were determined before therapy and in the last week of each cycle and liver biopsies were made in 5 cases. The patients ranged in age from 22-39 yr and had had 1-11 pregnancies. Findings were normal in 12 patients, 4 of whom had histories of idiopathic pruritus of pregnancy and 3 of whom had histories of idiopathic jaundice of pregnancy. Of the 9 others, 2 developed jaundice and intense generalized pruritus in the first and third cycles, resp.; liver function tests became abnormal, SGPT values rose, and biopsies showed evidence of severe liver damage and cholestasis. Both of these women had a history of idiopathic jaundice of pregnancy. Signs of subclinical hepatitis developed in 7 other women, 3 of whom had histories of idiopathic jaundice of pregnancy, between the 4th and 9th cycles of gestagen therapy. Laboratory findings subsequently returned to normal although administration of gestagens was not discontinued. Only 1 of these patients was given a different gestagen. Liver biopsies, made in 2 cases, showed lesions identical to those found in severe viral hepatitis. The etiology of these cases of subclinical hepatitis is in doubt since none of the laboratory tests available can differentiate between damage produced by contraceptives and that caused by viral hepatitis. It is recommended that women with histories of idiopathic jaundice of pregnancy not be given oral contraceptives.

7419 EFFECT OF INTERNAL IRRADIATION WITH ^{32}P ON THE DNA CONTENT AND MITOTIC INDEX OF REGENERATING RAT LIVER. *(Rum.)*

Turcu, G. (Biophys. Lab., IMF, Bucharest, Romania) and C. Dimoftache. *Stud Cercet Fiziol* 13(3):255-259, 1968.

In 9-12-month-old male Wistar rats given 2 μC/g of $Na_2H^{32}PO_4$ 24 hr before partial hepatectomy (67%), the DNA content and mitotic index of the liver 24-48 hr after hepatectomy were found to be significantly decreased compared to nonirradiated controls. At 24, 28 and 48 hr, the mitotic index was 7.6, 40.7 and 11.19, resp. in controls and 5.4, 20.8 and 8.5 in irradiated rats. The DNA content of the liver was 978.51 and 723.96 mg/100 g dry weight, resp. 24 and 48 hr after hepatectomy in controls and 858.44 and 704.14 mg/100 g dry weight in irradiated rats, compared to a value of 844.18 mg/100 g dry weight before hepatectomy. Maximal depression of DNA synthesis and mitosis were thus produced at 24 and 28 hr, resp. It is urged that only small doses of radioactive isotopes be used in biochemical studies in order to avoid harmful effects on physiological functions.

7420 ALCOHOLIC FATTY LIVER IN MAN ON A HIGH PROTEIN AND LOW FAT DIET. *(E.)*

Lieber, C. S. (Bellevue Hosp., New York, N. Y.) and E. Rubin. *Amer J Med* 44(2):200-206, 1968.

The chemical, histologic, and ultrastructural changes observed in liver biopsy specimens from 5 alcoholic volunteers (4 men and 1 woman, 35-58 yr old) fed a high-protein, low-fat diet are presented. All subjects had abstained from alcohol for 2-5 months prior to the study. During a control period, the diet consisted of 2100-2600 cal/day (25% protein, 25% fat, 50% carbohydrate). Alcohol (95% ethanol in 15% aqueous solution), divided into 6 equal daily portions was then given in increasing amounts (isocaloric exchange for carbohydrate): 24% of total calories for 4 days, 36% for 2 days, and 46% for 12 days. Liver biopsies were made at the end of both the control period and the alcohol ingestion period. In all subjec hepatic levels of total lipids and triglycerides at the end of th alcohol period were 3-15 times higher than in the correspondi controls. In 2 of 3 specimens taken at the end of the alcoho period, the amount of histologically visible fat was increased, while no change was noted in 1 as compared to the control specimens. In 2 subjects who did not have an intermediate liver biopsy, the specimen at the end of the alcohol period revealed evident steatosis. During the alcohol period, electron microscopy revealed a decrease in rough endoplasmic reticulu with hypertrophy of the smooth endoplasmic reticulum and enlarged mitochondria with irregular and disoriented cristae. It is concluded that alcohol, even in the presence of a high-protein, low-fat diet, is toxic to the liver.

7421 FINE STRUCTURAL CHANGES IN RAT LIVE CELLS IN RESPONSE TO PROLONGED THIOACETAMIDE. *(E.)*

Kendrev, G. (Med. U., Budapest, Hungar *Path Europ* 3(1):96-109, 1968.

The changes occurring in the hepatocellular fine structure of male albino rats receiving daily i.p. injections of thioacetamid (TAA; 50 mg/kg) were studied by electron microscopy 2-101 days after initiation of treatment. At day 2, the cisternae of the rough endoplasmic reticulum (rER) were moderately dilat and showed loss or ribosomes, glycogen spherules were decreased in number, and some mitochondria contained no crist and contained many electron-dense bodies. At day 8, the nuclei varied in size, cytoplasmic indentation of the nuclear men brane was frequent, increased numbers of microbodies were evident, and glycogen was absent. At day 15, the nuclei and nucleoli were enlarged with some nuclei containing several nucleoli; an increased number of mitochondria was also noted At day 31, alterations were no longer limited to the center of the lobules; the parenchyma showed signs of incipient destruction with even larger nuclei and nucleoli present. There was less rER and many of the membranes showed degranulation; the smooth endoplasmic reticulum (sER) was extensive, and glycogen spherules reappeared. At day 43, normal appearing rER was again apparent and at day 52, a few cells appeared almost normal, although others still contained an increased amount of sER. Extensive Golgi areas now appeared. At day 63, the sER was made up of vesicles and tubules and was increased in amount; the rER consisted of agranular cysts with electron-dense substances in the cisternae. At day 101, an epithelial-type cell 1st appeared, smaller than the hepatocytes, containing large nuclei of low electron density with 1 or mor nucleoli. Few mitochondria were present. In the course of TAA-hepatocarcinogenesis, the fine structural changes are mos evident in the organelles participating in the nucleic acid meta lism of the cell, in association with other metabolic disturban

422 **LOCAL FREEZING OF LIVER TISSUE AND TRANSAMINASE ACTIVITIES.** *(Ger.)* Stucke, . (Surg. Clin., U. Würzburg, Germany) and C. Ciroth. *Acta lepatosplen (Stuttgart)* 14(6):326-333, 1967.

aparotomies were performed on 17 adult rabbits of both exes who had been anesthetized with 2.5% thiopental. Their vers were cooled locally to -60 to -100C with a catheter througl hich liquid N_2 was passed so that frozen areas were produced anging from 7-31 mm in diameter. Local freezing of the liver ncreased serum transaminase activities significantly. SGOT alues reached a maximum immediately after surgery while GPT values did so only 15.4 hr later. This postoperative inrease in transaminase values was proportional to the extent f liver damage produced by freezing. SGOT and SGPT values eturned to normal 70.3 and 82.8 hr after surgery, resp. When ver tissue was not frozen no changes occurred in transaminase evels during laparotomies on normal animals, although postperative increases did occur in animals which were anesthetized vith ether.

423 **THE ROLE OF HEPATIC HYPOXIA IN THE DEVELOPMENT OF TOXIC HEPATITIS.** *(Rus.)* hanin, M. N. (Tashkent Med. Inst., USSR). *Eksp Khir Anest* 3(4):52-55, 1968.

dministration of sublethal doses of heliotrine (40 mg/100 g) o rats is followed in 12-24 hr by acute granulation of the epatic cytoplasm and in 36-48 hr by central necrosis of the epatic lobules, while the peripheral zones remain normal. his central necrosis is also typical of infectious or arsphenanine-induced hepatitis, and has been ascribed to hypoxia reulting from disturbances in the hepatic circulation. In order o clarify the role of hypoxia in the development of hepatic ecrosis, the gas composition of the arterial blood and blood rom the femoral, portal and hepatic veins was studied after dministration of heliotrine to dogs. The O_2 content of the emoral venous blood increased from 12-14 vol.%, the arteriovenous difference decreased from 5.2-3.2 vol.%, and the coeficient of O_2 utilization decreased from 29.9-18.5 vol.% in 24-8 hr; even more pronounced and longer-lasting changes were een in the hepatic venous blood, with a decrease from 3.1-.7 vol.% in the amount of O_2 retained by the liver, even hough central hepatic necrosis does not develop in dogs. In ats, the O_2 consumption was decreased by 45% in 24 hr and y 55% in 72 hr, and in animals with chronic hepatitis and leveloping cirrhosis the O_2 consumption remained at 70% of ormal. It is concluded that hepatic hypoxia plays a central ole in the development of toxic hepatitis and cirrhosis, and hat species differences in the sensitivity of the liver to hepatotoxic agents can be explained by differences in hepatic circulation. In view of the central role of hypoxia, therapy in such cases should be directed at reducing the toxic effects on oxidative processes and improving hepatic blood supply. In this connection, a surgical technique for arterialization of the liver has been developed in dogs, in which a portacaval shunt is followed by anastomosis of the splenic artery to the hepatic branch of the portal vein. This double arterial blood supply helps to prevent the common complications of portal hypertension surgery.

424 **EFFECT OF INTRAGASTRIC ADMINISTRATION OF OXYGEN ON SOME INDICES OF NITROGEN METABOLISM DURING ACUTE EXPERIMENTAL TOXIC HEPATITIS.** *(Rus.)* Uspenskii, V. I. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Biull Eksp Biol Med* 64(8):51-53, 1967.

7425 **HEPATITIS FROM METHIMAZOLE DURING ADRENAL STEROID THERAPY FOR MALIGNANT EXOPHTHALMOS.** *(E.)* Becker, C. E. (Natl. Inst. Arthr. Metab. Dis., NIH, Bethesda, Md.), P. Gordon and J. Robbins. *JAMA* 206(8):1787-1789, 1968.

7426 **CHOLESTATIC JAUNDICE DURING IMIPRAMINE THERAPY.** *(E.)* Short, M. H. (Midway Hosp., St. Paul, Minn.), J. M. Burns and M. E. Harris. *JAMA* 206(8):1791-1792, 1968.

7427 **LIVER DYSFUNCTION WITH MESTRANOL BUT NOT WITH NORETHYNODREL IN A PATIENT WITH ENOVID-INDUCED JAUNDICE.** *(E.)* Urban, E. (Colorado Gen. Hosp., Denver), B. W. Frank and F. Kern Jr. *Ann Intern Med* 68(3):598-602, 1968.

7428 **EFFECTS OF THE HEPATOTOXIC AGENTS RETRORSINE AND AFLATOXIN B_1 ON HEPATIC PROTEIN SYNTHESIS IN THE RAT.** *(E.)* Trevino, S. V. (Med. Res. Counc., Surrey, England) and D. D. Leaver. *Biochem J* 109(1):87-91, 1968.

7429 **EFFECTS OF CORTICOSTEROIDS ON LIVER INJURY FOLLOWING HEPATIC ARTERY LIGATION.** *(E.)* Prakash, A. (All-India Inst. Med. Sci., New Delhi, India) and M. C. Tanga. *Indian J Surg* 30(2):97-104, 1968.

7430 **NITROSAMINE-INDUCED GLYCOGENOSIS AND TUMOR FORMATION IN RAT LIVER: A COMPARATIVE LIGHT- AND ELECTRON-MICROSCOPIC STUDY.** *(Ger.)* Bannasch, P. (Inst. Path., U. Wurzburg, Germany). *Verh Deutsch Ges Path* 51:343-350, 1967.

7431 **THE SPECTRUM OF HEPATOTOXICITY.** *(E.)* Zimmerman, H. H. (Boston VA Hosp., Mass.). *Perspect Biol Med* 12(1):135-161, 1968.

7432 **THE ULTRASTRUCTURE OF THE RAT LIVER FOLLOWING CARBON TETRACHLORIDE POISONING.** *(It.)* Melis, M. (Inst. Anat. Path. Histol., U. Rome, Italy) and L. Orci. *Fegato* 13(2):231-252, 1967.

7433 **EXPERIMENTAL LIVER DAMAGE PRODUCED BY THIOACETAMIDE.** *(Sp.)* Irizarry, S. (Div. Clin. Appl., Nucl. Ctr., San Juan, Puerto Rico), F. Landrón-Becerra and A. Rodríguez-Olleros. *Bol Asoc Med P Rico* 60(5): 222-229, 1968.

7434 **OCCUPATIONAL LIVER DAMAGE DUE TO ALCOHOL.** *(Ger.)* Schuttmann, C. (Cent. Inst. Occup. Med., Berlin-Lichtenberg, Germany), W. Schuttmann and L. H. Kettler. *Z Ges Hyg* 14(5):337-344, 1968.

7435 **EXTENSIVE RUPTURE OF THE RIGHT LOBE OF THE LIVER WITH SIMULTANEOUS INJURIES TO OTHER ORGANS.** *(Pol.)* Wasowski, J. (3rd Surg. Clin., Acad. Med., Gdansk, Poland), Z. Gruca and J. Kossak. *Pol Przegl Chir* 40(6):689-691, 1968.

7436 **ULTRASTRUCTURAL DEVELOPMENT OF RADIATION INJURY IN HEPATIC PARENCHYMAL CELLS OF GAMMA-IRRADIATED MICE.** *(E.)* Hendee, W. R. (U. Colorado Med. Ctr., Denver) and M. A. Alders. *Lab Invest* 18(2):151-158, 1968.

7437 **NATURE OF THE BINDING OF CARCINOGENIC AMINOAZO DYES WITH LIVER PROTEINS. X. FURTHER EVIDENCE ON THE NATURE OF BINDING BETWEEN AMINOAZO DYES AND LIVER PROTEINS, WITH SPECIAL REFERENCE TO THE SITE OF BINDING.** *(E.)* Matsumoto, M. (Dept. Biophys., U. Tokyo, Japan), H. Takata and H. Terayama. *Gann* 59(3):231-238, 1968.

7438 **STRUCTURE AND IMPORTANCE OF INTRANUCLEAR INCLUSIONS IN THIOACETAMIDE HEPATOCARCINOGENESIS.** *(E.)* Kendrey, G. (U. Med. Sci. Budapest, Hungary). *Acta Morph Acad Sci Hung* 16(1):53-63, 1968.

7439 **HYPERTRIGLYCERIDEMIA DURING TREATMENT WITH ESTROGEN AND ORAL CONTRACEPTIVES: AN ALTERATION IN HEPATIC FUNCTION?** *(E.)* Gershberg, H. (New York U. Sch. Med., N. Y.), M. Hulse and Z. Javier. *Obstet Gynec* 31(2):186-189, 1968.

7440 **CHANGES OF THE SERUM PEPTIDASE ACTIVITY IN EXPERIMENTAL LIVER DAMAGE.** *(E.)* Horký, J. (Inst. Human Nutrit., Prague, Czechoslovakia), Z. Placer and V. Hromádková. *Acta Hepatosplen* 15(2) 97-101, 1968.

7441 **HEPATIC CARCINOGENESIS IN CBA/Cb/Se MICE AND Cb/Se RATS BY ISONICOTINIC ACID HYDRAZIDE AND HYDRAZINE SULFATE.** *(E.)* Severi, L. (Div. Cancer Res., U. Perugia, Italy) and C. Biancifi *J Nat Cancer Inst* 41(2):331-340, 1968.

See also: 6784,6792,6797,6822,6823,6884

42 **THE BEHAVIOR OF LYMPHOID CELLS IN INFECTIOUS HEPATITIS.** *(Ger.)* Jorke, D. unic. Hosp., Jena, Germany) and I. Hennig. *Z Ges Inn Med* (23):746-749, 1967.

e total number of lymphoid cells were counted and lympho- ms were recorded for 10 hospitalized patients (5 adults and children) with viral hepatitis; for 178 children from 3 kinder- tens which had more than 2 children sick with jaundice at e same time; for 80 children from kindergartens which had reported cases of viral hepatitis for the past 3 yr; and for hepatitis contacts. At the onset of jaundice the number of nphoid cells increased in all patients. This increase occurred marily in small and large cells with light cytoplasm. A par- el was established between the lymphoid cell count and um bilirubin values and the clinical course. Elevated lym- oid cell counts occurred significantly more often among chil- en from kindergartens with hepatitis and among hepatitis ntacts than among controls. Among 13 contacts with high nphoid cell counts, 6 of the 11 examined developed elevated nsaminase levels and 5 remained normal. Of these 6 hepa- is patients, 5 did not have jaundice. Although an increase the lymphoid cell count is not a specific test for viral hepa- is, it is a valuable way to detect patients with anicteric and ortive forms of hepatitis.

43 **RHESUS ERYTHROCYTE AGGLUTINATION IN HEPATITIS.** *(E.)* O'Connell, C. J. (Sisters Charity Hosp. Buffalo, N. Y.). *Amer J Med Sci* 256(1): -43, 1968.

glutinins for rhesus monkey red blood cells (1:10 titer or eater) were found in 15 of 20 children (75%) and 7 of 11 ults (64%) with acute viral hepatitis. This incidence is sig- ficantly greater than that found in normal persons. Little fference was observed between the pediatric and adult cases either the frequency of positive tests or the average titers tained. Significant changes in titer were seen in 2 of 3 sub- cts from whom multiple specimens were available during the urse of the disease. Following the acute phase of the disease -14 months) only 1 of 23 adult patients showed a titer greater an 1:8; this frequency of positive tests is less than that ex- cted for a normal population. A high frequency of positive sts was also found in infectious mononucleosis (10 of 15 ses) and in alcoholic liver damage (9 of 13 cases), but not in umps (4 of 8) or measles (4 of 10), suggesting that the de- lopment of rhesus agglutinins is not a nonspecific effect of ral infection. Results of tests using rhesus RBC treated with *cholerae* receptor-destroying enzyme (titers were 2-4 times gher than with untreated RBC) indicate that the antigen does ot contain sialic acid. A significant drop in titer occurred hen acute sera giving high titers were absorbed with dried beef BC antigen (2-32 fold drop) or guinea pig kidney antigen (4- fold drop), implying that the agglutinin is not directed against ther the Forssman antigen or the classic infectious mononu- eosis antigen. Absorption studies showed that the agglutinins e specifically directed against sheep RBC with cross-reactivity rhesus RBC.

444 **CLINICAL, ENZYMOLOGICAL, AND MORPHOLOGICAL PARALLELS IN VIRAL HEPATITIS N CHILDREN.** *(Rus.)* Shakhgil'dian, I. V. (Munic. Clin. Hosp. Infect. Dis. No. 82, Moscow, USSR), S. N. Kutchak, Ie. A. Paktoris and D. M. Braginskii. *Pediatriia* 47(1):12-18, 1968.

Studies were made of 76 children, aged 1-14 yr, with viral hepatitis. These included 8 with subclinical, 32 with anicteric, and 36 with mild, manifest hepatitis. None of the children with subclinical or anicteric hepatitis had elevated serum bilirubin or urinary bile pigment values. However, even in these patients, pronounced increases in serum alanine aminotransferase, aspartate aminotransferase, fructose-1,6-diphosphate aldolase, alkaline phosphatase, fructose-1-phosphate aldolase, and sorbitol dehydrogenase activities occurred early in the disease. Material obtained from punch liver biopsies consistently showed proliferative and infiltrative changes along the portal system and other blood vessels in the liver and a proliferative reaction of Kupffer's cells. Changes in interstitial liver tissue were found in 13 patients with anicteric hepatitis, 16 with mild hepatitis, and 2 with subclinical hepatitis who had been biopsied during the first 10 days after a diagnosis of hepatitis had been established. Changes in the hepatic parenchyma were slight and temporary and did not develop at all in some cases. A correlation was observed between increases in alanine aminotransferase and fructose-1-phosphate aldolase activities and dystrophic changes in the hepatic parenchyma in patients with anicteric hepatitis. Morphological changes in the liver are an excellent basis on which to establish a retrospective diagnosis of viral hepatitis since they occur more consistently than clinical and laboratory changes and often persist until the patient's discharge.

7445 **ELECTRON MICROSCOPE OBSERVATIONS OF SOME FORMS OF VIRAL HEPATITIS. CONTRIBUTION TO THE UNDERSTANDING OF THE MORPHOLOGICAL EVOLUTION AND PATHOGENETIC MECHANISM OF JAUNDICE.** *(E.)* Teodori, U. (Inst. Spec. Med. Path., U. Florence, Italy), P. Gentilini and C. Surrenti. *Gastroenterologia* 108(3):105-120, 1967.

Liver tissue taken by needle-biopsy from 15 patients with classic acute hepatitis (10) or cholestatic-cholangiolitic hepatitis (5) at various intervals (4-60 days) after the onset of jaundice were examined under the electron microscope. Similar changes were noted in both groups, but these were more marked in cases of classic acute hepatitis. Maximal changes in the hepatocytes, such as disruption of the cell membrane and degeneration of the microvilli, were noted at the vascular pole as early as day 4; rupture of cell membranes resulted in dilation of the Disse spaces. The endothelial membrane in some cases acquired a vascular aspect between day 8 and 13; Kupffer cells, particularly in classic cases, often showed excessive bile pigment early in the disease, which decreased progressively after day 20-25. After day 11, lysosomes appeared in increasing numbers. Electron-opaque particles, often forming rosette-shaped aggregates of 600-1000 Å and staining with lead hydroxide and uranyl acetate, were found consistently in the cytoplasm. At the biliary pole, the Golgi apparatus underwent changes and disappeared early in the disease. Only in the cholestatic form, however, were there considerable stores of pigment with alterations and dilation of the bile canaliculi. It is concluded that in classic acute hepatitis, jaundice is caused by inhibition of pigment secretion at the biliary pole, while in cholestatic-cholangiolitic hepatitis the jaundice is due to excretory dysfunction, resulting in excessive pigment accumulation at the biliary pole and in the canaliculi.

7446 DISTURBANCES OF DIGESTION AND ABSORPTION IN VIRUS HEPATITIS. *(E.)* Fikry, M. E. (Fac. Med., Alexandria, Egypt) and M. H. Aboul-Wafa. *Gastroenterologia* 108(3):135-146, 1967.

The function of the stomach, pancreas and small intestine was studied in 66 patients during and up to 6 months after brief episodes of viral hepatitis. The stomach, studied by means of the alcohol fractional test meal (27 patients), histamine-induced secretion test (20), and the hypoglycemia test (20), showed a decrease in maximal acid secretion, an increased fasting secretory volume low in hydrochloric acid, a reduced vagal response and reduced pepsin secretion. The pancreas, studied by means of the secretin test (19) and by serum amylase measurements (19), showed a reduction in the volume and enzyme activity of secretions, which did not improve in 5 of 12 patients studied after complete recovery. With regard to the small intestine, the xylose absorption-excretion test (20) showed inhibition of intestinal carbohydrate absorption; urinary xylose was only slightly below normal, however. These findings support clinical observations of gastrointestinal symptoms in association with viral hepatitis.

7447 CLINICAL AND EPIDEMIOLOGICAL PARALLELS IN VIRAL HEPATITIS DURING PREGNANCY AND LACTATION. *(Rus.)* Romanov, Iu. A. (Botkin Clin. Hosp. Infect. Dis., Leningrad, USSR). *Zh Mikrobiol* 45(6):109-113, 1968.

An analysis was made of 553 women who contracted viral hepatitis during pregnancy and 233 who contracted it during lactation. The incidence of viral hepatitis among women who were more than 3 months pregnant was 34.7% higher than that among all women of child-bearing age. Severe forms of viral hepatitis occurred twice as often in the second half of pregnancy as in the first and slightly more often among multiparae. Of these 553 women, 51 delivered at term and 54 prematurely; 123 had artificial abortions and 11 had spontaneous abortions. Among 105 women who had viral hepatitis during pregnancy, 6 had stillborn infants and 7 had infants who died *in utero*. Follow-ups of the children, made for 5-10 yr, showed that large numbers developed abnormally and had neurological symptoms which were sometimes associated with mental retardation and diffuse minor symptoms. In contrast to previous reports in the literature, severe forms of hepatitis occurred in 23.2% of the lactating women and in only 3.8% of the pregnant women. Hepatitis developed in the former within 3 months after delivery in 139 of 233 cases and in 99 of the 137 women who had undergone surgery or were given blood transfusions or injections. This suggests that the incidence of serum hepatitis is high among lactating women. Because of the strain it places on the liver, breast feeding is contraindicated when the mother contracts viral hepatitis.

7448 CHANGES IN THE ACTIVITY OF INTESTINAL ENZYMES IN CHILDREN WITH VIRAL HEPATITIS. *(Rus.)* Kasimovskaia, N. A. (Bashkir Med. Inst., Ufa, USSR). *Pediatriia* 47(10):74-76, 1968.

Enterokinase and alkaline phosphatase activities in the stools and enterokinase activity in the intestinal juice were determined in 55 patients, aged 3-7 yr, with viral hepatitis and in a group of normal controls. During the acute stage of the infection, duodenal enterokinase activity was reduced in 28 of the 55 patients and had not increased to normal levels by the time the children were clinically recovered. These differences were statistically significant and are associated with a decrease in the production of this enzyme by the duodenal mucosa an with a reduction in bile flow into the intestine. During the same period, fecal enterokinase and alkaline phosphatase value increased significantly in all patients and did not return to no mal after recovery in many cases. These changes are probably caused by chemical processes occurring in the large, rather tha the small, intestine. These changes in intestinal enzyme activi emphasize the need for patients to return for follow-up and t observe a strict diet after their discharge.

7449 THE SIGNIFICANCE OF PERICHOLEDOCHAL LYMPHADENITIS IN THE ORIGIN OF THE OBSTRUCTIVE COMPONENT OF JAUNDICE IN VIRAL HEPATITIS. *(Rus.)* Nikiforov, V. N. (Cent. Clin. Hosp., Moscow, USSR) and T. V. Gogoleva. *Sovet Med* 31(6):26-31 1968.

Radiological examinations of 70 patients with confirmed viral hepatitis showed that 20 had pericholedochal lymphadenitis. Of these 20 patients, 3 had mild, 8 had moderately severe, an 9 had severe hepatitis. In addition, 1 had prolonged edema a ascites, 1 had chronic recurrent hepatitis with cirrhosis in the early stages, and 8 had biliary tract and pancreatic diseases. All 20 had jaundice, hepatomegaly, and high aldolase, SGOT, and SGPT levels which returned to normal later than usual. Two patients developed acute pancreatitis, 1 an exacerbation chronic pancreatitis, 1 a bleeding duodenal ulcer, and 1 suspected hepatic dystrophy. Staphylococci were isolated from 3 of 8 bile samples and enterococci were isolated from 2; 3 cultures remained sterile. Pericholedochal lymphadenitis developed 8-24 days after the onset of symptoms and lasted unt the jaundice and clinical symptoms disappeared. The commo bile duct was dilated in 8 cases. Of these 20 patients, 19 recovered and 1 died. It is concluded that pericholedochal lym adenitis is relatively common in viral hepatitis and that it is o of the causes of obstructive jaundice, causing compression of the extrahepatic biliary tract. This might account for the development of recurrent and cholestatic forms of viral hepatitis

7450 IMMUNOLOGICAL STUDIES ON CHRONIC POSTVIRAL HEPATITIS. *(Fr.)* Buligesco, L. (Fundeni Hosp., Bucharest, Romania). *Acta Gastroent Belg* 31(8/9):533-542, 1968.

Clinical and immunological studies were made of 542 patients with chronic viral hepatitis (188 with cirrhosis and 354 with chronic progressive hepatitis). In a small number of cases live disease was associated with allergies and autoimmune diseases such as bronchial asthma, hemolytic anemia, hemorrhagic recto colitis, collagen diseases, and Hashimoto's thyroiditis. Splenomegaly was present in 23% and anemia, ranging from mild to severe, in 50%. Elevated serum bilirubin levels in 9.5% of tho with chronic hepatitis and 27% of those with cirrhosis suggest that hemolysis is a factor involved in the production of anemi

Low white-blood counts with large numbers of lymphocytes and monocytes were found in 10%, and platelet counts were reduced in 69.7%. LE tests were negative and myelograms were normal in all cases. Immunoelectrophoresis showed that 28.7% of the patients with chronic hepatitis and 92% of those with cirrhosis had increases of more than 25% in plasma γ-globulins. Characteristic changes in cirrhotic patients included ncreases in γ and β_2M globulins, the presence of a β_2C arc, and the absence of a haptoglobin arc. Liver biopsies, performed n 300 cases, showed lymphopolyblastic infiltration in Kiernan's spaces and the perilobular region in 20%. Complement-fixation tests with liver antigen prepared by Pasnick's method were positive at high dilutions (1:128 and 1:512) in only 2 of 22 cases in which they were performed. These results suggest that an immunological mechanism is involved in about 20% of the patients studied. It is considered to be a secondary mechanism, eading to the development of chronic forms of viral hepatitis and the perpetuation of hepatic lesions.

7451 DURATION OF THE COURSE OF CHRONIC POSTHEPATITIC LIVER DISEASE. *(Ger.)*

Seifert, E. (Hanusch Hosp., Vienna, Austria) and H. Dittrich. *Acta Hepatosplen (Stuttgart)* 14(6):341-348, 1967.

Of 500 patients with chronic liver disease, 381 had confirmed cirrhosis and 119 confirmed chronic hepatitis. Studies of the etiology showed that 24.7% of those with cirrhosis had histories of hepatitis, 30.4% were alcoholics, 13.9% had diabetes mellitus alone or in combination with other histological abnormalities, and 4.2% were alcoholics who had diabetes. Miscellaneous disorders (biliary cirrhosis, hemochromatosis, and hepatic porphyria) and unknown causes accounted for the remaining 26.8%. Thymol turbidity, zinc sulfate, and BSP tests and serum protein fractions were abnormal in about 80% of those with posthepatitic cirrhosis; the diagnostic values of these tests was about the same. Weltmann's coagulation band was changed in only a few cases. The percentage of abnormal laboratory findings was much smaller among patients with chronic hepatitis, and serum protein fractions were of the greatest diagnostic value. No abnormalities were found on palpation or in liver function tests in 3 of the 94 with posthepatitic cirrhosis and in 10 of the 119 with chronic hepatitis. Esophageal varices occurred significantly more often in posthepatitic cirrhosis, but the incidence of ascites was the same in all forms with the exception of diabetic cirrhosis. Mortality rates were about the same in all forms of cirrhosis with the exception of diabetic cirrhosis. The period between diagnosis and death appeared to be longer in posthepatitic cirrhosis than in other forms, but in many cases it was difficult to establish the actual time at which cirrhosis developed. In some cases chronic hepatitis or posthepatitic cirrhosis was only diagnosed 10-20 yr after the onset of viral hepatitis.

7452 EPIDEMIOLOGIC BEHAVIOR OF ACUTE PRIMARY LIVER DISEASE IN THE PROVINCE OF MODENA IN THE PERIOD 1961-66. *(It.)*

Renzulli, L. (Inst. Hyg., U. Modena, Italy) and E. Preite. *Fracastoro* 61(1):73-82, 1968.

An epidemiologic survey revealed 495 officially reported cases of acute primary liver disease in the province of Modena during he period 1961-66. No mortality occurred in this series, conirming the mildness and the short duration of the illnesses. The frequency of this form of hepatitis increased in the autumn-winter months and in June. Except for the 14-21 yr-old age group, in which males were more often affected (26.4 cases per 100,000) than females (15.0 per 100,000), no statistically significant sex-related differences in morbidity were found. Children up to 6 yr old and between 6 and 14 yr old showed high morbidity (46.8 and 60.9 cases per 100,000, resp.); this may be related to the higher incidence of anicteric forms in young children and to increased contacts among school children. Persons living in apartment-type dwellings showed an average morbidity of 20.4 cases per 100,000 compared to 12.5 for those living in homes widely separated from each other. In addition, significant statistical differences were observed in the relative morbidity of families with 4-5 members (18.2 cases per 100,000) and those with fewer (9.9 cases per 100,000) or more (12.1 cases per 100,000) family components. This increased incidence may be due to the greater representation of young children in the families with 4 or 5 members. Because of inadequacies in the disease-report forms used, it was not possible to evaluate the role of crowded living conditions or of oro-fecal contagion in the incidence of these diseases. Certain changes are proposed in the report forms so that further studies may be conducted into the socioenvironmental factors in acute primary liver diseases.

7453 CLINICAL ASPECTS OF VIRAL HEPATITIS ASSOCIATED WITH GASTROINTESTINAL DISORDERS. *(It.)*

Scalise, G. (Inst. Gen. Med. Clin., U. Siena, Italy), M. Gentili, C. A. Boggiano and A. Fedi. *Settimana Med* 55(17):1051-1063, 1967.

Clinical and histological findings and the results of liver function tests are analyzed for 134 patients with acute viral hepatitis and 22 patients with the chronic form. These findings were completely characteristic of viral hepatitis in 14 patients with gastroduodenitis, 10 with gastric atonia and ptosis, 2 with duodenal diverticulosis, 4 with stomach resections, and 2 with gastric carcinomas. An abnormal course of viral hepatitis was often observed, however, in patients with preexisting peptic ulcers or associated cholelithiasis. Thus, routine radiological examination showed that 8 patients with acute and 3 with chronic hepatitis also had peptic ulcers (10 duodenal, 1 gastric), but only 3 of these patients had histories of peptic ulcers. Needle biopsies of the liver showed the presence of granulocytic cholangiolitis in 6 of the 8 patients with acute hepatitis and 1 of the 3 with chronic hepatitis. Periductal thickening was associated with signs of epithelial desquamation and regneration in the bile ducts. No signs of cholestasis were found. These patients had persistent jaundice and abnormal liver function tests for up to 2 months. Viral hepatitis was also diagnosed in 8 patients who definitely had cholelithiasis and in 14 who probably had chronic calcareous gallbladder disease before the onset of hepatitis. These patients also had protracted courses of hepatitis. Blood bilirubin and SGOT and SGPT levels remained elevated even after clinical recovery but no abnormalities were found in the electrophoretic behavior of serum proteins, prothrombin times, or flocculation and turbidity tests. Histological findings included marked signs of intrahepatic cholestasis and cholestatic cholangitis.

7454 CLINICAL AND HISTOLOGICAL FEATURES OF ADVANCED, PROTRACTED, AND CHRONIC VIRAL HEPATITIS. *(It.)*

Verme, G. (Inst. Gen. Med. Clin.,

U. Siena, Italy), P. Gioannini, C. A. Boggiano and G. Scalise. *Epatologie* 13(3):485-496, 1967.

Needle biopsies were taken from 162 patients with acute viral hepatitis during various stages of the infection. Typical histological features of acute viral hepatitis were found in 78% of 48 patients biopsied 10-20 days after the onset of symptoms, in 41% of 44 biopsied after 20-30 days, and in 26% of 13 biopsied after more than 30 days. No appreciable differences were found in the percentage of subjective disturbances (asthenia, anorexia, nausea, hypochondrial pain, pruritus) in these 3 groups. Jaundice was present in 94.1% of the patients in the 1st group, 82.3% in the 2nd, and 41.1% in the 3rd. The regression of jaundice generally coincided with the appearance of pigment in Kupffer's cells. Hepatomegaly was found in 50% of those in the 1st group, 64.7% in the 2nd, and 84% in the 3rd; 64.7% of those examined 20-30 days after the onset of symptoms had fever. No differences were found between the erythrocyte sedimentation rates and serum alkaline phosphatase levels of these 3 groups of patients. Of the 34 patients with subacute (protracted or intermittent) hepatitis, 65% showed scattered necrosis with a histological picture of acute hepatitis, 31% showed few signs of regression, parenchymal infiltration or necrosis but persistent or accentuated portal infiltration, and 4% showed persistent or accentuated intralobular cholestasis with formation of intracanalicular biliary calculi. Jaundice was present in 86.6%, hepatomegaly in 80%, and fever in 60%. Liver function studies helped to establish a diagnosis of subacute hepatitis in many of these patients. Only 3 patients developed chronic hepatitis. These included 1 with a perforated duodenal ulcer and chronic suppurating peritonitis, 1 with cholelithiasis, and 1 who died several months later of acute hemorrhagic enterocolitis. Histological findings included persistence of portal and intralobular inflammation, regressive polymorphic lesions similar to those seen in the acute stage, and regeneration with the development of circumscribed adenomatous nodules.

7455 **REGENERATION OF THE LIVER IN VIRAL HEPATITIS.** *(It.)* Sposito, M. (St. Giacomo Hosp., Rome, Italy), G. Hassan, G. Marinucci, L. Valeri and V. A. Petroni. *Epatologia* 13(3):531-544, 1967.

Acid phosphatase and ATPase were determined by cytochemical methods, and *in vitro* incorporation of ^{3}H into thymidine and methionine was studied by autoradiography in material obtained by needle biopsy from 120 patients during various stages of viral hepatitis. During the acute stage, alkaline phosphatase, which is localized to the lysozymes, was found throughout the necrotic cells. During recovery, the lysozymes returned to their normal pericanalicular location; this always preceded the regression of jaundice. During the acute stage of the infection the cell membrane of the bile capillaries, which normally contains ATPase, did not stain. During recovery, the bile capillaries appeared contorted and had irregular dimensions similar to those seen during liver regeneration in the rat. In extremely severe cases of hepatitis, postnecrotic regeneration of the bile capillaries was irregular due to their dislocation and compression by regenerating cell nodules. Bile pigments accumulated in lysozymes of the hepatocytes. In the 12 specimens examined, only 1 nucleus was labeled with ^{3}H-thymidine. *In vitro* incorporation of ^{3}H-methionine occurred mainly in cells of prolifera ing ductules and only rarely in the hepatocytes. This is attribu to the greater permeability of the membranes of ductule cells to methionine.

7456 **A STUDY OF THE SERUM ACID-SOLUBLE DESOXYRIBOSIDES IN THE ACUTE PHASE OF VIRAL HEPATITIS.** *(It.)* Lomanto, B. (Inst. Gen. Med. Clin., U. Milan, Italy), L. Natale and A. Agostoni. *Arch Ital Mal Appar Dig* 34(1):5-9, 1967.

7457 **HEPATITIS AS IT RELATES TO THE ARCTIC.** *(E.)* McCollum, R. W. (Dept. Epidem. Publ. Health, Yale U. Sch. Med., New Haven, Conn.). *Arch Environ Health (Chicago)* 17(4):529-536, 1968.

7458 **NEUROLOGICAL DISORDERS IN VIRAL HEPATITIS.** *(Rus.)* Gol'del'man, M. G. (Zaporozhe Inst. Postgrad. Med., USSR) and P. G. Gaft. *Vrac Delo* (12):59-62, 1967.

7459 **LACTIC DEHYDROGENASE ISOENZYMES IN THE SERUM OF CHILDREN WITH INFECTIOUS HEPATITIS.** *(Rus.)* Iurkov, Iu. A. (Pediat. Inst., Acad. Med. Sci. USSR, Moscow), V. V. Alatyrstev and M. Kh. Ismailova. *Sovet Med* 30(10):42-45, 1967.

7460 **BONE MARROW DAMAGE IN VIRAL HEPATITIS.** *(Ger.)* Spatz, R. (2nd Med. Clin., U. Munich, Germany), H. G. Borchers and H. Zobl. *Med Klin* 63(44):1761-1765, 1968.

7461 **STUDIES ON THE EPIDEMIOLOGIC EFFECTI NESS OF OBLIGATORY HOSPITALIZATION OF VIRAL HEPATITIS PATIENTS.** *(E.)* Szmuness, W. (Prov. Sanitary-Epidemiol. Station, Lublin, Poland). *Epidemio Rev* 21(3/4):233-236, 1967.

7462 **APLASIA OF THE BONE MARROW AFTER VIRAL HEPATITIS.** *(E.)* Judkiewicz, L. (1st Clin. Child. Dis., Med. Acad., Lodz, Poland) and H. Radzikows Orlowska. *Epidemiol Rev* 21(3/4):205-208, 1967.

7463 **EPIZOOTIC VIRAL HEPATOSIS IN A NEW STRAIN OF MICE (NZBr/Bl).** *(E.)* Bielschowsk M. (Dept. Cancer Res., U. Otago, New Zealand), T. Maguire an C. M. Goodall. *Proc U Otago Med Sch* 46(3):56-58, 1968.

See also: 6852,6854,6865,6867,6868,6869,7278

7464 **HEPATIC CIRRHOSIS IN THE TROPICS—A HAEMATOLOGICAL STUDY.** *(E.)* Gupta, J. P. (Coll. Med. Sci., Benares Hindu U., Uttar Pradesh, India), H. S. Bajpai, Y. N. Gupta and I. M. Gupta. *J Trop Med Hyg* 71(10):264-266, 1968.

The various aspects of anemia in cirrhosis and its correlation with other clinical manifestations were investigated in 45 patients (38 males and 7 females, 18-55 yr old). Jaundice was observed in 12 patients and ascites in 40; there were 15 patients with mild anemia (10-12 g hemoglobin), 20 with moderate anemia (6-10 g hemoglobin), and 6 with severe anemia (less than 6 g hemoglobin). The high incidence of macrocytic anemia (39.9%) was associated with multiple nutritional deficiencies. Normocytic anemia was present in 42.2% of the cases. Iron deficiency was attributed to chronic blood loss from esophageal varices or hemorrhoids. Some degree of splenomegaly was observed in 33 patients, and there was a positive correlation between the degree of splenomegaly and the degree of anemia. Splenomegaly also correlated well with the degree of leukopenia. The data support the concept that hypersplenism contributes to the degree of anemia in liver cirrhosis.

7465 **THORACIC DUCTO-JUGULAR VEIN ANASTOMOSIS IN THE MANAGEMENT OF PORTAL HYPERTENSION.** *(E.)* Bhalerao, R. A. (K.E.M. Hosp., Bombay, India), S. R. Panday, D. R. Trivedi, M. G. Nerurkar and P. K. Sen. *J Postgrad Med* 14(4):177-186, 1968.

A simple, new technique for end-to-end thoracic duct-internal jugular vein anastomosis was used in 42 patients with portal hypertension, including 34 cirrhotics, 5 with portal cavernoma and 3 with primary portal hypertension. Of the 42, 11 had major variceal hemorrhage at the time of surgery, 18 had a previous history of variceal bleeding and 13 had gross varices on splenoportography; 36 patients had ascites at the time of surgery and 1 had a past history of ascites. Fifteen patients were frankly jaundiced at the time of surgery and an additional 12 had a past history of jaundice. The massive variceal hemorrhage stopped in 7 patients, but 2 had recurrent fatal variceal hemorrhage within 10 days of surgery; in the remaining 4 patients the intensity of bleeding was reduced. Three additional patients had minor variceal hemorrhage at an interval of 6 weeks, 6 months and 1 yr, but none of them died due to hemorrhage. Ascites disappeared in most of the patients. Splenic size and pressure fluctuated for a few days, with a tendency to decrease, while liver function tended to improve in a majority of patients. Postoperative lymphorrhea and sepsis, with resultant thrombosis of the shunt, occurred in 4 cases, with fatal hemorrhage in 2.

7466 **CEREBRO-HEPATO-RENAL SYNDROME. A NEWLY RECOGNIZED HEREDITARY DISORDER OF MULTIPLE CONGENITAL DEFECTS, INCLUDING SUDANOPHILIC LEUKODYSTROPHY, CIRRHOSIS OF THE LIVER, AND POLYCYSTIC KIDNEYS.** *(E.)* Passarge, E. (Good Samaritan Hosp., Cincinnati, Ohio) and A. J. McAdams. *J Pediat* 71(5):691-702, 1967.

A newly recognized hereditary disorder is described in 5 sisters with similar clinical and pathological features. The main features were: 1) severe, generalized hypotonia and absent Moro response; 2) craniofacial abnormalities; 3) renal cortical cysts; and 4) hepatomegaly. All 5 siblings failed to thrive, 2 had jaundice, 3 had gastrointestinal bleeding, and all 5 died in infancy. The parents, who were not known to be related had 8 other normal children (5 boys and 3 girls). Congenital heart disease, minor skeletal abnormalities, and abnormal brains showing sudanophilic leukodystrophy were also found in the 5 sisters. In 1 girl examined at autopsy, the liver weighed 208 g and was yellow and hard. Several small infarcts within the parenchyma were noted, but no bile stasis or gross biliary duct dilatation were seen. Histologic examination showed diffuse micronodular cirrhosis; there was no inflammatory infiltration, although small clusters of lymphocytes were seen. Bile stasis was present in scattered foci, and mild pseudoductular proliferation was evident. A few superficial stress ulcers in the duodenum were also noted. The descriptive term "cerebro-hepato-renal syndrome" is proposed for this previously unrecognized clinical and genetic entity.

7467 **SERUM CERULOPLASMIN IN LIVER CIRRHOSIS.** *(E.)* Ventura, E. (Dept. Clin. Med., U. Modena, Italy), M. L. Zeneroli and S. Calandra. *Gastroenterologia* 107(6):360-367, 1967.

Serum ceruloplasmin levels were determined in 26 patients with alcoholic cirrhosis, 2 with hemochromatosis, 4 with biliary cirrhosis, and 40 normal subjects. The patients included 23 males and 9 females. The diagnoses were established by clinical and laboratory findings and confirmed by laparoscopy and needle liver biopsy. Considering the normal range to be 22.3-40.3 mg/100 ml, there were 25 patients (20 with alcoholic cirrhosis, 4 with biliary cirrhosis and 1 with hemochromatosis) who had abnormally high serum ceruloplasmin levels; 20 of these patients including all 4 with biliary cirrhosis, showed serum bilirubin levels above 1 mg/100 ml, while the remaining 5 patients had normal serum bilirubins. Six patients (5 with alcoholic cirrhosis and 1 with hemochromatosis) had a normal serum ceruloplasmin with serum bilirubin levels above 1 mg/100 ml, and one patient had normal serum ceruloplasmin and bilirubin levels. The 4 patients with biliary cirrhosis showed the highest values for serum ceruloplasmin. There was no significant correlation between serum ceruloplasmin and billirubin levels. In all patients, fractional analysis of the serum proteins revealed a sharp inversion of the albumin/globulin ratio. Electrophoretic partition detected a significant increase in α_2-globulins when compared with the normal mean value found in the laboratory. There was a significant correlation between serum ceruloplasmin and α_2-globulin levels in all 3 diseases. These findings are tentatively ascribed to impairment of both estrogen metabolism and biliary secretion, accompanied by increased globulin synthesis.

7468 **THE SURGICAL TREATMENT OF PORTAL HYPERTENSION.** *(Rus.)* Samofalov, V. P. (Volgograd Med. Inst., USSR). *Khirurgiia (Moskva)* 44(3):14-19, 1968.

Among 142 patients with portal hypertension, 125 (88%) had cirrhosis, 13 had thrombosis or stenosis in large branches of the portal and hepatic veins, 2 had liver cancer, 1 had stomach cancer, and 1 had stones in the common bile duct. Surgery was performed on 100 cases (60 males and 40 females). Operations performed included 18 vascular anastomoses (2 portacaval, 1 mesenterocaval, and 15 splenorenal) and 68 organ anastomoses (60 omentorenopexies with splenectomies, 3 omentorenopexies alone, 2 omentohepatopexies, and 2 omentohepatopexies combined with other procedures). Miscellaneous procedures included ligation of blood vessels along the smaller curvature of the stomach, ligation of the splenic artery, Kalb's operation, and gastric and intestinal resections. Causes of the 17 operative deaths were renal insufficiency (7 cases), gastrointestinal bleeding (4 cases), shock (3 cases), and thrombosis (3 cases). The postoperative course was uneventful in 60 cases and complicated in 40. Best results were obtained on patients in the early stages of portal hypertension and the worst results in those with esophageal varices and ascites (15 deaths out of 62). Follow-up studies done 1-11 yr after surgery on 57 patients showed that 42 had improved, 6 had stabilized, 3 had deteriorated, and 6 had died. Results were good for all 5 patients with extrahepatic blocks and for 18 of 20 with compensated forms of portal hypertension regardless of the surgical procedure employed. Some good results were obtained by organopexies on patients with esophageal varices and ascites, but 5 had recurrent bleeding which was fatal in 2 cases. Of 7 patients with vascular anastomoses 4 had mild recurrent bleeding.

7469 **GASTRIC ACID SECRETION WITH MAXIMAL AUGMENTED HISTAMINE STIMULATION IN INDIAN CHILDHOOD CIRRHOSIS.** *(E.)* Chandra, R. K. (All-India Inst. Med. Sci., New Delhi). *J Trop Med Hyg* 71(6): 150-152, 1968.

Maximal histamine-stimulated gastric secretion (0.04 mg/kg body weight) was studied in 23 fasting infants (10-22 months) with Indian childhood cirrhosis and in 10 control subjects. Promethazine (0.5 mg/kg body weight) was administered i.m. 30 min before the injection of histamine. The basal acid output did not differ in the 2 groups (0.64 mEq/hr in controls, 0.56 mEq/hr in patients), but the peak secretion was significantly reduced in cirrhotic subjects (1.04 mEq/30 min) compared to the controls (3.75 mEq/30 min). The peak response was also significantly delayed in cirrhotics, occurring in the 3rd 15-min collection period compared to the 2nd 15-min period for the controls. In addition, the peak response waned more rapidly in the patient group. It is suggested that active hepatocellular necrosis during childhood cirrhosis may lead to the release of histaminase which may modify the gastric response to exogenous histamine.

7470 **PROBLEMS IN THE TREATMENT OF ACUTE BLEEDING FROM ESOPHAGEAL VARICES PRODUCED BY HEPATIC CIRRHOSIS.** *(Ger.)* Matzander, U. (Surg. Clin., U. Saar., Homburg, Germany). *Thoraxchirurgie* 16(4):338-347, 1968.

In order to evaluate the suitability of general hypothermia to protect the liver against hypoxia caused by acute hemorrhage, experiments were performed on 4 groups of 15 rabbits, 30 of which had been given 0.5 ml CCl_4/kg s.c. 48 hr before. In 2 groups (one with liver damage and one without), the rectal temperature was then reduced to 30C by giving a mixture of 20 mg/kg chlorpromazine, 20 mg/kg isopromethazine, and 40 mg/kg pethidine and placing the animal in ice water, after which 20 ml of blood was withdrawn through a cannula in the femoral artery every 10 min from all 60 rabbits. Although metabolic acidosis was less evident in hypothermia, both intact and liver-damaged rabbits survived longer when their body temperatures were normal. It is concluded that cooling is not an effective means of treating acute blood loss. Since another problem in the treatment of esophageal varices is the reduction in hepatic blood flow following portacaval shunt, a surgical technique was developed on laboratory animals to improve arterialization in the portal circulation. After creation of an end-to-side portacaval anastomosis, the portal system is arterialized with the ileocolic artery by a venous transplant from the large saphenous vein. This technique was successfully used on a patient with cirrhosis, portal hypertension, and bleeding esophageal varices.

7471 **THE USE OF SOLUBLE 6-METHYLPREDNISOLONE COMBINED WITH FUROSEMID IN HEPATIC CIRRHOSIS.** *(It.)* Ferrucci, A. (United Hosp., Livorno, Italy), G. Silvi, F. Ghiara, M. G. Franzò and G. P. Zucchelli. *Clin Ter* 44(5):406-411, 1968.

The therapeutic efficacy of 6-methylprednisolone and furosemid, alone or combined, was evaluated in 17 patients (11 males and 6 females, 48-68 yr old) with hepatic cirrhosis and ascites. All patients received collateral treatment (paromomycin, enemas, detoxicants, special diets) to combat high blood nitrogen and ammonia levels or to prevent hepatic coma. Furosemid alone (80-120 mg/day x 10-20 days) satisfactorily improved diuresis in 3 of 6 cases, increasing daily urine output from an average of 420 to 700 ml. The latent period from start of treatment to increased diuresis was 3-7 days. Five patients given 6-methylprednisolone alone (40 mg/day x 15-25 days) showed marked improvement in diuresis (from an average of 430 to 980 ml/day) within 10-15 days. Once diuresis was established the dosage of steroid could be reduced to 16 mg/day without significantly affecting the urine output. In 6 patients, combined diuretic-cortisone therapy (each at a dose of 40 mg/day x 10-15 days) produced a considerable increase in diuresis (from an average of 483 to 1351 ml/day) in 2-4 days. Subsequent reduction of the steroid dose and suspension of the diuretic did not reduce the urine volume. In all 3 groups the blood nitrogen, glucose and ammonia were within normal limits during treatment.

7472 **DRAINAGE OF THE THORACIC DUCT IN CIRRHOSIS OF THE LIVER.** *(Fr.)* Leger, L. (Cochin Hosp., Paris, France) and P. Lagneau. pp. 67-71 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7473 **SURGERY ON THE TERMINATION OF THE THORACIC DUCT IN PORTAL HYPERTENSION AND ITS COMPLICATIONS.** *(Fr.)* Alivisatos, C. N. pp. 64-66 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7474 **HISTOLOGICAL APPEARANCE OF THE INTRAHEPATIC LYMPHATICS IN CIRRHOSIS.** *(Fr.)* Feroldi, J. pp. 55-60 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7475 **THE LYMPHATIC CIRCULATION OF THE LIVER IN CIRRHOSIS.** *(Fr.)* Baggenstoss, A. H. (Mayo Clin., Rochester, Minn.). pp. 49-54 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

7476 **FEVER IN THE CIRRHOTIC PATIENT.** *(Fr.)* Naudin, G. *Concours Med* 90(1):257-260, 1968.

7477 **ELECTRON MICROSCOPY OF THE CIRRHOTIC LIVER.** *(It.)* Bianchi, F. B. (Inst. Gen. Med. Clin., U. Bologna, Italy) and G. Bacci. *Arch Ital Mal Appar Dig* 34(2): 141-151, 1967.

7478 **FREQUENCY AND SIGNIFICANCE OF HYPERKALEMIA DUE TO SPIRONOLACTONE TREATMENT IN PATIENTS WITH DECOMPENSATED CIRRHOSIS OF THE LIVER.** *(Hun.)* Radó, J. (Janos Hosp., Budapest, Hungary), J. Marosi and J. Takó. *Orv Hetil* 109(35):1911-1914, 1968.

7479 **ATTEMPTS AT IMMUNOSUPPRESSANT THERAPY IN ALCOHOLIC CIRRHOSIS.** *(Fr.)* Lamotte, M. (Lariboisière Hosp., Paris, France), J. M. Segrestaa and C. Caulin. *Entret Bichat [Ther]* pp. 118-120, 1967.

7480 **ZIEVE'S SYNDROME.** *(Fr.)* Cachin, M. and J. P. Petite. *Rein Foie* 11:27-41, 1968.

7481 **HEMODYNAMICS OF THE INTRAHEPTATIC CIRCULATION IN PATIENTS WITH CIRRHOSIS: REHABILITATION OF THE ROLE OF THE HEPATIC ARTERY IN ORDINARY CIRRHOSIS.** *(Fr.)* Mouktar, M. (Vittel, France). *Rein Foie* 11:197-210, 1968.

7482 **COMPARATIVE STUDIES ON CIRRHOSIS OF THE LIVER IN MUNICH, ZURICH AND CHICAGO.** *(Ger.)* Meister, H. P. (Inst. Path., U. Munich, Germany). *Verh Deutsch Ges Path* 51:421-424, 1967.

7483 **EMERGENCY TREATMENT OF HEMORRHAGE DUE TO PORTAL HYPERTENSION BY MEANS OF A TEMPORARY EXTRACORPOREAL PORTACAVAL SHUNT.** *(Fr.)* Champeau, M. (Corentin-Celton Hosp., Issy-les-Moulineaux, France), M. Arsac, P. Pineau, P. Bruchou, G. Elbhard and A. Apoil. *Mem Acad Chir (Paris)* 93(24/25):807-816, 1967.

7484 **POST-INFECTIOUS CIRRHOSIS IN CHILDREN.** *(It.)* Madon, E. (Pediat. Clin., U. Turin, Italy) and M. Martini Marui. *Minerva Pediat* 20(34):1716-1729, 1968.

7485 **THE POSSIBILITY OF PURIFYING THE BLOOD BY EXTRACORPOREAL PERFUSION OF AN HETEROLOGOUS LIVER IN THE TREATMENT OF HEPATIC COMA: REPORT OF FOUR CASES.** *(It.)* Dagradi, A. (Inst. Surg. Symptomatol., U. Padua, Italy), V. Puchetti, S. Radin, N. Di Fulvio, M. Zennaro and A. Bano. *Chir Ital* 19(5):569-581, 1967.

7486 **THE RESULTS OF SPLENORENAL SHUNT IN CIRRHOSIS.** *(Fr.)* Hepp, J., J. Moreaux and H. Bismuth. *Bull Soc Med Hop Paris* 118(12):1101-1107, 1967.

7487 **LONG-TERM RESULTS OF END-TO-SIDE TRUNCAL PORTACAVAL SHUNT FOR CIRRHOSIS: A STUDY OF 100 PATIENTS OPERATED ON BETWEEN 1959 AND 1964.** *(Fr.)* Fouet, P., M. Hivet and Chevrel. *Bull Soc Med Hop Paris* 118(12):1109-1122, 1967.

7488 **RENAL LESIONS ASSOCIATED WITH CIRRHOSIS OF THE LIVER IN INFANCY AND CHILDHOOD.** *(E.)* O'Kell, R. T. (Child. Mercy Hosp., Kansas City, Mo.). *Amer J Dis Child* 116(1):13-15, 1968.

See also: 6679,6948,6966,6968,6973,6995,7423,7451

7489 INVESTIGATIONS ON THE ROLE OF MUCUS IN THE PATHOGENY OF CHOLELITHIASIS.

(E.) Juvara, I. (Dr. I. Cantacuzino Hosp., Bucharest, Romania), D. Velican and C. Velican. *Rev Roum Med Intern* 4(4):239-246, 1967.

Mucosa from the neck, body, and fundus of 38 adult gallbladders (5 normal, 3 with acute cholecystitis, 2 with cholesterolosis, 28 with cholelithiasis) was analyzed histochemically for the presence of acid, vic-glycol and amino groups. In the normal gallbladders, both carboxyl and sulfate groups were present in the mucous layer at the epithelial surface. Protein digestion in this layer was followed by liberation of free acid groups with a resultant increase in the electronegative charge of the mucous membrane in the zone of contact with bile. In cholecystitis, the number of free acid groups in the mucous membrane was increased due to increased sialic acid and mucoprotein synthesis and to protein hydrolysis. In the more chronically inflamed gallbladders with cholelithiasis, the increased accumulation of acidic groups at the surface persists with a predominance of the sulfate groups of the mucoproteins. An increased secretion of mucus in cholelithiasis was accompanied by an accumulation of negative electric charges at the lining epithelium, possibly due to proteolysis occurring due to a decreased pH of the bile. This abnormal accumulation of electronegative charges at the surface of the mucous membrane may be considered as a destabilizing factor for the colloidal systems in the bile, resulting in apolar molecules leading to flocculation of the micelles and formation of the initial precipitation nuclei for biliary calculi.

7490 JAUNDICE WITH CHOLECYSTITIS.

(E.) Fish, J. C. (U. Texas Med. Branch, Galveston), D. D. Williams and R. D. Williams. *Arch Surg* 96(6):875-877, 1968.

The cause of the jaundice could be established in 92% of 200 patients with both jaundice and cholecystitis without the necessity of implicating biliary tract inflammation: common duct stones in 114, pancreatitis in 27, cancer in 20, hepatocellular disease in 18, hemolytic disorders in 5, and unknown causes in 16. No evidence was found that cholecystitis produces jaundice in the absence of lesions causing obstruction of the bile ducts. The operative finding of acute or chronic cholecystitis is not an adequate explanation for jaundice of any degree and further search, including common bile duct and pancreas exploration and liver biopsy, should be performed if an obvious ductal obstruction is not noted.

7491 ONE THOUSAND CONSECUTIVE CHOLECYSTECTOMIES.

(E.) Magee, R. B. (Altoona Hosp., Pa.) and R. C. MacDuffee. *Arch Surg* 96(6):858-862, 1968.

A retrospective study of 1000 consecutive patients who had undergone cholecystectomy is presented. There were 790 women and 210 men, with an age range of 15-81 yr, the highest incidence being in the 4th and 5th decade. Right upper quadrant or epigastric pain was present in 83%, while back or scapular radiation of pain was present in 98%. Pain was present for more than 2 yr prior to surgery in 55% of patients and previous abdominal operations (cholecystostomy, 2.2%, appendectomy, 17.0%; hysterectomy, 13.1%; other, 12.4%) had been performed in 42.5%. A constant physical finding was tenderness and percussion tenderness in the right upper quadrant (95%). In only 25% was there a leukocyte count above 10,000 on admission. Biliary calculi were present on survey films of the abdomen in 10.4%. The twice-repeated oral cholangiogram was 97% accurate in diagnosis, but calculi or some degree of inflammation were present in 91% of those operated on with normal X-ray findings. Common duct exploration was performed in 20.4%; calculi were found in the common duct in 12.7%. In only 0.8% were calculi found in the common duct and not in the gallbladder. Normal gallbladders without calculi were removed in 1.5% of patients. The gross mortality was 2.2%; under age 65 it was 0.7%. The mortality for those undergoing common duct exploration in the presence of choledocholithiasis was 4.7%.

7492 THE EFFECT OF BILE DIVERSION ON INDUCED HYPERCHOLESTEROLEMIA.

(E.) Keig, H. E. (Creighton U. Sch. Med., Omaha, Neb.), R. L. Belmonte Jr., J. M. McKain, J. F. Duesman and F. A. Miller. *Arch Surg* 96(4):613-618, 1968.

After producing hypercholesteremia in 20 adult mongrel dogs by near-total thyroidectomy complemented by 100 mg propylthiouracil daily and a daily diet consisting of 20 g cholesterol and 40 g animal fat, biliary diversion procedures were performed in 3 groups. The technique consisted of division of the common bile duct and cholecystoenterostomy to 3 different levels of the terminal small bowel (100, 200 and 300 cm proximal to the ileocecal junction). Dogs in a 4th group, used as controls, had sham celiotomy. In the group in which bile was diverted into the terminal ileum (100 cm level), the serum cholesterol dropped precipitously from a preoperative average of 800 mg% to normal (100-200 mg%) and remained at a normal level for the entire 6-month observation period (except for 1 animal that died of gastrointestinal hemorrhage). In the group with diversion to the 200-cm level, a gradual decline of serum cholesterol was noted from an average preoperative level of 670 mg% to 218 mg% over 18 weeks, with maintenance at this level for 6 months. In the group with diversion to the 300-cm level, the initially widely divergent serum cholesterol levels gradually decreased to an average of 226 mg% over 16-19 weeks. In the control group, the preoperative value was 816 mg% and the postoperative level was 790 mg%. Bile diversion thus reduces cholesterol absorption even though the ileal absorptive surface is unaffected, indicating that a significant proportion of cholesterol is normally absorbed from the proximal small intestine.

7493 THE USE OF "CYACRINE" ADHESIVE IN SURGERY FOR ACUTE CHOLECYSTITIS.

(Rus.) Melkumov, B. A. (All-Union Sci.-Res. Inst. Surg. Appar. Instr., Moscow, USSR), S. A. Mushegian, I. A. Kul'kova and G. S. Lipovetskii. *Khirurgiia (Moskva)* 44(3):97-100, 1968.

"Cyacrine", an acrylonitrile adhesive, was used to close the bed of the gallbladder and the stump of the common bile duct after cholecystectomies performed on 30 patients (28 women and 2 men) with acute cholecystitis. Blind sutures were then made in the abdominal wall. In all cases, inflammation was limited to the gallbladder and had not spread to adjacent organs or tissue. All patients had cholelithiasis and phlegmon of the gallbladder; 4 had gangrene. No evidence of blood or bile was tained in serous fluid obtained from 2 patients in whom cap- ıry drains had been inserted. No operative complications curred which could be attributed to the use of the adhesive. o patients developed pneumonia and 1, an exacerbation of ronic pyelonephritis. Follow-up examinations made 6 months 1 yr after surgery showed that none of these patients had veloped postoperative hernias.

94 CHOLESTEROSIS OF THE GALLBLADDER: A CLINICAL, RADIOLOGICAL, AND STATIS- CAL STUDY. *(Fr.)* Crismer, R. (Beauregard Med. Ctr., ge, Belgium). *Actualites Hepatogastroent Hotel Dieu* 3(2): 3, 1967.

a 5-yr period, polypoid cholesterosis of the gallbladder was gnosed in 34 patients (21 women, 13 men), 60% of whom re aged 40-60 yr. Symptoms referable to the biliary tract re present in 18 cases. In 16 cases, however, symptoms were erable to the stomach or colon, and cholesterosis was only gnosed on radiological examination. Cholesterosis was as- iated with migraine in 4 cases. None of the 34 patients had ras' functional syndrome, but 2 had "strawberry gallbladders" which there were concomitant adenomyomatosis and neuro- tosis. All of these patients responded to medical treatment l a low-fat diet. These findings are compared to others re- rted in the literature.

95 BENIGN TUMORS AND PSEUDOTUMORS (DYSPLASIAS AND HAMARTOMAS) OF THE LLBLADDER: A CLINICAL AND RADIOLOGICAL UDY. *(Fr.)* Albot, G. (Hotel Dieu, Paris, France) and J. isson. *Actualities Hepatogastroent Hotel Dieu* 3(2):55-78, 67.

48 patients (38 women and 10 men) with adenomyomatosis, erticulosis, hamartomas, and adenomas of the gallbladder, 17 re subjected to cholecystectomy because of symptoms re- able to the biliary tract and radiological signs of biliary dys- esia. In 31 cases the diagnosis was made preoperatively by olecystography. Fifteen of the 48 patients were between 40 l 50 yr of age. The most common clinical symptoms were n (38 cases), including biliary colic (6 cases); migraine (7 es); and dyspepsia. Pain was extremely variable in nature. ly 1 patient had vesicular colic; another had acute pain accom- ied by a fever. Migraine was usually not ophthalmic in na- e, but was accompanied by digestive disorders and varied in quency from once a week to once a month; in 5 cases it was ompanied by pain. Dyspepsia was most commonly associated h intestinal symptoms including constipation (13 cases), rrhea (5 cases), and alternating constipation and diarrhea (8 es). Six patients had biliary lithiasis. Radiological signs and results of preoperative biliary radiomanometry are described. contrast to other reports in the literature, obesity did not m to be a predisposing factor. Neuroautonomic dystonia oc- red frequently among the women in this series. One patient, o had been treated with estrogens during pregnancy, had sodes of premenstrual biliary colic and another had her first ack of biliary colic after a pregnancy during which she had n given estrogen therapy.

96 THE VALUE OF PLASTIC SURGERY ON THE DUODENAL PAPILLA: RESULTS OF FOLLOW- 'S ON PATIENTS OPERATED ON BETWEEN 1956 AND 1965. *(Ger.)* Dadgar-Dehkordie, I. (Munic. Hosp., Ulm/Donau, Germany). *Bruns Beitr Klin Chir* 216(3): 256-263, 1968.

In a 15-yr period, 1300 operations performed on the extrahepatic biliary tract included 358 (27.5%) reconstructive operations on the duodenal papilla. The patients (77.3% women, 22.7% men) ranged in age from 24-74 yr. Of these 358 patients, 149 had histories of duodenal disorders, 22 had histories of stomach disease (9 gastric resections), 78 had histories of acute pancreatitis (recurrent in 34 patients), and 44 had undergone cholecystectomies, either alone or with choledochoduodenostomy. Symptoms disappeared after surgery in 32 of these 44 patients, but 12 probably had undiagnosed papillary stenosis at the time of surgery. There were 16 operative deaths (4.47%) due to pancreatitis (8 cases), failure of duodenal sutures (3 cases), pulmonary embolisms (3 cases), and circulatory insufficiency (3 cases). Another 5 patients died of unrelated causes. Operative mortality has been reduced since patients have been given Trasylol before surgery. Questionnaires from 215 patients revealed that surgery gave good or excellent results in 76.7% of the cases, satisfactory results in 15.4%, and poor results in 7.9%. Clinical examination of 144 patients showed that results were good or excellent in 90.9%, satisfactory in 5.6%, and poor in 3.5%. Cholangitis was responsible for symptoms in all 5 patients in the latter group. Radiological examination of 138 patients showed the presence of stones in the common bile duct in 6 asymptomatic patients and papillary insufficiency in 9. None of these patients had the postcholecystectomy syndrome. These results are compared with others reported in the literature.

7497 DRAINING THE ABDOMINAL CAVITY IN OPERATIONS ON THE EXTRAHEPATIC BILE DUCTS. *(Rus.)* Dzhavadian, A. M. (N. I. Pirogov 2nd Moscow Med. Inst., USSR), L. B. Krylov and I. V. Rudchenko. *Khirurgiia (Moskva)* 44(3):88-92, 1968.

Results obtained by 3 methods of drainage and suture following biliary tract surgery were compared: 1) use of a gauze tampon with or without a rubber drain (305 patients operated on for chronic cholecystitis); 2) use of a rubber drain inserted into the wound by the method of S. I. Spasokukotskii, plus antibiotic therapy (671 patients treated for chronic and acute cholecystitis); and 3) use of primary sutures (59 patients). Of the patients in group (1), 255 (74%) had cholecystectomies, 20 (6%) the procedure of Pribram and Vishnevskii, and 53 (18% cholecystectomies with internal or external drainage or primary sutures. The percentage of operative deaths (12.5%) was highest among patients in group (1). Death was due to peritonitis in 13 cases and to liver and renal failure in 12. Other complications, which were also most frequent among patients in group (1), included subdiaphragmatic and subhepatic abscesses (7 cases, 2.3%) and infection (28 cases, 9.2%). The flow of bile was noted in 24% of the patients in group (1) and in only 11% of those in group (2). The method of Spasokukotskii is recommended for drainage of the abdominal cavity following biliary surgery.

7498 PRESSURE CHANGES IN THE BILE DUCTS DURING THE EARLY POSTOPERATIVE PERIOD. *(Pol.)* Jablonka, S. (1st Surg. Clin., Acad. Med. Lublin, Poland) and E. Dacka. *Pol Przegl Chir* 39(12):1299-1304, 1967.

Six hr after sphincterotomy or exploratory choledochotomy, cholangiomanometry revealed an elevated resting pressure in the common bile duct (above 150 mm H_2O) in 16 of 22 and 7 of 8 patients, resp., and in 10 of 22 and 3 of 8 the pressure was above 200 mm H_2O; increased sphincter resistance (above 300 mm H_2O) was noted in 10 of 22 and 4 of 8 cases at this same time. One day postoperatively, abnormally high common bile duct pressures were recorded in 18 of 22 and 5 of 8 cases, resp., while increased sphincter resistance was noted in 11 of 22 but 0 of 8. Later, the bile duct pressure and sphincter resistance decreased in both groups, so that by the 4th postoperative day an elevated common bile duct pressure was seen in only 11 of 22 (above 200 mm H_2O in only 1 of 22) and 0 of 8, and increased sphincter resistance was found in 2 of 22 and 0 of 8 cases. During the entire 4 days, however, the average common bile duct pressures and sphincter resistances were higher in the sphincterotomy patients than in the choledochotomy patients, the average difference reaching a maximum of 65 and 98 mm H_2O, resp., on the 2nd postoperative day. Five days postoperatively, there was still a difference of 13-14 mm H_2O between the readings in the 2 groups. It is concluded that the motor function of the common bile duct and sphincter of Oddi returns to normal within 4 days after exploratory choledochotomy and within 7 days after sphincterotomy.

7499 MICROSCOPIC AND FUNCTIONAL CHANGES IN THE LIVER IN CHOLELITHIASIS. *(Pol.)* Albert, Z. (Dept. Path. Anat., Acad. Med. Wroclaw, Poland), K. Czyzewski, K. Kawecki and T. Szepietowski. *Pat Pol* 18(3): 359-369, 1967.

Following surgery for cholelithiasis in 87 patients (21 men and 66 women, 21-72 yr old), microscopic examination of liver biopsy specimens revealed changes in the capsule in 71.7% (62 cases) and changes in the parenchyma and stroma in 89.7% (78 cases). The duration of cholelithiasis in these patients ranged from a few weeks to 40 yr; 26 patients had obstructive jaundice of 8 days - 3 months duration, and 30 patients (including 24 of those without jaundice and 6 with it) had signs of inflammation around the gallbladder. The 62 patients with abnormalities in the capsule (51 of whom showed inflammatory changes) included 19 with jaundice and 20 with gallbladder inflammation. The 78 patients with stromal and parenchymal changes included 28 with severe changes, of whom 15 had jaundice and 8 had inflammation. Among the entire group of 78, 25 had jaundice and 27 had inflammation. Abnormalities observed included cholangitis and other inflammatory changes (77 cases, including 25 with jaundice and 26 with gallbladder inflammation), connective tissue proliferation (34 cases, including 13 with jaundice and 10 with gallbladder inflammation), polynucleated cells (40 cases), hyperemia (30 cases), and fatty or aqueous degeneration (17 and 14 cases, resp.). The severity of the pathological changes was independent of age and duration of cholelithiasis and was affected only slightly by the presence or absence of gallbladder inflammation, but was directly related to the presence of jaundice and cholestasis. Liver function tests (performed postoperatively in 72 cases) were abnormal in 59.7% but showed no clear correlation with the microscopic findings. Among 27 cases with several abnormal liver function tests, 14 had severe histological changes and 25 showed changes of some type, while among 29 with normal liver function tests, 3 had severe histological changes and 23 had changes of some type. Of 22 patients with severe histological changes, 19 thus had at least one abnormal liver function test, but of 8 with normal hepatic histology, 2 had abnormal tests. It is concluded that the hepatic changes seen in these patients represent a nonspecific reaction to pathological processes taking place in the area of the portal vein.

7500 EXPERIMENTAL REPLACEMENT OF THE CO MON BILE DUCT: OPERATIVE TECHNIQUE AND FIRST MORPHOLOGICAL AND FUNCTIONAL RESULTS. *(E.)* Rigotti, E. (Dept. Gen. Clin. Surg., U. Turin, Italy), R. De Matteis and M. Dellepiane. *Panminerva Med* 9(11):520-530, 1967.

A new procedure for substitution of the common bile duct is described which consists of the introduction of a corrugated tubular Teflon prosthesis into a tunnel in the duodenal wall by means of a previously described technique for ureteral replacement. The clinical, functional, and morphological results in 10 dogs have been assessed for up to 4 months after surgery. Three dogs died: 1 during the operative procedure, 1 o the 5th postoperative day from bile peritonitis due to an anast motic rupture, and 1 on the 68th postoperative day from a pe forated gastric ulcer. Tolerance of the procedure was good in the remaining 7 dogs. Serum bilirubin showed a slight increas (direct fraction) on the 10th postoperative day, but had returned or was returning to normal by the 30th day. Urinary and fecal bile pigments were in accordance with the serum bili rubin observations. Radiological contrast studies after sacrific revealed a patent prosthesis in every case, although in some, slight dilatation of the bile ducts was noted above the prosthe dilatation of the gallbladder was never seen. Histological exam tion of the liver revealed a normal architectural pattern in all cases. Neither scar tissue stenosis nor occlusion of the lumen due to bile incrustations were noted at the anastomoses. Thes excellent initial results await confirmation over more prolonge follow-up periods.

7501 LONG-TERM RESULTS OF PAPILLOSPHINCT OTOMY FOR BILIARY TRACT DISEASE: PE SONAL EXPERIENCE. *(It.)* Nicora, E. (Galliera Hosp., Gen Italy), G. C. Venturi and C. Schenardi. *Rass Arch Chir* 5(5, Suppl. 1):115-119, 1967.

Transduodenal sphincterotomies accounted for 202 (11.8%) o all operations performed on the biliary tract in a 7-yr period. Of these, 58 (28.7%) were performed for stenotic papillo-oddi with or without cholelithiasis; 66 (32.6%) for stenotic papillo-odditis associated with calculi in the pancreatic duct or in the papilla; 71 (34.8%) for calculi in the pancreatic duct alone; an 7 (3.9%) for papillo-odditis associated with chronic inflammat of the head of the pancreas. Sphincterotomies were combine with external drainage of the pancreatic duct by a Kehr tube Flörcken's end-to-end choledochoduodenostomy if the pancre duct was greatly dilated, if there was severe inflammation, or there was chronic inflammation of the head of the pancreas. Follow-ups were made of 128 (63%) of these patients: 82 by examination and 46 by questionnaires. Results were very goo in 89 cases (71%), good in 33 cases (25%), and mediocre in 6 cases (4%). These last 6 patients had dyspepsia, episodes of diarrhea requiring diet therapy, and occasional pain in the rig hypochondrium. None had colic, jaundice, or fever. Results were about the same when sphincterotomies were the only rocedure employed as when they were combined with other perations.

502 THE EFFECTS OF PHLOROGLUCINOL ON BILIARY LITHIASIS IN MICE. *(Fr.)* Duteil, J. .. Lafon Res. Ctr., Maisons-Alfort, France), F. Rambert and . M. Desgranges. *Therapie (Paris)* 23(2):433-440, 1968.

ddition of 0.2 or 0.8% phloroglucinol to the diets of female ice for 110 days produced no changes in the weights of the ver or gallbladder, no cholelithiasis, and no changes in blood r liver cholesterol. Discoloration of the liver, seen in some ice receiving phloroglucinol, is an early sign of its effect on e liver. Addition of phloroglucinol to diets rich in cholesterol d cholic acid produced a further increase in the weight of the ver. However, phloroglucinol prevented the increase in liver eight produced by sodium dehydrocholate. Cholelithiasis, resent in 50% of the mice fed a diet rich in sodium dehydrocho- te, occurred in only 20% of those receiving the same diet plus hloroglucinol. Addition of phloroglucinol to diets rich in olesterol and cholic acid further increased the weight of the llbladder by about 200%. When added to diets rich in sodium ehydrocholate, 0.8% phloroglucinol reduced the increase in llbladder weight by about 20%. Addition of phloroglucinol to iets rich in cholesterol and cholic acid and to those rich in dium dehydrocholate reduced the increases in liver cholesterol ut had little effect on blood cholesterol or liver lipids.

503 DOUBLE GALLBLADDER (VESICA FELLEA DUPLEX). *(Hun.)* Mincsev, M. (U. Med. Sci., udapest, Hungary) and M. Ihász. *Magy Sebesz* 20(6):405-409, 967.

504 TORSION OF THE GALLBLADDER. *(Hun.)* Szabó, B. (Kaposvar Hosp., Hungary), M. Võlgyi nd G. Varga. *Magy Sebesz* 20(5):305-308, 1967.

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506 THE COURSE OF COMPLICATED CHOLELITHIASIS IN THE AGED. *(Dut.)* De Groote, J. (St. afael Acad. Hosp., Louvain, Belgium), G. Van Driessche and . Vandenbroucke. *T Gastroent* 10(3):267-281, 1967.

507 AN UNUSUAL CASE OF PERFORATION OF THE EXCRETORY BILE DUCTS. *(Cz.)* Frydl, . (Dept. Path. Anat., OUNZ, Rokycany, Czechoslovakia). *esk Gastroent Vyz* 21(7):436-440, 1967.

508 BUD-LIKE IMPLANTATION OF A BILIARY TRACT FISTULA INTO THE SMALL INTESTINE SING A SKIN GRAFT. *(Bul.)* Koichev, I. (Distr. Health Soc., use, Bulgaria). *Khirurgiia (Sofiia)* 21(2):163-168, 1968.

509 DIABETES MELLITUS AND GALLSTONES WITH SPECIAL REFERENCE TO INSULIN SSAY. *(E.)* Norton, J. J. (El Cajon Valley Hosp., Calif.), .. R. Smith and E. Hamper. *Amer J Gastroent* 50(3):214-220, 968.

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7513 NECROTIZING PANCREATITIS AS A COMPLICATION OF BILIARY SURGERY. *(Fr.)* Isserlis, G. (Bichat Hosp., Paris, France). *Entret Bichat [Chir]* pp. 47-52, 1967.

7514 TREATMENT OF THE BILIARY DYSKINESIA RESULTING FROM CHRONIC INTOXICATION WITH NITROBENZENE DERIVATIVES. *(Rus.)* Kleiner, A. I. (Kharkov Inst. Occup. Hyg., USSR), L. N. Kazinskaia and N. V. Timofeiev. *Vrach Delo* (12):104-105, 1967.

7515 CLINICAL SYMPTOMS AND TREATMENT OF LAMBLIASIS OF THE BILIARY TRACT. *(Rus.)* Boiko, G. F. (Odessa Med. Inst., USSR), I. A. Savel'iev and A. Ie. Zolotarev. *Vrach Delo* (11):136-138, 1967.

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7517 CLINICAL ASPECTS OF CHOLESTASIS DUE TO CHOLEDOCHOLITHIASIS. *(Sp.)* Gasto Parramona, J. M. (Nuestra Senora del Sagrado Corazon de Jesus Hosp., Barcelona, Spain) and J. Badosa-Gallart. *Rev Esp Enferm Apar Dig* 26(9):1093-1097, 1967.

7518 CHOLESTASIS AND DILATATION OF THE COMMON BILE DUCT. *(Sp.)* Pi-Figueras, J. (Santa Cruz y San Pablo Hosp., Barcelona, Spain), V. Artigas, J. Pi-Figueras Jr. and R. Gajo. *Rev Esp Enferm Apar Dig* 26(9):1113-1119, 1967.

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7520 BILIARY ANASTOMOSES TO A DEFUNCTIONALIZED JEJUNAL LOOP IN THE TREATMENT OF CHOLESTASIS. *(Sp.)* Isern Arus, J. (Santa Cruz y San Pablo Hosp., Barcelona, Spain), R. Camprodon Bertran, R. Trias Rubies and A. Llaurado Tomas. *Rev Esp Enferm Apar Dig* 26(9):1141-1144, 1967.

7521 ICTERIGENIC CARCINOLITHIASIS (ASSOCIATION OF GALLBLADDER CHOLELITHIASIS WITH A CANCER OF THE HEPATIC DUCT). *(Fr.)* Demarty, R., L. Allouard and Ben Moussa. *Mem Acad Chir (Paris)* 93(24/25):773-776, 1967.

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7523 THE CONSERVATIVE TREATMENT OF BILIARY DISEASES. *(Ger.)* Heinkel, K. (Bad Cannstatt Hosp., Stuttgart, Germany). *Landarzt* 44(4):173-176, 1968.

7524 MALFORMATIONS AND DISEASES OF THE BILIARY TRACT IN CHILDREN. *(Ger.)* Hasse, W. (Surg. Clin., Free U. Berlin, Germany). *Landarzt* 44(4):177-182, 1968.

7525 MULTIPLE CHOLELITHIASIS NOT INVOLVING THE COMMON BILE DUCT: RESULTS OF OPERATIVE CHOLANGIOGRAPHY. *(Por.)* Barroso, F. L. (Guanabara State Tradesmen's Hosp., Rio de Janeiro, Brazil), S. Kaplan, M. Ferrari and S. G. Meira. *Hospital (Rio)* 72(6):1699-1708, 1967.

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7527 ACUTE CHOLANGITIS WITH HIGH BLOOD NITROGEN LEVELS. *(Fr.)* Trad, J. (Franco-Moslem Hosp., Bobigny, France) and A. Paraf. *Gaz Med France* 75(17):3585-3594, 1968.

7528 SURGERY OF THE GALLBLADDER AND BILE DUCTS: QUESTIONS AND ANSWERS. *(E.)* Boyd, C. E. (Doctor's Hosp., Shreveport, La.), C. H. Lupton and M. O. Cantor, Eds. Melrose, Mass., Amer. Soc. Abdom. Surg., 1967. 146 pp.

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7531 CYSTICODUODENOSTOMY: REVIEW OF CASES. *(Fr.)* Salembier, Y. (Lille, France). *Lyon Chir* 63(5):690-694, 1967.

7532 THE ROLE OF THE VASCULARIZATION O THE COMMON BILE DUCT IN THE PATHOGENESIS OF SOME POSTOPERATIVE COMPLICATIONS *(Fr.)* Constantinesco, O. (Emerg. Hosp., Bucharest, Roman V. Ranga and I. Cristesco. *Lyon Chir* 63(5):674-679, 1967

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7535 SOME TACTICAL PROBLEMS IN THE TRE MENT OF CHRONIC CHOLECYSTITIS. *(Ru* Galkin, V. A. (Moscow, USSR). *Sovet Med* 30(10):67-72,

7536 THE DISTRIBUTION AND KINETICS OF RE MOVAL OF CARBON 14 LABELED BILIRU IN THE DOG WITH LIGATION OF THE COMMON BILE *(E.)* Abei, T. (Johns Hopkins U. Sch. Med., Baltimore, Md. F. L. Iber. *Johns Hopkins Med J* 122(2):112-127, 1968.

7537 GALLSTONE ILEUS. *(E.)* Cantor, M. O. (G Hosp., Detroit, Mich.). *J Abdom Surg* 10(3): 80, 1968.

7538 SURGERY OF THE COMMON BILE DUCT. Kuker, L. H. (Carroll, Iowa). *J Abdom Surg* 11-16, 1968.

7539 CHOLEDOCHODUODENOSTOMY. *(E.)* Lo C. J. (Kenyatta Natl. Hosp., Nairobi, Kenya). *Afr Med J* 44(9):358-361, 1967.

7540 CORRELATIONS BETWEEN THE DYSFUNCTIONAL AND ORGANIC FORMS OF EXTRAHEPATIC BILIARY TRACT DISEASE. *(It.)* Pistacchi, E. (Inst. Clin. Surg., U. Pavia, Italy). *Rass Clin Sci Ist Biochim Ital* 44(4):117-122, 1968.

See also: 6668,6674,6721,6878,6879,6892,6933,6935,6955,7016,7017,7102,7265

7541 "SPASMOPLUS" (CIBA) IN THE ABDOMINAL SPASTIC PAIN SYNDROME: A CLINICAL CONTRIBUTION. *(It.)* Galliani, A. (Maggiore Hosp., Bologna, Italy) and P. P. Testoni. *Gazz Int Med Chir* 73(16):1458-1469, 1968.

"Spasmoplus" suppositories (0.5 g aminophenazone, 0.6 g allobarbital, 0.05 g adiphenine, and 0.04 g codeine phosphate) were used in the treatment of 80 patients (25 men and 55 women, aged 16-72 yr) with renal colic, biliary colic, gastroduodenitis, peptic ulcers, typhlocolitis, acute cystopyelitis, and adnexial colic. Depending upon the severity of the symptoms, 1 suppository was given b.i.d. or t.i.d. for 1-11 days. The therapeutic effect developed about 30 min after administration and lasted for 4-6 hr with intense pain and 8-10 hr in less severe cases. Complete remission of pain was obtained in 70% of these patients and almost complete remission in 30%. One patient developed an allergic rash which disappeared soon after treatment was discontinued.

7542 THE THERAPEUTIC EFFECTS OF HYPERBARIC OXYGENATION ON ACUTE INTESTINAL OBSTRUCTION WITH SPECIAL REFERENCE TO ITS PATHOLOGY. *(E.)* Matsukura, S. (Nippon Med. Sch., Tokyo, Japan) and A. Shirota. *Int Surg* 49(1):71-78, 1968.

The effect of hyperbaric oxygenation on acute intestinal obstruction and the lethal action of *Escherichia coli* endotoxin was studied in dogs, rats, and mice. A strangulating obstruction was established in the upper portion of the small intestine of dogs or rats, after which the animal was kept for 2 hr at 3 atmospheres absolute pressure of oxygen. Control animals were similarly prepared but were not placed in hyperbaric chambers. Arterial levels of oxygen and carbon dioxide, blood, pH, kidney, and adrenal glands, the amount of *E. coli* endotoxin in the blood and peritoneal fluid, and the acetylcholine level in the blood were determined in oxygenated and control animals 4 hr after obstruction and 2 hr after hyperbaric oxygenation. In dogs, the lowering of oxygen partial pressure, the elevation of the partial pressure of carbon dioxide, and the lowering of arterial blood pH were inhibited fairly well by hyperbaric oxygenation, as were the liberation of *E. coli* endotoxin into the blood and the increase of acetylcholine in the blood. The effectiveness of hyperbaric oxygenation against anoxic changes in the liver, kidneys and adrenal glands was verified by light and electron microscopy in rats. The survival time of mice given twice the minimum lethal dose of *E. coli* endotoxin i.p. 2 hr before hyperbaric oxygenation was remarkably prolonged compared to nonoxygenated controls. Whereas all controls died within 12 hr after endotoxin administration, 6 of 10 oxygenated mice survived 24 hr. Neither pyribenzamine, cyproheptadine, the antiplasmin DV-79, nor cortisone had any significant protective effect, but 2 of 10 atropine-treated mice survived 24 hr and 6 of 10 survived 12 hr. These results confirm the probable role of acetylcholine in fatal endotoxin shock.

7543 PATHOGENESIS AND PATHOPHYSIOLOGY OF HYPERTONIC DEHYDRATION WITH DIARRHEA. A CLINICAL STUDY OF 59 INFANTS WITH OBSERVATIONS OF RESPIRATORY AND RENAL WATER METABOLISM. *(E.)* Bruck, E. (Sch. Med., State U. New York, Buffalo), G. Abal and T. Aceto Jr. *Amer J Dis Child* 115(2):122-144, 1968.

In a planned, prospective study, detailed clinical and laboratory analyses were made of 59 children (19 days-25.2 months old) with hypertonic dehydration and diarrhea in an effort to elucidate the pathogenesis and pathophysiology of this condition. All patients showed signs of severe dehydration with 3-26% loss of body weight. The cause of dehydration in 57 of 59 cases was diarrhea lasting 1-7 days. Many patients refused or were unable to retain any feedings or fluid. Excess solute intake could be documented in only 4 patients. Twenty-eight patients had signs of respiratory or ear infection; case histories of a few unusual cases are presented. No pathogenic *Shigella, Salmonella* or *E. coli* were isolated from the stools of any patient. The mean concentration of serum sodium before treatment was 161 mEq/liter. Blood urea nitrogen and inorganic phosphorus were elevated in most cases. Serum calcium was usually in the normal range and the mean potassium concentration was 5.0 mEq/liter. With two exceptions, significant metabolic acidosis was observed. In ¼ of the cases in which it was determined, blood sugar was above 200 mg%. Minute ventilation (measured in 16 cases) and respiratory water loss (measured in 10 cases) were frequently 2-3 times normal levels. Although glomerular function was impaired initially, there was no evidence of impaired concentrating ability of the renal tubules. No pathologic lesions were demonstrated to account for the clinical signs of temporarily disturbed CNS functions (hyperirritability, coma or semicoma, shrill cry). No single cause of the hypertonicity of body fluids could be identified in the majority of cases and it is concluded that several factors were probably operating. Phenomena related to the acute illness (such as water loss via hypotonic diarrheal stools, high insensible water loss, low fluid intake) were probably more significant than any intrinsic defects of homeostasis.

7544 ANTIBODY RESPONSE TO THE COMMON ENTEROBACTERIAL ANTIGEN OF CHILDREN WITH SHIGELLOSIS, SALMONELLOSIS OR URINARY TRACT INFECTION. *(E.)* Diaz, F. (Dept. Pediat., State U. New York, Buffalo) and E. Neter. *Amer J Med Sci* 256(1): 18-24, 1968.

Using a previously-described hemagglutination and hemagglutination-inhibition test, paired serum specimens from 50 children with shigellosis, 50 with *Salmonella* enteritis, and 50 with acute or chronic urinary tract infection due to gram-negative bacteria were tested for the antibody response to O antigen and the common antigen (CA) of enteric bacteria. O antibody titers equaled or exceeded titers for CA antibodies in all patients with salmonellosis and urinary tract infection, whereas 18% of the subjects with shigellosis had CA antibody titers exceeding those of O antibodies. CA antibody titers of 1:160 or greater were observed in 56% of patients with shigellosis and in only 24% or 18% of subjects with salmonellosis or urinary tract infection, resp. When the data were reanalyzed for patients above one yr of age (since 9 cases of salmonellosis occurred at less than 1 yr of age but shigellosis was not encountered in this age group), the respective percentages were 56% for shigellosis, 29% for salmonellosis and 19% for urinary tract infection. Significant changes in the titers of CA antibodies

(4-fold or greater increase or decrease) occurred more often in the shigellosis group (38%) than in the other two groups (16%). Excluding patients under 1 yr old, 19 children with shigellosis (38%), 8 with urinary tract infection (17%) and 8 with salmonellosis (20%) showed significant changes in antibody titers. The more frequent CA antibody response of children with shigellosis was not due to a general enhanced immunologic reactivity since significant or persistent high titers (1:640 or higher) of the respective O antibodies were documented in 84% of patients with shigellosis, 76% with salmonellosis and 92% with urinary tract infection. Two additional patients with typhoid fever and another with splenomegaly of unknown cause demonstrated significant CA antibody titers. These results suggest that the CA antibody response of some patients with enterobacterial infections may depend on the site of the infection.

7545 THE ETIOLOGY AND PATHOGENESIS OF FUNCTIONAL FORMS OF DUODENAL STASIS IN ORGANIC DISEASES OF THE STOMACH AND DUODENUM. *(Rus.)* Mirzaiev, A. P. (Leningrad Sanit. Hyg. Med. Inst., USSR) and M. A. Petushinov. *Vestn Khir Grekov* 100(6): 13-16, 1968.

Degenerative changes were found in the intramural nerve ganglia of Auerbach's plexus in a number of patients with dilatation and atonia of the duodenum. Studies of 14 patients with peptic ulcer (4 duodenal, 6 gastric, and 6 pyloroduodenal ulcers) showed that these changes were most severe in patients with severe clinical symptoms and long histories, particularly in those with stenosis and penetrating ulcers, and when the ulcers were located in the duodenum or the pyloroduodenal region. The same types of changes were found in 4 patients with stomach cancer who had had symptoms of gastritis for 2-10 yr, in 1 patient who developed afferent-loop syndrome after gastrectomy for peptic ulcer, and in 1 patient with a gastroenterostomy. Less severe, and possibly reversible, changes were found in the intramural nerve apparatus of the duodenum in 26 patients with peptic ulcer, 5 with stomach cancer, 3 with gastric polyps, 1 with pancreatitis, and 2 with cholecystitis even though these patients had no symptoms of duodenal stasis. Hofmeister's and Finsterer's techniques are recommended for patients with duodenal stasis. If mild signs of stasis appear after surgery, the duodenal contents should be aspirated twice a day until intestinal peristalsis reappears. In patients with severe duodenal stasis, better evacuation of the duodenal contents can be obtained by performing a duodenojejunal anastomosis or by completing a gastric resection with a Roux-en-Y gastroenterostomy.

7546 THE GEOGRAPHIC DISTRIBUTION OF THE SHIGELLAE. I. CHANGES IN THE ETIOLOGICAL STRUCTURE OF DYSENTERY IN RUSSIA AND THE USSR (1900-1950). *(Rus.)* Krasheninnikov, O. A. (Sechenov 1st Moscow Med. Inst., USSR). *Zh Mikrobiol* 45(6):21-31, 1968.

An analysis of published data revealed that at the beginning of the 20th century most cases of dysentery in Russia were caused by *Shigella grigoriewa-shigae* although a small number of *Sh. flexneri* outbreaks were reported. In the middle of the 1920's a considerable increase occurred in the number of cases caused by *Sh. flexneri* in Leningrad with a decrease in those caused by *Sh. grigoriewa-shigae.* This was accompanied by a decreased severity and changes in morbidity. During the middle of the 1930's another increase occurred in the incidence of dysentery caused by *Sh. grigoriewa-shigae* so that more than 50% of the total number of *Shigella* cultures isolated in the entire USSR were of this species. A subsequent decrease occurred in the *Sh. grigoriewa-shigae* morbidity and an increase in *Sh. flexneri* morbidity. This shift, which occurred to different extents in different parts of the country, was associated with increased morbidity and decreased mortality due to the use of sulfa drugs During World War II no changes occurred in the etiological structure of dysentery. Afterwards, dysentery caused by *Sh. grigoriewa-shigae* was eradicated completely because of measures taken to prevent and limit epidemics.

7547 GASTROINTESTINAL TUMORS IN MÉRIDA. *(Sp.)* Ocaríz, J. H. (Los Andes Hosp., Mérida, Venezuela) and S. Parra. *Rev Col Med Estado Merida* 26(9): 39-57, 1968.

Of 7405 patients hospitalized in Los Andes Hospital in Mérida, Venezuela in the 5 yr between 1960 and 1964, 387 had confirmed malignant tumors of which 237 (61.24%) were found in the gastrointestinal tract. A table showing the incidence of gastrointestinal tumors per 100,000 inhabitants as a factor of age reveals a peak incidence at age 60-69. The patients included 161 men and 76 women. Tne most common form was stomach cancer which accounted for 174 cases (73.41%); this proportion was similar to that found among the 242 patients treated for gastrointestinal malignancies at the Digestive Disease Clinic during the period 1957-67, including 181 cases of stomach cancer (74.8%). Of those who developed stomach cancer, almost all were farmers and 97 of the 142 who were questioned used "chimo," a tobacco extract containing 2.7-3.7 g% nicotine; salts of Fe, Si, Ca, Mg, K, and Na; sucrose; glucose; and other substances. Chimo is chewed and may also be a factor in the development of cancer of the mouth and esophagus. Another thing all these patients had in common was their deficient diet which was composed primarily of grains, bananas, root crops, and cheese with very little meat, eggs, fish, vegetables, and fruits.

7548 COMBINATION OF *ESCHERICHIA COLI* ENTERITIS WITH STAPHYLOCOCCAL INFECTIONS IN INFANTS. *(Rus.)* Timofeieva, G. A. (Leningrad Pediat. Med. Inst., USSR). *Pediatriia* 47(3):40-44, 1968.

In a 3-yr period (1962-1964), 688 children (aged from birth to 1.5 yr) had intestinal infections of bacterial etiology as confirmed by cultures of stools, blood, throat smears, and pus. These included 342 (49.7%) with *E. coli* infections, 162 (23.5%) with staphylococcal infections, and 45 with mixed *E. coli* and *Staphylococcus* infections. There were no age differences between infants with mixed infections and those with *E. coli* or *Staphylococcus* infections alone. Mixed infections were more severe than single infections. The mortality rate for mixed infections was 6.6% compared to 2.6% for *E. coli* infections and 3.7% for *Staphylococcus* infections. In 19 cases staphylococcal infections preceded *E. coli* infections, in 16 cases *E. coli* infections preceded staphylococcal infections, and in 10 cases the infections occurred simultaneously. Pneumonia was found in all 3 children who died: 2 had catarrhal-desquamative enteritis and 1, catarrhal-desquamative gastroenteritis.

7549 **STAPHYLOCOCCAL ENTEROCOLITIS IN INFANTS.** *(Rus.)* Plotnikova, N. G. (Crimean Med. Inst., Simferopol, USSR), Ie. A. Shapovalenko and S. S. Filippskaia. *Pediatriia* 47(3):44-47, 1968.

Staphylococci were isolated from cultures of blood, pus, and stools collected from 78 infants, aged 10 days to 11 months, with enterocolitis. Many of the strains isolated were resistant to antibiotics, particularly penicillin. Gastrointestinal symptoms predominated in 23 cases. Symptoms included subfebrile or normal temperature, asthenia, refusal to feed, slight loss of weight, and skin pallor. Nine of these children had acute forms with vomiting, convulsions, and hyperesthesia. In addition to meteorism and abdominal pain, patients passed liquid stools containing mucus, large numbers of white cells, and bacteria. The blood picture showed reduced hemoglobin values, high neutrophil counts, and accelerated erythrocyte sedimentation rates. Symptoms lasted 18-20 days. Another 10 infants had localized and superficial skin infections, 36 had involvement of other organs, and 9 had combined staphylococcal and *E. coli* infections. Besides the symptoms described above, these 9 patients spiked fevers periodically, rapidly developed signs of dehydration, and had steatorrhea. The blood picture showed reduced hemoglobin values, leukocytosis or leukopenia, eosinophilia, and sedimentation values within normal limits. Therapy consisted of antibiotics, vitamins, enzymes, γ-globulin, plasma, erythrocytes, *Staphylococcus* anatoxin, methylandrostendiol, Apilak (crude drug from secretions of the allotrophic gland of bees), i.v. glucose, s.c. insulin, and therapeutic enemas.

7550 **VARIATIONS IN THE HEPATIC ARTERIES AND THE VASCULARIZATION OF THE PANCREAS: THEIR CLASSIFICATION AND FREQUENCY, THEIR ARTERIOGRAPHIC AND SURGICAL DETECTION, AND THEIR EFFECT ON SURGERY OF THE HEPATIC PEDICLE, PANCREATECTOMY, AND TRANSPLANTATION OF THE LIVER AND PANCREAS.** *(Fr.)* Mellière, D. (La Pitié Hosp., Paris, France). *J Chir (Paris)* 95(1):5-42, 1968.

The vascular architecture of the liver and pancreas is described in detail on the basis of 50 autopsy specimens and 150 arteriograms, and the importance of a correct understanding of the hepatic and pancreatic vasculature for the prevention of hemorrhage and/or ischemia during surgery on these organs is emphasized. It is pointed out, for example, that there are 4 possible configurations of the left gastric, splenic and superior mesenteric arteries, from which the 3 fetal hepatic arteries are derived: in 95% of the cases, the left gastric and splenic arise from the celiac trunk while the superior mesenteric arises separately, but in 3% of these, the hepatic artery arises directly from the aorta instead of from the celiac trunk; in 4%, furthermore, all 3 arteries arise separately, while in 0.5% all 3 have a common origin and in another 0.5% the splenic and superior mesenteric have a common origin while the left gastric artery arises separately. There are also 6 possible arrangements of the hepatic arteries themselves: in 60% of all adults, there is only a single hepatic artery arising from the celiac trunk, the other 2 fetal arteries having been reabsorbed; in another 4%, the picture is the same except for a very thin, nonfunctional, accessory hepatic artery; in 36%, however, there are important variations: for example, in about 19% of the cases the single hepatic artery supplies only half the liver, the other half being supplied by a branch of the left gastric or superior mesenteric; in 6%, the hepatic artery divides into 2 branches near its origin; in 3%, all 3 fetal hepatic arteries have persisted; and in 8% the main hepatic artery has been reabsorbed, the liver being supplied only by the residual fetal hepatic artery or arteries. The head of the pancreas is supplied with blood via both the celiac and mesenteric systems, which anastomose to form the pancreatic arcades (plus a large mesentericoceliac anastomiasis in 5%). In all cases studied, 2 superior and 2 inferior pancreaticoduodenal arteries arose from the gastroduodenal artery and superior mesenteric artery, resp., although in some cases the posterior superior pancreaticoduodenal artery also supplied the biliary tract. The gastroduodenal artery, however, may arise from either branch of the main hepatic artery, from an accessory hepatic artery, or from the celiac trunk (1 of 49 cases). Variations are also found in the blood supply to the body and tail of the pancreas, the main problem being the presence or absence of a transverse circulation (in addition to the pancreatic artery which is derived from the splenic) which is derived from the dorsal pancreatic; the latter was found in 44 of 50 specimens and was derived from either the hepatic artery, the celiac trunk, or the splenic artery. The pancreatic circulation generally also gives rise to visceral arteries supplying the jejunum and colon. On the basis of a detailed comparative study, it is concluded that selective arteriography is more effective than dissection for elucidating the variations in the vasculature of the pancreas and the hepatic pedicle. Detailed conclusions are drawn from the anatomical findings with regard to the most suitable techniques for surgery on the pancreas and hepatic pedicle, with particular attention to the problems presented by transplantation of the pancreas and liver.

7551 **THE TREATMENT OF PROTOZOAL INFESTATION OF THE INTESTINES.** *(Por.)* Neto, V. A. (Fac. Med., U. São Paulo, Brazil) and A. Bianchi. *Rev Goiana Med* 13(3/4):155-166, 1967.

7552 **THE SUTURING OF ABDOMINAL INCISIONS: A COMPARISON OF MONOFILAMENT NYLON AND CATGUT.** *(E.)* Tagart, R. E. B. (Addenbrooke's Hosp., Newmarket, England). *Brit J Surg* 54(11):952-957, 1967.

7553 **DEGENERATIVE ARTERIOPATHY ORIGINATING IN THE THREE ABDOMINAL VISCERAL ARTERIES: A STUDY OF THE ANATOMY AND POSTMORTEM BLOOD FLOW.** *(Fr.)* Martin, E. (Bichat Hosp., Paris, France), P. Besancon and C. Debray. pp. 178-182 in: Circulation Lymphatique et Arterielle en Pathologie Digestive Abdominale [Lymphatic and arterial circulation in abdominal digestive pathology], Proc. Ann. Meet. French Gastroent. Soc., Paris, Sept. 30-Oct. 1, 1967, Paris, Masson & Co., 1967, 354 pp.

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7560 EFFECTIVENESS OF LEVOMYCETIN IN THE TREATMENT OF TYPHOID FEVER. *(Rus.)* Lyskovtsev, M. M. (Kharkov Med. Inst., USSR). *Vrach Delo* (12):84-86, 1967.

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DESCRIPTION OF INDEXES

Starting with Volume 3, Number 1, the SUBJECT and AUTHOR INDEXES have been generated directly from computer-stored files. The Indexes to prior volumes were manually prepared.

The Subject Index uses a system of permuted *major class* terms combined with SEE and SEE ALSO references. A *major class* term is one embracing a broad concept which can be broken down into *minor class* terms.

The *major class* terms have been permuted so that the accession numbers of the indexed abstracts and citations will be found by the User under each of the principal component words of the *major class* term, no matter which component word he looks up first in the alphabetical index. For example, the accession numbers of abstracts and citations concerning

BENIGN GASTROINTESTINAL NEOPLASMS

will also be found under

GASTROINTESTINAL NEOPLASMS, BENIGN

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In several cases, more than one term is used in the literature to express a single concept. To ensure consistency of ndexing and facilitate complete retrieval, it was necessary to select one of these synonyms for indexing purposes. The SEE references have been inserted to guide the User to the preferred term.

The SEE ALSO references are designed to call the User's attention to related terms.

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NCER SURVIVAL RATES
06972* 07007* 07026* 07029* 07052 07086* 07120 07207*
07209* 07338*
NCER TREATMENT
07066 07209* 07257 07258 07286
NCER TREATMENT,RADIOTHERAPY IN
06936 06972* 06996 07565
NCER,ABDOMEN
07559
NCER,AGE FACTORS IN
07007* 07086* 07247 07343*
NCER,ANUS
07232 07258 07260
NCER,BILIARY TRACT
06892 07516 07520 07521
NCER,DISEASES ASSOCIATED WITH
07021* 07050 07287 07343* 07394 07521 07545* 07585
NCER,DUODENUM
07120.
NCER,ESOPHAGUS
06838* 06972* 06987 06990 06996 06997 07003 07587
NCER,GALLBLADDER
07528
NCER,GASTROINTESTINAL
06899 07155 07547* 07569 07585
CANCER,LARGE INTESTINE
06882* 06949 07207* 07208* 07209* 07213* 07221 07226
07232 07234 07237 07238 07247 07258 07273 07557
CANCER,LIVER
06788* 06837* 06842* 06893 07338* 07342* 07343* 07349*
07372 07380 07394 07476 07585
CANCER,METABOLIC CHANGES IN
07019*
CANCER,METASTATIC
06788* 06837* 06864* 06963 06996 07007* 07029* 07050
07067 07092 07209* 07338* 07342* 07343* 07559
CANCER,PANCREAS
06880* 07279* 07286 07287 07291 07297 07585
CANCER,RECTUM
06882* 06888 06949 07206* 07207* 07213* 07232 07257
07258
CANCER,SMALL INTESTINE
07081* 07086* 07557
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06748* 06755 06788* 06838* 06843* 06859* 06904 06936
07007* 07008* 07014* 07015* 07018* 07019* 07020* 07021*
07023* 07026* 07029* 07032 07034 07035 07041 07048
07050 07051 07052 07053 07054 07055 07056 07061
07065 07066 07071 07072 07073 07076 07146 07147
07155 07180 07286 07329 07545* 07587
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06691 06699* 06701* 06702* 06703* 06706* 06707* 06714
06717 06810 06836* 06851* 06881* 07112 07123 07194*
07197 07202 07203 07391 07446* 07577
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06699* 07577
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07187*
CARBOHYDRATE METABOLISM
06717 06810 06826* 07132* 07191* 07277* 07371
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06778* 06786* 06787* 06788* 07187* 07355 07430
CARCINOGENESIS,LARGE INTESTINE
07229
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07342* 07380 07421* 07430 07437 07438 07441 07587
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07008* 07014* 07018* 07022* 07023* 07126*
CARCINOGENS
07437 07438
CARCINOID SYNDROME
SEE ALSO CARCINOIDS
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07258
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SEE ALSO CARCINOID SYNDROME
07011*
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06990 07018* 07026* 07048 07061 07066 07338* 07372
07394 07569
CARDIOSPASM
SEE ALSO ACHALASIA
06974*
CARDIOVASCULAR SYSTEM
SEE ALSO PORTAL HYPERTENSION
06666* 06679* 06771* 06866* 06886 07075 07254 07279*
07287 07340* 07344* 07360 07379 07396 07412 07550*
07553
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06856*
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06856*
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07216* 07252
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06863* 07189* 07197
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06810 07565
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06741* 07017* 07039 07170 07344* 07392 07458 07466*
07572
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07467*
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SEE ALSO TOXIC EFFECTS ON ESOPHAGUS
SEE ALSO TOXIC EFFECTS ON GASTROINTESTINAL TRACT
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07424 07431 07432 07514 07587
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07286
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07225 07245

CHILDREN,APPENDICITIS IN
07253 07566
CHILDREN,BILIARY DISEASE IN
07466*
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07334* 07335* 07524
CHILDREN,CANCER IN
07035 07234
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06855* 07334* 07347* 07397 07466* 07469* 07484 07488
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07200 07543*
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07548* 07549*
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06970* 06982 06989
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07543* 07544*
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06858*
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07111 07566
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06855* 07347*
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07058 07524
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07211* 07224 07234 07246
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06938 07334* 07335* 07413
CHILDREN,MALABSORPTION IN
06669* 07112 07200
CHILDREN,PANCREAS IN
07091 07276* 07292
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07159 07165
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06970*
CHILDREN,SALMONELLOSIS IN
07544*
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07544*
CHILDREN,SMALL INTESTINE IN
06669* 07091 07097 07105 07106 07109 07112 07113
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06695 06739* 06751* 07012* 07035 07058 07059 07070
07074
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06852* 06854* 06869* 07278* 07442* 07444* 07448* 07452*
07459 07462 07484
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06743*
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06674* 06853* 06878* 06937 06947 06962 07265 07503
07525 07528 07538
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07445*
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06879* 07357 07426 07491* 07496* 07497* 07498* 07510
07511 07522 07532 07534 07537 07538
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06858* 06872* 06955 07000 07489* 07490* 07493* 07499*
07511 07522 07523 07526 07529 07535 07561
CHOLECYSTITIS DIAGNOSIS
06879*
CHOLECYSTITIS ETIOLOGY
07528
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07303* 07361 07362 07528
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06879* 06933 07265
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06957
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06668* 07311 07337* 07453* 07490* 07496* 07507 07518
07540
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06879* 06880* 06892 06923 07265 07402 07491* 07517
07524 07525
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07512
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07489* 07502* 07512 07525 07526
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07512 07526 07580
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07491* 07511 07522 07525 07531 07534
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07493* 07506 07517 07523 07530 07538
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07506
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07102 07304* 07325 07327 07346* 07499* 07501* 075
07506 07509 07521 07526 07537 07580
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07575
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06799*
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06923 06928 06931 06932 06939 07337* 07346* 073
07376 07381 07382 07385 07386 07399 07417 074
07425 07426 07440 07445* 07449* 07453* 07454* 074
07516 07517 07518 07520 07523
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SEE ALSO HORMONAL CONTROL OF GALLBLADDER
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06802 07502*
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06698* 06813 06815 07439 07492*
CHOLESTEROL METABOLISM
06827*
CHOLESTEROL METABOLISM,LIVER
06783* 06813 07494*
CHOLESTEROL SYNTHESIS
06813 07083*
CHOLESTEROL,SERUM
07083*
CHRONIC HEPATITIS
06787* 06855* 06868* 06901 06947 07134* 07354 073
07365 07370 07391 07404 07408 07411 07416 074
07451* 07453*
CHRONIC PANCREATITIS
06871* 06872* 06880* 07036 07285 07286 07299* 073
07302* 07303* 07304* 07305* 07309* 07313 07314 073
07321 07325 07326 07328
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07454*
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07481
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06992
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07266
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06892 07532
CIRCULATION,ESOPHAGUS
06724*
CIRCULATION,GASTROINTESTINAL
06666* 06702* 06817 06835 06886 06891 06896 068
07550* 07553 07554 07555 07556
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06887 07254 07557
CIRCULATION,LIVER
06666* 06679* 06682 06683 06798* 06814 06864* 068
06885 06893 06894 06948 06960 06965 07331* 073
07333* 07344* 07352 07356 07357 07358 07359 073
07366 07375 07384 07388 07403 07412 07423* 074
07470* 07472 07473 07474 07475 07481 07550*
CIRCULATION,MESENTERY
06666* 06681 06834 06888 07024* 07115 07264* 075
07555
CIRCULATION,PANCREAS
06681 06684 06771* 07279* 07280* 07296 07324 075
CIRCULATION,PORTAL
06705* 06864* 06948 06992 07331* 07340* 07347* 073
07375 07384 07396 07403 07405 07406 07407 074
07481 07486 07487 07499*
CIRCULATION,RECTUM
06888
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06890 06897 06898 06977 07024* 07084* 07088 070
07090 07204 07557
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07126* 07127* 07130* 07167
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SEE ALSO ESOPHAGUS VARICES
SEE ALSO LIVER COMA
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07085* 07349*
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06840* 06855* 06893 06900 06901 06923 06932 069
06947 06959 06966 07354 07391 07464*
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07482
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07397 07423* 07432 07434 07438 07451* 07464* 074
07482 07484
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07354 07415 07450* 07479
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06855* 07334* 07347* 07397 07466* 07469* 07484 074
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07354 07360 07469* 07474 07475 07476 07477 074

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07340* 07341* 07347* 07404 07468* 07470* 07471* 07472
07478 07479 07481 07485
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07464*
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07344* 07465* 07471* 07472 07474 07475 07478
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07334* 07337* 07387 07405 07488
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07481
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06848* 06948 06968* 06973* 07016* 07136* 07343* 07344*
07406 07407 07416 07423* 07451* 07464* 07465* 07466*
07470* 07473 07476 07480 07483 07485 07486 07487
07488 07558
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07433
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06787* 07337* 07467*
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06791*
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SEE ALSO ENTEROCOLITIS
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07222
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06880* 06887 06921 07263* 07265 07267 07270
COLITIS ETIOLOGY,ULCERATIVE
07185 07254 07261* 07262* 07264* 07270 07271
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07261* 07263* 07264*
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06882* 07267 07269 07271 07557
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07267 07273
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07272 07273
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07266
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07238 07265
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07262*
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07183* 07266 07268
COLON
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COLON,IRRITABLE
06882*
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SEE LIVER COMA
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07344* 07347* 07355 07485
COMMON BILE DUCT
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06668* 06674* 06720* 06721* 06734* 06879* 06914 07294
07339* 07346* 07356 07357 07358 07366 07385 07426
07449* 07491* 07498* 07500* 07507 07513 07517 07518
07519 07528 07531 07532 07534 07536 07538 07539
CONGENITAL ANOMALIES
06886 06989 07059 07068 07084* 07109 07121 07211*
07224 07236 07245 07334* 07335* 07406 07413 07447*
07466* 07488 07503 07524 07538 07558
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07376 07377 07418* 07427 07439
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06770* 07009* 07470*
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07467*
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07397
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06672* 06746* 06747* 06766 06800* 06867* 07047 07148
07184 07212* 07272 07273 07307* 07320 07397 07425
07429
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06887 06890 07090 07181* 07182* 07183* 07184 07185
07186 07212* 07215* 07251 07252 07263* 07267 07269
07271 07557
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07075 07422*
CURLINGS ULCER
07131*
CYSTIC FIBROSIS
SEE MUCOVISCIDOSIS
CYSTS,LIVER
06842* 06893 06894 06938 07339* 07395 07409 07410
07417
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07283 07284 07285 07297 07507
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06859* 06936
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06678*

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06719* 07214*
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06802 06824 07079* 07192* 07198 07276* 07547*
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06851* 07202 07203
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06732 06967*
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06880* 07191* 07302* 07328 07451* 07509 07510
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06999*
DIAPHRAGM HERNIA
06942 06991 06994 06999
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06999
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06851* 06863* 06873* 07062 07078* 07089 07114 07129*
07145 07183* 07189* 07191* 07192* 07194* 07197 07198
07199 07262*
DIARRHEA IN CHILDREN
SEE ALSO CELIAC DISEASE
07200 07543*
DIETARY DEFICIENCIES
06802 06824 07079* 07192* 07198 07276* 07547*
DIETARY DEFICIENCIES IN LIVER INJURIES
06778* 06808 06810 07330*
DIETARY FACTORS
06709 06710 06712 06713 06714 06717 06735* 06735*
06794* 06795* 06802 06808 06820 06827* 06881* 07022*
07023* 07028* 07093 07106 07143 07188* 07195 07288
07380 07420* 07502* 07577
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07094 07194* 07195 07494* 07502* 07523 07535 07563
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07193*
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06699* 07577
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07290
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06769 06833 07577
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07193*
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06851* 07202 07203
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06669* 06699* 06707* 06851* 07202 07203
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06863* 07189* 07197
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06887 06890 07090 07181* 07182* 07183* 07184 07185
07186 07212* 07215* 07251 07252 07263* 07267 07269
07271 07557
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06680* 07224 07236 07246
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07045 07046
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07446*
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07089 07196
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07266
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07021* 07050 07287 07343* 07394 07521 07545* 07585
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07102 07304* 07325 07327 07346* 07499* 07501* 07505
07506 07509 07521 07526 07537 07580
DISEASES ASSOCIATED WITH CIRRHOSIS
06848* 06948 06968* 06973* 07016* 07136* 07343* 07344*
07406 07407 07416 07423* 07451* 07464* 07465* 07466*
07470* 07473 07476 07480 07483 07485 07486 07487
07488 07558
DISEASES ASSOCIATED WITH GASTRITIS
07046 07274*
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07016* 07134* 07344* 07416
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07058 07348* 07349*
DISEASES ASSOCIATED WITH PANCREATITIS
06992 07290 07299* 07302* 07309* 07310 07312 07313
07317 07318 07324 07558

DISEASES ASSOCIATED WITH PARASITIC DISEASES
07339* 07570
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06757 07134* 07136* 07137* 07152 07154 07155 07162
07179 07180 07274* 07304* 07327 07545* 07580
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06980 06995 07318
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07238 07265
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07446* 07449* 07451* 07453* 07454* 07458 07460 07462
07484
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07293
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06858*
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06742* 06748* 06766 06858* 06934 06956 06958 07014*
07019* 07027* 07039 07040 07041 07068 07134* 07152
07154 07160 07176 07361 07362 07581 07582 07583
07584
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07471*
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07495*
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07104 07110
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06974*
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07495*
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06689
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06921 07100 07227 07233 07270
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07100 07104 07110
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07249
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07100
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07100
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06887 07238
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06890 07109
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06702*
DRUG EFFECTS ON BILIARY TRACT
07530
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06672* 06678* 06906 06912 07135* 07254
DRUG EFFECTS ON LARGE INTESTINE
07216*
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06769* 06780* 06799* 06805 06812 06818 06821 06822
06823 06853* 07401 07425 07426 07438
DRUG EFFECTS ON MOTILITY
06702* 06719* 06721* 06726* 06731 06906 06912
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06775
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06758 07013* 07038
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06744* 06752* 06754* 06769*
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06825*
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06795* 06811 06812 06818 06823
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07514 07515 07523 07540
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07535
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06953 07064 07181* 07184 07382 07541* 07549* 07551
07560 07563 07564 07574 07578 07588 07590 07591
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07272
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06965 07351* 07367 07404 07408 07471* 07478 07479
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07307* 07315 07319 07320 07321 07323
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07047
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06763 06766 07020* 07038 07064 07125* 07142 07143
07144 07156 07157 07160 07161 07163 07170 07171
07172 07173 07174 07175 07176
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06822 06823 06868* 07364 07425 07438 07441
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06758 06766 06769* 07127* 07148 07179 07397
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07376 07377 07418* 07427 07439
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06776 06935 07324 07325 07326
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07036 07060 07062 07129* 07132* 07177
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SEE ALSO SMALL INTESTINE
06670* 06687 06720* 06729 06733 07043 07531
DUODENUM CANCER
07120
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06843*
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DUODENUM DIVERTICULA
07104 07110
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06843* 06849*
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07120
DUODENUM MOTILITY
06857* 06872* 07545*
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07080* 07448*
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06849*
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07108
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06843* 07158
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07448*
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07108 07110 07277* 07496* 07539
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07017* 07108 07118
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06843* 06883* 07006 07010* 07062 07071 07115 0712
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07158 07162 07164 07165 07177 07178 07180 0723
07285 07397
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SEE ALSO SHIGELLOSIS
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06915 06916
DYSENTERY DIAGNOSIS
06870* 06874* 06952 06953 06954
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07546* 07571
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06874* 07546*
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07210*
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07551
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07006 07028*
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Public Health Service
National Institutes of Health

SEPTEMBER/OCTOBER 1968
VOLUME 3, NO. 9/10
7592–8916

ume 3, No. 9/10 Abstract and Citation Nos. 7592-8916

GASTROENTEROLOGY ABSTRACTS AND CITATIONS

A monthly publication of the
National Institute of Arthritis and Metabolic Diseases

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arth
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The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the Nati
Institute of Arthritis and Metabolic Diseases will provide a much needed current awareness tool to scientists
will facilitate greater integration of research and clinical efforts in this field The number and great diversit
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provide the readers with a readily systematized compilation of current published work. The publication
provide the greatest usefulness if these interested investigators will contribute their ideas and comments
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Pancreatitis	8395-8421	998
Liver and Biliary Tract	8422-8538	1001
Hepatic Injury	8539-8591	1011
Viral Hepatitis	8592-8630	1018
Cirrhosis	8631-8654	1022
Gallbladder and Biliary Tract Diseases	8655-8716	1025
General	8717-8916	1030

Use of Funds for printing this publication
approved by the Director of the Bureau of
the Budget on October 7, 1968.

sale by the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. scription price per year in the United States, Canada, and Mexico, $8.50; foreign $2.25 additional; $1.00 per gle copy. Payment is required in advance, and check or money order should be made payable to the perintendent of Documents.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. F journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medi Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mC, μC	milli-, microcurie(s)
ADP	adenosine diphosphate	mg	milligram(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
BSP	sulfobromophthalein	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes),
μg	microgram(s)		red blood count
hr	hour(s)	resp.	respectively
i.m.	intramuscular	Rev.	review (only in citations)
i.p.	intraperitoneal	RNA	ribonucleic acid
IU	international unit(s)	s.c.	subcutaneous
i.v.	intravenous	sec	second(s)
kg	kilogram(s)	SGOT	serum glutamic-oxalacetic transaminase
LD$_{50}$	median lethal dose(s)	SGPT	serum glutamic-pyruvic transaminase
LDH	lactic acid dehydrogenase	U	unit(s)
m	meter(s)	UV	ultraviolet
M	molar	WBC	white blood cells (leukocytes),
mEq	milliequivalent(s)		white blood count
mM	millimolar	yr	year(s)
μM	micromolar		

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

)2 THE VASCULAR ARCHITECTURE OF THE SMALL INTESTINAL MUCOSA OF THE MON-
Y *(MACACA MULATTA).* *(E.)* Reynolds, D. G. (Walter
ed Army Med. Ctr., Washington, D. C.), J. Brim and T. W.
eehy. *Anat Rec* 159(2):211-218, 1967.

cone-rubber microvascular injection compounds were used
reveal the mucosal vascular architecture of the monkey
ll intestine. The mucosal vascular patterns of this animal
e found to differ from the classical description: instead of
villus blood supply being delivered via a central villus ar-
ole, the arterioles ramify to the undersurface of the mucosa
ere they terminate in the capillary plexus surrounding the
pts of Lieberkuhn. The villus capillary net is derived directly
m the cryptic capillary plexus and is drained by a single,
tral villus vein. A secondary venous return system also ex-
which directly drains the cryptic plexus. These secondary
ous channels might represent a mechanism for regulating
proportional distribution of blood in the cryptic and villus
illary beds. Observations of rat and rabbit mucosa prepared
he same way revealed that these species more closely re-
ble the classical description.

)3 PHYTOSPHINGOSINE GROUPS AS QUANTITATIVELY SIGNIFICANT COMPONENTS OF
E SPHINGOLIPIDS OF THE MUCOSA OF THE SMALL
ESTINES OF SOME MAMMALIAN SPECIES. *(E.)* Okabe,
(Tufts U. Sch. Med., Boston, Mass.), R. W. Keenan and G.
midt. *Biochem Biophys Res Commun* 31(1):137-143, 1968.

mogenized mucosal scrapings of the small intestine of adult
fasted for 72 hr and dogs and rabbits fasted for 24 hr were
pared for the extraction and analysis of lipids by thin-layer
gas-liquid chromatography. The lipid base obtained by
tion of fraction X (absent from the reference chromatogram
g sphingolipids of monkey spinal cord) from the thin-layer
omatograms moved as a single spot at the same rate as C_{18}-
tosphingosine during rechromatography on thin-layer plates.
-liquid chromatography of the trimethylsilyl derivatives of
tion X yielded 1 symmetrical peak identical with that of
ethylsilyl phytosphingosine. Aldehydes formed during the
iodate oxidation of fraction X showed 1 major peak of penta-
anal and 2 small peaks of hexa- and heptadecanal. In the
mach and colonic mucosa of dogs, phytosphingosine ac-
nted for much smaller percentages of the total lipid bases
n in the small intestine. The unusually high phytosphingo-
/total lipid bases ratio might represent a tissue-specific fea-
of the sphingolipids of the small intestine, although a di-
exogenous origin of intestinal phytosphingosine groups by
orption of plant sphingolipids cannot be excluded.

)4 IDENTIFICATION OF A MACROMOLECULAR FACTOR IN THE ILEUM WHICH BINDS IN-
INSIC FACTOR AND IMMUNOLOGIC IDENTIFICATION
INTRINSIC FACTOR IN ILEAL EXTRACTS. *(E.)*
thenberg, S. P. (New York Med. Coll., N. Y.). *J Clin Invest*
4):913-923, 1968.

identification of immunologically intact intrinsic factor
) in guinea pig ileal extracts after *in vivo* incubation with
min B_{12} bound to normal human gastric juice, and the
identification, extraction, and properties of a factor in ileal extract which binds IF are described. ^{57}Co-labeled vitamin B_{12} ($^{57}CoB_{12}$) with a specific activity of 120-170 $\mu C/\mu g$ was used for all the studies and normal human gastric juice obtained after augmented histamine stimulation was the source of the intrinsic factor. There was virtually no radioactivity in the precipitate which resulted when $^{57}CoB_{12}$ bound to normal human gastric juice was subjected to 15% Na_2SO_4; following *in vivo* incubation of the gastric juice-$^{57}CoB_{12}$ mixture in the distal guinea pig ileum, however, the dialyzed extract of the washed mucosa contained traces of radioactivity which was precipitated by 15% Na_2SO_4. The formation of a 15% Na_2SO_4-insoluble fraction which contained $^{57}CoB_{12}$ also took place during *in vitro* incubation of gastric juice-$^{57}CoB_{12}$ with an extract of the ileal mucosa or brush border membranes. This insoluble fraction either did not form or was diminished in quantity by the addition of an excess of vitamin B_{12}-free normal human gastric juice, reducing the incubation pH to 2, incubating the mixture at 4C, pretreating the ileal extract at 56C for 30 min, incubating the reaction in sodium EDTA but not calcium EDTA, and incubating gastric juice-$^{57}CoB_{12}$ with an extract of jejunal mucosa. It is concluded that immunologically intact IF can be extracted from the ileum after *in vivo* incubation with gastric juice-$^{57}CoB_{12}$, and that a macromolecular factor is present in the distal ileal mucosa which binds IF both *in vitro* and *in vivo*, changing its solubility and electrophoretic properties. It is thought that this ileal binding factor is the previously postulated intestinal receptor for intrinsic factor.

7595 HISTOCHEMICAL LOCALIZATION OF ACID HYDROLASES IN PANETH CELLS OF THE HUMAN JEJUNUM.

(It.) Riva, A. (Inst. Norm. Human Anat., U. Cagliari, Italy) and D. Zaccheo. *Boll Soc Ital Biol Sper* 44(11):933-934, 1968.

Using various histochemical techniques, segments of small intestine from 3 male surgical patients (30-45 yr old) without pathology involving the intestine were examined for evidence of lysosomal acid hydrolase activity in the Paneth cells. Specific acid phosphatase, arylsulfatase, E 600-resistant esterase, β-glucuronidase and N-acetyl-β-glucosaminidase activity was demonstrated in minute granulations dispersed throughout the cytoplasm of the Paneth cells, but not in the characteristic Paneth cell granules. The distribution of the stained granules within the cytoplasm differed from that described for the lysosomes of absorptive villous cells. The lack of specific enzyme activity in the Paneth cell granules excludes the possibility that these are equivalent to lysosomes, but does not negate the hypothesis that Paneth cells participate in the digestive processes of the intestine. These observations support the ultrastructural findings (reported elsewhere) that lysosome-like dense bodies, not identifiable with the typical granules, are present in the cytoplasm of Paneth cells from the human jejunum.

7596 HEXOKINASE AND GLUCOKINASE ACTIVITIES IN BILE DUCT EPITHELIAL CELLS AND HEPATIC CELLS FROM NORMAL RAT AND HUMAN LIVERS.

(E.) Morrison, G. R. (Washington U. Sch. Med., St. Louis, Mo.). *Arch Biochem* 122(3):569-573, 1967.

Bile duct epithelial cells from interlobular portal tracts and hepatic cells from midzonal areas of the lobules were microdissected and separted from the livers of normal male Sprague-Dawley rats and 2 human cholecystectomy patients with normal liver function. These cells were then assayed for hexokinase and glucokinase activities. The results indicate that hepatic parenchymal cells have both hexokinase and glucokinase activity, while bile duct epithelial cells have little or no glucokinase activity but a hexokinase activity greater than that found in liver parenchymal cells.

7597 **SUBCELLULAR LOCATION OF VITAMIN D AND ITS METABOLITES IN INTESTINAL MUCOSA AFTER A 10-IU DOSE.** *(E.)* Stohs, S. J. (Dept. Biochem., U. Wisconsin, Madison) and H. F. DeLuca. *Biochemistry* 6(11):3338-3349, 1967.

The intracellular localization of ^{3}H from 10 IU of ^{3}H-vitamin D in rat or chick intestinal mucosa indicated that a major site of accumulation is the nuclear membrane. When the outer membrane of the nucleus was fragmented or stripped off with citric acid or Triton X-100, 60-80% of the nuclear radioactivity from ^{3}H-vitamin D could be removed. Of the ^{3}H from ^{3}H-vitamin D associated with the nuclei, 80% was in the form of polar metabolites which are known to be biologically active. The nuclear receptor sites for vitamin D and/or its metabolites could be saturated by the administration of large doses of the vitamin or of dihydrotachysterol-2, but not 7-dehydrocholesterol.

7598 **ORIGIN OF VASCULAR CHANNELS FROM EPITHELIAL TISSUE AS SHOWN IN LIVERS.** *(E.)* McDonald, H. G. (Diagn. Cell Res. Inst., Waco, Texas). *J Amer Med Wom Ass* 23(6):545-553, 1968.

Based on previous observations suggesting that lysis and transformation of carcinoma cells lead to vessel formation within the tumor, embryonic and adult chicken livers were studied histologically and histochemically in single and serial sections in a search for a similar mechanism in the histogenesis of blood vessels in normal tissues. Liquefaction of liver parenchyma leading to the formation of vascular spaces and the origin of endothelium from the peripheral remaining liver cells was demonstrated. In this process of vessel formation, the liquefied cell product contributed to the formation of the first plasma. No significant differences were noted in the histogenesis of blood vessels in the embryonic and adult chicken livers; however, the cellular lysis and differentiation of endothelium were more readily demonstrable in embryos due to the rapid proliferation of the vascular bed.

7599 **VARIATIONS IN THE JUNCTION OF THE HEPATIC DUCTS.** *(Fr.)* Nikolic, V. (Inst. Anat., Fac. Med., Zagreb, Yugoslavia). *Acta Anat (Basel)* 67(4):516-522, 1967.

The way in which the common hepatic duct was formed was studied on 300 cadavers of adults of both sexes. The junction was formed by the union of a right and left hepatic duct in 167 cases (55.7%), by the union of 3 bile ducts in 98 cases (32.7%), by the union of 4 ducts in 31 cases (10.3%), and by the union of 5 bile ducts in 4 cases (1.3%). When the hepatic ducts joined below the hepatic portal, the common hepatic duct was usually very short and, in 3 cases (1%), was not found at all. In the portal region the junction of the hepatic ducts was located in the right 3rd of the transverse fissure of the liver in 113 cases (37.7%), in the middle 3rd in 183 cases (61 and in the left 3rd in 4 cases (1.3%). In 173 cases (57.66%) the junction of the hepatic ducts was located in front of the right branch of the portal vein, in 103 cases (34.33%) in fron of the portal bifurcation, and in 14 cases (4.66%) as far as 2 cm to the left of the portal bifurcation. In 8 cases (2.66%) the junction was more than 2 cm to the right and in 2 cases (0.66%) more than 2 cm to the left of the portal bifurcation.

7600 **RADIOLOGICAL STUDY OF THE GASTROESOPHAGEAL PASSAGE IN INFANTS.** *(It.)* Cinque, N. A. (L. Gaslini Inst. Puericult., U. Genoa, Italy), A. Pelizza and D. G. Rovetta. *Pathologica* 59(875/876):193-206 1967.

7601 **ANATOMICAL BASIS FOR LOBAR RESECTI OF THE LIVER.** *(Rus.)* Ostroverkhov, G. Ie. (N. I. Pirogov 2nd Moscow Med. Inst., USSR), V. F. Zabrodsk and V. D. Zatolokin. *Khirurgiia (Moskva)* 44(5):151-157, 19

7602 **ELECTRON MICROSCOPIC OBSERVATIONS ON THE GANGLIA OF AUERBACH'S PLEXU AND AUTONOMIC NERVE ENDINGS IN *MUSCULARIS EXTERNA* OF THE MOUSE SMALL INTESTINE.** *(Jap.)* Ono, M. (Sapporo Med. Coll., Japan). *Sapporo Med J* 32(1): 56-74, 1967.

7603 **BIOCHEMICAL ANALYSIS OF THE STOMAC WALL IN NORMAL RATS.** *(E.)* Mózsik, G. (Med. Sci., Debrecen, Hungary), A. Szabó, M. Krausz and T. Jávor. *Scand J Gastroent* 2(4):321-325, 1967.

7604 **BIOCHEMICAL ANALYSIS OF VAGOTOMIZE STOMACH WALL IN RATS.** *(E.)* Mózsik, G. (U. Med. Sci., Debrecen, Hungary), A. Szabó, M. Krausz and T. Jávor. *Scand J Gastroent* 2(4):326-332, 1967.

7605 **AN ELECTRON MICROSCOPIC STUDY OF T EPIDERMIS OF THE TRANSITIONAL SKIN I THE HUMAN ANAL CANAL.** *(Jap.)* Baba, E. (Gifu U. Sch. Med., Japan). *J Jap Soc Coloproctol* 21(2):3-41, 1968.

7606 **THE DEMONSTRATION OF THE MESENTER COLLATERAL CIRCULATION IN YOUNG PA-TIENTS.** *(E.)* Sacks, R. P. (Letterman Gen. Hosp., San Francisco, Calif.), D. J. Sheft and J. H. Freeman. *Amer J Roentgen* 102(2):401-406, 1968.

7607 **NUCLEAR ELIMINATION FROM THE NORM BLAST OF FETAL GUINEA PIG LIVER AS STUDIED WITH ELECTRON MICROSCOPY AND SERIAL SECTIONING TECHNIQUES.** *(E.)* Campbell, F. R. (Dept. Anat., Stanford U., Calif.). *Anat Rec* 160(3):539-553, 1968.

7608 **THE HEPATIC LOBULES OF SUIDAE, TAYASSUIDAE, AND HIPPOPOTAMIDAE.** *(E.)* Steiner, P. E. (Sch. Med., U. Pennsylvania, Philadelphia) and H. L. Ratcliffe. *Anat Rec* 160(3):531-538, 1968.

7609 THE DYNAMIC MORPHOLOGY OF THE NORMAL AND ABNORMAL SMALL INTES-
INAL MUCOSA OF MAN. *(E.)* Shiner, M. (Cent. Middlesex
losp., London, England). *Bibl Paediat* 87:5-21, 1968.

7610 AN ELECTRON MICROSCOPIC STUDY OF ESOPHAGEAL EPITHELIUM IN THE NEW-
ORN AND ADULT MOUSE. *(E.)* Parakkal, P. F. (Boston
. Sch. Med., Mass.). *Amer J Anat* 121(2):175-196, 1967.

7611 ELECTRON AND LIGHT MICROSCOPIC OBSERVATIONS ON RELATIONSHIPS BETWEEN
YMPHOCYTES AND INTESTINAL EPITHELIUM. *(E.)*
leader, R. D. (U. Nebraska Coll. Med., Omaha) and D. F.
anders. *Amer J Anat* 121(3):763-774, 1967.

7612 FINE STRUCTURE OF NERVE TERMINALS IN THE HUMAN GUT. *(E.)* Pick, J. (New York
. Sch. Med., N. Y.), C. De Lemos and A. Ciannella. *Anat
ec* 159(2):131-146, 1967.

613 ELECTRON MICROSCOPIC OBSERVATIONS OF DENSE BODIES IN PARIETAL CELLS OF
HE HAMSTER *(MESOCRICETUS AURATUS)*. *(E.)* Winborn,
. B. (U. Tennessee Med. Coll., Memphis) and D. E. Bockman.
nat Rec 159(4):387-404, 1967.

614 THE FINE STRUCTURE OF BRUNNER'S GLANDS IN THE RABBIT. *(E.)* Leeson, C. R.
Dept. Anat., U. Missouri, Columbia) and T. S. Leeson. *Anat
ec* 159(4):409-420, 1967.

615 THE LIVER OF GUNN RATS: OBSERVATIONS BY ELECTRON MICROSCOPY. *(E.)* Picardi, R.
Dept. Electron Microscopy, U. Lausanne, Switzerland), D.
ardiol and P. Magnenat. *Path Europ* 3(1):49-61, 1968.

7616 THE NEURONS OF THE GALL BLADDER AND GUT. *(E.)* Sutherland, S. D. (U. Manchester, England). *J Anat* 101(4):701-709, 1968.

7617 FINE STRUCTURE OF CRYSTALLINE INCLUSIONS IN THE GLOBULE LEUCOCYTE OF THE MOUSE INTESTINE. *(E.)* Carr, K. E. (Dept. Anat., U. Glasgow, Scotland). *J Anat* 101(4):793-803, 1967.

7618 ULTRASTRUCTURAL STUDIES ON DIFFERENTIATION, BOTH NORMAL AND IN CULTURE, OF THE EMBRYONIC HEPATIC CELL. *(It.)* Franceschini, M. P. (Inst. Normal Human Anat., U. Messina, Italy) and P. Motta. *Boll Soc Ital Biol Sper* 43(20):1315-1317, 1967.

7619 A STUDY OF INORGANIC POLYPHOSPHATES IN THE MITOCHONDRIA OF RAT LIVER. *(Fr.)* Stahl, A. J. C. (Fac. Pharm., U. Strasbourg, France), S. Felter and J. P. Ebel. *Bull Soc Chim Biol* 49(12):1863-1872, 1967.

7620 INTESTINAL DISACCHARIDES. I. MALTASE ACTIVITY OF THE MUCOSA OF THE EMPTY INTESTINE. *(It.)* Montagnani, M. (Inst. Gen. Clin. Med., U. Siena, Italy), A. Antonelli, P. F. Bayeli, P. L. Zampetti, C. Riario-Sforza and E. Marinello. *Settimana Med* 55(19):1165-1169, 1967.

7621 INTESTINAL DISACCHARIDES. II. SACCHARASE ACTIVITY OF THE MUCOSA OF THE EMPTY INTESTINE. *(It.)* Riaro-Sforza, G. (Inst. Biol. Chem., U. Siena, Italy), P. F. Bayeli, A. Antonelli, M. Montagnani and P. L. Zampetti. *Settimana Med* 55(20):1199-1203, 1967.

See also: 7721,8142,8554

7622 **WATER AND SODIUM ABSORPTION IN THE HUMAN INTESTINE.** *(E.)* Love, A. H. G. (U.S. Naval Med. Res. Unit No. 2, Taipei, Formosa), T. G. Mitchell and R. A. Phillips. *J Physiol* 195(1):133-140, 1968.

Net water and sodium absorption and bidirectional fluxes of sodium ion were measured in 5 normal subjects during total intestinal lavage; a steady state was attained after 1 hr by subjects drinking an electrolyte solution (2-6 liter/hr) containing 140 mM sodium, 10 mM potassium, 105 mM chloride, and 35 mM bicarbonate. Under steady-state conditions, a known quantity of radioactive sodium was given orally; 10-min serial samples of effluent stool were then analyzed for volume, electrolyte composition, and radioactive sodium. Increasing rates of perfusion from 16 to 100 ml/min caused an average increase in water absorption from 6.7 to 14.3 ml/min, accompanied by an increase in sodium transport. Relating the water absorption to sodium absorption, it was noted that the fluid absorbed had a mean sodium concentration of 138.8 mEq/liter, which was almost identical to the mean plasma sodium concentration in this group (139.8 mEq/liter). The intestinal volume increased with increasing perfusion rates to reach a plateau of 1900 ml at rates of 70-100 ml/min. Transit time diminished with increasing perfusion rate, and showed a reciprocal linear relation to perfusion rate at rates producing a constant intestinal volume. When the water absorption (ml/min) and sodium transport (mEq/min) were expressed as a function of a standard intestinal volume, the absorptive capacity of the intestine was shown not to change with changing perfusion rates. None of the differences in ml/min/liter or mEq/min/liter were significant between any of the values at any flow rate. The increase in absorption capacity with increasing flow rates does not seem to reflect a true increase in absorption per unit of intestine but rather a progression to more nearly continuous flow conditions.

7623 **EFFECT OF ANTIDIURETIC HORMONE ON HUMAN SMALL INTESTINAL WATER AND SOLUTE TRANSPORT.** *(E.)* Soergel, K. H. (Milwaukee County Gen. Hosp., Wis.), G. E. Whalen, J. A. Harris and J. E. Geenen. *J Clin Invest* 47(4):1071-1082, 1968.

The effect of i.v. vasopressin (1 U/hr) on the permeability characteristics and absorptive capacity of the small intestine was investigated in adult males, 21-64 yr old, using the technique of continuous intestinal perfusion with polyethylene glycol 4000 as the nonabsorbable marker. Unidirectional flux rates of Na and H_2O were calculated from the disappearance of ^{22}Na and ^{3}HOH from isotonic saline within the intestinal lumen. Water and sodium absorption from isotonic saline in the jejunum occurred while the subjects were hydrated but was abolished during vasopressin infusion and by dehydration. Net gain of water and sodium in the lumen was evident in some instances. Vasopressin and dehydration caused a decrease in water and sodium absorption in the ileum, while unidirectional flux into the intestinal lumen of water and sodium remained unchanged by vasopressin. During perfusions with hypertonic urea solutions the rates of sodium and water entry into the intestine were greatly increased during i.v. vasopressin infusion, whereas urea loss from the study segment remained constant. Vasopressin in the dosage used did not affect intestinal motility. These results suggest that circulating vasopressin in physiologic concentrations affects the small intestine either by increasing the secretion of water and salt into the lumen or by direct interference with the active sodium transport mechanism.

7624 **THE MECHANISMS OF SODIUM ABSORPTION IN THE HUMAN SMALL INTESTINE.** *(E.)* Fordtran, J. S. (U. Texas Southwest. Med. Sch., Dallas), F. C. Rector Jr. and N. W. Carter. *J Clin Invest* 47(4):884-900, 1968.

The mechanism of sodium transport in the jejunum and ileum of normal male and female subjects, ranging in age from 21 to 65 yr, was characterized with respect to bulk water flow, sodium concentration gradients, and the effects of the addition of glucose and galactose or variations in anionic composition of the luminal fluid. Sodium absorption in the jejunum and ileum differed markedly. When net water movement across the intestinal wall was zero, sodium was secreted in the jejunum along a chemical concentration gradient, whereas in the ileum sodium was absorbed against its concentration gradient. Bulk movement of water had a dramatic effect on sodium transport in the jejunum but not in the ileum. Sodium absorption was more or less constant in the ileum regardless of whether water was absorbed or secreted into the intestinal lumen. In the jejunum the movement of water into the intestinal lumen enhanced sodium secretion, whereas absorption of water diminished sodium secretion and eventually promoted net sodium absorption against a concentration gradient. Sodium as the chloride salt can be absorbed against a gradient of only 13 mEq/liter in the human jejunum, assuming the plasma sodium concentration to be 140 mEq/liter. Furthermore, sodium as the chloride salt can be absorbed from the jejunum in the absence of water flow, glucose, or bicarbonate, but only when the luminal concentration is nearly equal to that of the plasma. Ileal sodium absorption does not cease until the luminal concentration is less than 30 mEq/liter. Thus, the sodium absorption mechanism exhibits some evidence of saturation kinetics. Studies of the effect of the mean glucose concentration in the ileum and jejunum and of glucose absorption rate on sodium movement demonstrated that in the jejunum, glucose absorption has a markedly stimulatory effect on sodium absorption, whereas in the ileum the glucose concentration and the rate of glucose absorption have no significant effect on sodium absorption. Galactose stimulates jejunal sodium absorption at any given rate of water movement, while in the ileum galactose has no significant effect on sodium transport. Studies of the effects of the anion composition of the luminal fluid demonstrated that the addition of bicarbonate to the jejunal contents has much the same effect on sodium absorption as glucose (i.e. both enhance net sodium absorption independent of any effect on overall net water movement). It is concluded that a small fraction of jejunal sodium absorption is mediated by active transport, coupled either to active absorption of bicarbonate or secretion of hydrogen ions, while the major part of sodium absorption (NaCl absorption) is mediated by a process of bulk flow of solution along osmotic pressure gradients.

625 ABSORPTION OF L-METHIONINE FROM THE HUMAN SMALL INTESTINE. *(E.)*

chedl, H. P. (State U. Iowa Coll. Med., Iowa City), C. E.
ierce, A. Rider and J. A. Clifton. *J Clin Invest* 47(2):417-
25, 1968.

bsorption of L-methionine was measured at all levels of small
ntestine in 4 normal subjects and 2 patients with adult celiac
isease, using transintestinal intubation and steady-state per-
usion. The 4 normal subjects (3 males and 1 female) ranged
age from 37 to 62 yr, while the 2 patients (1 male and 1
emale) were 60 and 62 yr old. In the normal subjects, methi-
nine absorption proceeded at a higher rate in the proximal
han in the distal small intestine, especially at higher methionine
oncentrations. In contrast, patients with nontropical sprue
ad a zone of low absorption proximally, where mucosal dam-
ge was presumably greatest, and a zone of higher absorption
istally. In all subjects, the absorption rate in relation to intra-
uminal concentration showed rate-limiting kinetics in both the
roximal and distal small intestine. The K_m and V_{max} data
howed marked differences between the proximal and distal
mall intestine. In normal subjects, the proximal K_m values
anged from 12 to 28 and the distal from 2.0 to 5.7, depend-
ng on concentration range used for calculation. These con-
rasting K_m values suggest differences in transport behavior of
nethionine consistent with differences in carrier function.

7626 THE ROLE OF THE SMALL INTESTINE IN THE DISPOSAL OF ENZYMES FROM BLOOD.

E.) Fleisher, G. A. (Mayo Clin., Rochester, Minn.) and K. G.
Wakim. *Enzym Biol Clin* 9(2):81-96, 1968.

Highly purified preparations of glutamic pyruvic transaminase (GPT) and 2 glutamic oxaloacetic transaminases (GOT I and GOT II) isolated from dog liver and skeletal muscle were injected i.v. into 6 mongrel dogs (10-12 kg) with Thiry-Vella duodenal or ileal fistulas in order to investigate the role of the small intestine in the elimination of plasma enzymes. The enzymes were given as isotonic solutions containing 60,000-90,000 μM/hr/ml of enzymatic activity. Lactic dehydrogenase (LDH) activity was also measured. Although the rate of enzyme transfer varied greatly, depending on whether isotonic or hypertonic solutions (5% or 50% glucose) were used to rinse the loops, and although the plasma-disappearance half-lives of the 3 enzymes varied from 2.6 days to less than 1 hr, there was relatively little difference in their transfer rates and no correlation between intestinal fluid and serum enzyme activities could be established. Although the clearance rates increased slightly from GPT to GOT I to GOT II, the series was too small to establish the significance of the differences. Sterilization of the intestinal loops with neomycin and tetracyclin did not alter the findings, and administration of bethanechol affected neither the endogenous activities nor the transfer of intravenously injected GOT I. Zymosan, an inhibitor of the metabolic clearance of enzymes, also did not affect intestinal enzyme clearance. A slower but significant transfer of enzymes was noted in the direction from intestine to blood. Nonspecific transudation of enzymes across the intestinal mucosa seems to contribute very little to their metabolic clearance, in contrast to the clearance of serum proteins, and the main source of enzymes in the intestinal fluid seems to be the intestinal mucosal cells.

7627 PASSIVE MOVEMENT OF WATER AND SODIUM ACROSS THE HUMAN SMALL INTESTINAL MUCOSA. *(E.)*

Soergel, K. H. (Marquette U. Sch. Med., Milwaukee, Wis.), G. E. Whalen and J. A. Harris. *J Appl Physiol* 24(1):40-48, 1968.

Healthy male volunteers, 21-65 yr of age, were subjected to jejunal and ileal perfusion with isotonic, hypotonic and hypertonic solutions in order to study the permeability of the intact small intestinal mucosa to water, Na, urea and mannitol. The isotonic test solution (290 ± 2 milliosmoles/kg) contained 140 mM NaCl, 16.7 mM dextrose, 10 g/liter polyethylene glycol, 5 μC/liter ^{22}NaCl, and 100 μC/liter ^{3}HOH; hypotonic solutions contained no dextrose and the NaCl concentrations were decreased to 40, 20, or 0 mM; hypertonic solutions contained 140 mM NaCl and 10 g/liter polyethylene glycol to which D-mannitol or urea was added to a final osmolality of 800 milliosmols/kg. Isotonic perfusion studies in 17 subjects showed that more water was absorbed per cm serosal length in the jejunum than in the ileum. When dextrose was replaced by D-mannitol, identical absorption rates were obtained, showing Na and water absorption to be independent of the presence of dextrose. Unidirectional water flux per unit of mucosal surface area was slightly lower in the jejunum, and Na absorption per unit mucosal surface area was lower in the ileum, while unidirectional Na flux was the same in both areas. In hypertonic perfusion studies, the fall in osmolality of intestinal contents was more rapid in the jejunum than in the ileum. The mannitol absorption rate did not differ significantly from zero. The Na concentration of the perfusion solution decreased from 140 mEq/liter at the perfusion tip to 95-111 mEq/liter at the midpoint of the study segment, while a net gain of water and Na occurred. A slightly higher increase was observed in Na concentration during urea infusion than during mannitol infusion, it may be that urea facilitated or mannitol interfered with Na entry into the intestinal lumen. Hypotonic test solutions showed a rapid rise in osmolality; mean osmolalities were similar in jejunum and ileum, as were net and unidirectional flux rates of Na and water, when expressed per unit of mucosal surface area. Mean Na and K concentrations were nearly identical in both segments. Na entry into the lumen occurred due to existing osmotic and concentration gradients, but it is possible that active Na absorption proceeded in the opposite direction simultaneously. The human intestinal mucosa seems to behave as a serial membrane where resistance to filtration flow is provided by the luminal cell wall, penetrated by pores of 2.9-8 Å in radius, while diffusion is restricted by one or more additional barriers which do not restrict filtration flow.

7628 LOCALIZATION OF BILE SALT ABSORPTION *IN VIVO* IN THE RABBIT. *(E.)*

Buchwald, H. (U. Minnesota Med. Sch., Minneapolis) and R. L. Gebhard. *Ann Surg* 167(2):191-198, 1968.

The absorption of force-fed sodium cholate-4-^{14}C (0.2 μC/mg) was determined in rabbits 10-14 days after surgical bypass of either the upper (jejunal) or lower (ileal) half of the small intestine, or the lower half of the small intestine, cecum, appendix and most of the colon (ileocecal subtotal colic bypass), combined with creation of a total biliary fistula. Bile radioactivity was assumed to represent absorbed radioactivity since the total amount of cholate absorbed from the intestinal tract should be rapidly cleared from the circulation by the liver and excreted via the bile. The mean 48-hr cumulative absorption (measured at 2, 4, 6, 8, 10, 12, 24 and 48 hr after feeding the bile salt), expressed as the percentage of test dose radioactivity recovered in the bile, was 77.8% for the control animals with a biliary fistula and an undisturbed intestine and 73.5%, 69.5% and 39.3% for rabbits with a jejunal, ileal and ileocolic bypass, resp. The control group showed a peak absorption increment at 2 hr, while the peaks in the other 3 groups occurred at 4 hr. These findings are contrary to the prevailing concept that bile salts are absorbed exclusively in the ileum.

7629 **A RADIOAUTOGRAPHIC COMPARISON OF GLYCEROL-H^3 AND GALACTOSE-H^3 UPTAKE DURING INTESTINAL GLYCERIDE SYNTHESIS.** *(E.)* Jersild, R. A. Jr. (Indiana U. Med. Ctr., Indianapolis). *Anat Rec* 160(2):217-230, 1968.

The *in vivo* incorporation of glycerol-2-^{3}H (153 mC/mM) and galactose-1-^{3}H (500 mC/mM) into intestinal absorptive cells during simultaneous fat absorption (fatty chyme obtained from the jejunum of rats fed 75% pure linoleic acid) was studied in ligated jejunal segments of Wistar rats. Radioautographs of the intestinal absorptive cells exposed to glycerol-^{3}H and fatty chyme revealed rapid (in 5 min) incorporation of glycerol into the apical endoplasmic reticulum and into cytoplasmic areas containing small droplets of fat. After 20 min, labeled fat droplets accumulated in dilated cisternae of the Golgi apparatus and in lateral intercellular spaces. A relatively small number of grains were present over microvilli, mitochondria, nuclei and areas of cytoplasm in which fat could not be detected. With regard to the incorporation and distribution of glycerol-^{3}H, these findings are very similar to those following glucose-^{3}H uptake. Absorptive cells from intestinal segments injected with labeled glycerol, but not fatty chyme, showed little incorporation compared with cells absorbing glycerol and fat simultaneously. Glycerol labeling was also considerably reduced when the fat was extracted prior to radioautography. Five minutes after absorption of galactose-^{3}H (whether or not fatty chyme was simultaneously present), the label was concentrated primarily in the Golgi region of the absorptive cells; at 20 min, radioactivity was also scattered throughout the apical cytoplasm. The pattern of galactose labeling was not affected by lipid extraction procedures. These observations indicate that there is significant incorporation of glycerol, but not galactose, into newly synthesized glycerides in the upper jejunum.

7630 **RELATION BETWEEN SODIUM CONCENTRATION, ELECTRICAL POTENTIAL AND TRANSFER CAPACITY OF RAT SMALL INTESTINE.** *(E.)* Barry, R. J. C. (Dept. Physiol., U. Sheffield, England), J. Eggenton, D. H. Smyth and E. M. Wright. *J Physiol* 192(3): 647-655, 1967.

The effect of replacement of sodium in the incubation medium on the hexose-dependent potential and the capacity of rat intestine to transfer fluid and glucose was studied in everted sacs prepared from the mid-portion of the combined jejunum and ileum. Replacement of NaCl with mannitol or Tris HCl had little effect on the hexose-dependent potential, while replacement with LiCl or KCl had an inhibitory effect on the potential. Replacement with KCl, LiCl, mannitol, and Tris HCl all reduced the capacity to transfer fluid and galactose, but the effects of Li and K replacement were greater than those of mannitol or Tris replacement. It is concluded that there is no fixed relation between the magnitude of the potential and the amount of galactose transferred.

7631 **EFFECT OF DIETARY CALCIUM, BUFFERING CAPACITY, LACTOSE AND EDTA ON pH OF AND CALCIUM ABSORPTION FROM GASTROINTESTINAL SEGMENTS OF THE GROWING RAT.** *(E.)* Ali, R. (Dept. Animal Sci., Rutgers U., New Brunswick, N. J.) and J. L. Evans. *J Nutr* 93(3):273-279, 1967.

The effects of dietary calcium, buffering capacity (BC), lactose and ethylenediaminetetraacetic acid (EDTA) on the pH pattern of and calcium absorption from 6 segments of the gastrointestinal tract were studied in hooded Norway rats (70-90 g). A total of 144 rats was used for the replicated, factorially arranged experiment which involved calcium (0.19%, 0.48%, and 0.78%), BC (0.46, 0.54, and 0.76 ml N HCl, required to reduce the pH of 5.0 g of the diet by 1 pH unit), lactose (0% and 12%), and EDTA (0% and 0.2%). Chromic oxide served as the reference substance for calcium absorption. The increased calcium absorption with decreased calcium intake was a result of increased hydrogen ion concentration in the intestinal secretion, decreased endogenous secretion of calcium in the duodenum which increased dietary calcium absorption in the lower gastrointestinal tract, and increased calcium absorption all along the intestinal tract distal to the duodenum. The BC controlled the pH of the gastrointestinal contents and lactose produced a more acid pH in the ileum. The percentage calcium absorption increased with an increase in intestinal acidity and decreased with dietary increases in calcium, BC or EDTA. Dietary calcium and BC interacted and influenced calcium secretion in the duodenum and calcium absorption in the jejunum and ileum.

7632 **THE INFLUENCE OF THE VITAMIN D AND PHOSPHATE CONTENT OF THE DIET ON CALCIUM AND STRONTIUM ABSORPTION FROM THE GASTROINTESTINAL TRACT.** *(E.)* Kostial, K. (Inst. Med. Res., Yugoslav Acad. Sci. Arts, Zagreb), T. Maljkovic, N. Gruden and A. Durakovic. *Arh Hig Rada* 19(1):5-9, 1965.

The effect of varying amounts of dietary phosphates and vitamin D on calcium and strontium absorption was studied in female albino rats aged 11-12 weeks. The animals were kept on an experimental diet for 6 days, and on the 3rd and 4th day they received radioactive calcium (^{45}Ca) and strontium (^{85}Sr) in the drinking water. Skeletal retention of radioactive calcium and strontium was determined, after sacrifice 2 days later. Increasing the dietary phosphates from 0.5 to 1.2% caused a reduction in the retention of radioactive calcium and strontium after oral administration of these isotopes. The effect of phosphates was much more pronounced on strontium than on calcium absorption. The effect of phosphates on calcium and strontium metabolism was independent of the vitamin D content in diet as long as this content ranged from 200 to 800 IU per 100 g of dry food.

7633 **ABSORPTION AND SECRETION OF WATER AND ELECTROLYTES BY THE INTACT COLON IN A PATIENT WITH PRIMARY ALDOSTERONISM.** *(E.)* Shields, R. (Royal Infirm., Cardiff, Wales), J. B. Miles and C. Gilbertson. *Brit Med J* 1(5584):93-96, 1968.

The net transport and unidirectional fluxes of sodium, potassium nd water through the intact colon were studied in a 58-yr-old male with primary hyperaldosteronism 1 week before and 3 months after removal of an adrenocortical tumor, as well as n 8 male controls (20-65 yr old). All subjects were fasted for 2 hr before colonic perfusion at 25 ml/min with modified Tyrode's solution containing ^{24}Na (2 μC/liter), ^{42}K (4 μC/iter), deuterium oxide (1% v/v) and a nonabsorbable marker 1% w/v of polyethylene glycol of molecular weight 4000). Net transport rates were calculated according to the formulae of Levitan et al. (1962); the rates of sodium, potassium and water unidirectional movement were calculated according to he formulae of Shields (1966). Before removal of the tumor he experimental colon secreted 4-5 times as much potassium s the control colons and the unidirectional potassium flux nto the colonic lumen as well as the daily potassium fecal loss were increased; however, potassium transport rates returned to within the control range after removal of the tumor. Sodium nd water absorption rates were within the control range before nd after surgery.

7634 RATE-LIMITING STEPS IN STEADY-STATE INTESTINAL ABSORPTION OF TRIOCTANOIN-1-^{14}C. EFFECT OF BILIARY AND PANCREATIC FLOW DIVERSION.

(E.) Clark, S. B. (St. Luke's Hosp. Ctr., New York, N. Y.) and P. R. Holt. *J Clin Invest* 47(1):612-623, 1968.

Experiments on male Wistar rats with indwelling duodenal cannulae showed that a steady state of intestinal absorption is achieved during continuous intraduodenal infusion of emulsified fat. Maximal steady-state absorption of trioctanoin, a medium-chain triglyceride (MCT), by unanesthetized, restrained rats was found to be the same after total bile diversion as in controls (1560 μmoles fatty acid/hr. After pancreatic and bile diversion, absorption of MCT was still 1/3 as rapid as in controls, and mucosal uptake apparently occurred in the form of unhydrolyzed triglyceride. Returning bile to the lumen during pancreatic diversion did not increase the absorption rate. From intestinal tissue lipid-^{14}C concentrations measured during steady-state maximal absorption, it was possible to calculate turnover times for labeled lipid passing through the mucosal cells. Mucosal turnover times of 4 min for control and bile-diverted rats and 20 min for animals with pancreatic diversion were noted. The rate-limiting step in octanoic acid absorption in control and bile-diverted rats was thought to be mucosal penetration. During absorption of unhydrolyzed triglyceride by pancreatic flow-diverted rats, both passage from the lumen into the mucosal cell and intracellular lipolysis were rate-controlling factors.

7635 PATHWAYS OF ABSORPTION OF RETINAL AND RETINOIC ACID IN THE RAT.

(E.) Fidge, N. H. (Columbia U. Coll. Phys. Surg., New York, N. Y.), T. Shiratori, J. Ganguly and D. S. Goodman. *J Lipid Res* 9(1):103-109, 1968.

The chemical and anatomical pathways of absorption of dietary etinal, retinoic acid, and retinol were examined in rats bearing ymph, bile, and duodenal cannulae. In each rat, an uninterupted flow of bile into the duodenum was maintained by connecting the duodenal cannula to the bile duct of a second rat. Labeled vitamin A compounds were introduced into the duodenum in very small amounts (7-14 μg) in the form of a bile-lipid mixture resembling normal intestinal contents. Under these conditions, most (70-80%) of the radioactivity recovered after the feeding of labeled retinol or retinal was found in the lymph, predominantly in saturated retinyl esters. In contrast, 92-95% of the radioactivity recovered after the feeding of labeled retinoic acid was found in the bile, and was contained in a mixture of polar metabolites, most being more polar than free retinoic acid. These results indicate that under normal conditions the major pathway of retinal absorption involves its reduction to retinol, which is then esterified and transported via the lymphatics in a manner similar to that of dietary retinol. A small proportion of retinal is apparently normally oxidized and is then transported via the portal vein and excreted in the bile in a manner similar to that of dietary retinoic acid. The relative importance of these 2 pathways of retinal metabolism can vary, depending upon the status of the animal.

7636 THE GRADIENT OF ELECTRICAL POTENTIAL DIFFERENCE AND OF SODIUM AND POTASSIUM OF THE GUT CONTENTS ALONG THE CAECUM AND COLON OF NORMAL AND SODIUM-DEPLETED RATS.

(E.) Edmonds, C. J. (U. Coll. Hosp. Med. Sch., London, England). *J Physiol* 193(3):571-588, 1967.

The Na, K, and water content of stools and of gut contents removed from the terminal ileum, cecum, and colon were determined in normal and Na-depleted rats and the potential difference across the colon wall was measured at the site of removal of each specimen. During passage through the cecum and colon, the fecal Na and water content fell considerably, K content being unchanged in the normal rats and falling in the Na-depleted rats. The Na concentration of the fecal water fell but K concentration increased due to water absorption. Feeding normal rats with a sulfonated polystyrene resin caused a considerable Na loss in the stool, the ratio Na/(Na + K) being consistently greater than in rats that did not receive the resin; the resin induced little Na loss but much K loss in Na-depleted rats. The electrical potential difference across the colon wall varied little over the length of the cecum and colon in normal rats (not greater than 20 mvolts), the serosa being positive with respect to the lumen. Potential difference measurements were greater in Na-depleted rats, and those in the cecum and descending colon were consistently higher than those in the ascending colon. There was a similar pattern in the resin-fed rats, but potentials tended to be higher. The K concentration of the gut contents was always greater than could be accounted for if K were passively distributed across the colonic mucosa. It is concluded that 1) active Na absorption is stimulated by Na depletion, 2) K is probably actively transported into the colonic lumen, and when unabsorbable anions are present in the gut, K secretion is critically important in Na absorption, and 3) the elevation of potential difference associated with Na depletion is probably associated with the stimulated Na transport.

7637 TRANSPORT OF SODIUM AND SECRETION OF POTASSIUM AND BICARBONATE BY THE COLON OF NORMAL AND SODIUM-DEPLETED RATS.

(E.) Edmonds, C. J. (U. Coll. Hosp. Med. Sch., London, England). *J Physiol* 193(3):589-602, 1967.

Ascending and descending colonic segments of normal and Na-depleted rats were perfused *in vivo* with isotonic solutions of varying Na concentration and the unidirectional Na fluxes and secretion rates of K and bicarbonate as well as the transmucosal electrical potential differences were measured. Potential differences were greater in the Na-depleted rats, especially towards the distal end of the descending colon. With a reduction of luminal Na concentration, the potential difference was reduced. The ascending and descending segments were similar in regard to Na transport except that the latter had a lower passive permeability. Na depletion caused an increase in the Na influx rate, Na net flux rate, and Na exchange diffusion while the mucosal passive Na permeability decreased. These changes resulted in a reduction in the critical luminal Na concentration (the concentration at which the unidirectional fluxes were equal). K secretion was similar in the ascending and descending colon and was increased by Na-depletion. In all rats, it was reduced when the luminal Na concentration was low. Bicarbonate secretion was unaffected by Na-depletion, and all solutions remained isotonic during perfusion. The results confirmed that active Na transport is stimulated by Na-depletion but indicated that this is probably not the only factor in the elevation of the transmucosal potential difference.

7638 TRANSPORT OF POTASSIUM BY THE COLON OF NORMAL AND SODIUM-DEPLETED RATS.

(E.) Edmonds, C. J. (U. Coll. Hosp. Med. Sch., London, England). *J Physiol* 193(3):603-617, 1967.

Ascending and descending segments of colon or normal and Na-depleted rats were perfused with solutions of varying KCl concentration. Net K flux, electrical potential difference, and unidirectional K fluxes were measured. It was noted that variation of luminal K concentration over the range 0-40 mM had no effect on potential difference or K efflux rate. K secretion fell 30% when Na-free choline chloride was used as the perfusing medium. Net flux was found to be a linear function of the luminal K concentration, and fell as the latter increased. Na depletion increased K secretion rate and passive permeability of the mucosa to K; adrenalectomy had the reverse effect. The luminal K concentration associated with zero net K flux was much greater than expected if the colonic mucosa behaved passively with respect to K. Unidirectional fluxes, determined with 5 mM KCl in the lumen, revealed that the influx/efflux ratio was much less than predicted by Ussing's flux ratio equation. It is concluded that K influx is due to simple diffusion and K efflux to diffusion and active transport, both processes being increased by Na depletion.

7639 ACTINOMYCIN D EFFECT ON AMINO ACID ABSORPTION FROM RAT JEJUNAL LOOPS.

(E.) Yamada, C. (Sch. Publ. Health, U. California, Los Angeles), A. J. Clark and M. E. Swendseid. *Science* 158(3797): 129-130, 1967.

The absorption of amino acids from jejunal loops was suppressed in anesthetized rats pretreated with 1.0 or 1.5 μg of actinomycin D/g of body weight. The absorption of the acidic, neutral, and basic amino acids showed selective degrees of response that were related to the degree of inhibition and to the time interval required to demonstrate actinomycin sensitivity. The specific effect of actinomycin D on the absorption of the various groups of amino acids is some indication that the antibiotic does not act by producing a generalized toxic condition in the intestinal tissue, which interferes with all absorption processes. In further experiments measuring the absorption of D-glucose, it was noted that actinomycin D, given 4 hr before the test, did not inhibit and may even have enhanced the absorption of glucose.

7640 ABSORPTION AND METABOLISM OF DIETARY TRIGLYCERIDES IN GERMFREE AND CONVENTIONAL RATS.

(E.) Wiech, N. L. (Tulane U. Sch. Med., New Orleans, La.), J. G. Hamilton and O. N. Miller. *J Nutr* 93(3):324-330, 1967.

The effect of the absence of an intestinal microflora on the absorption and metabolism of triglycerides derived from dietary corn oil was studied using germfree and conventional male rats (Fisher strain). Both types of animals consumed a semipurified diet within a 1.5-hr period daily. The fasting plasma triglycerides were significantly elevated in the germfree rats. In addition, a higher percentage of linoleic acid was found in the germfree rats when the fatty acid composition of the fasting plasma triglycerides was analyzed. No apparent difference was demonstrated in the rate of absorption of the dietary triglycerid in the germfree rats, but postprandial plasma triglyceride analysis indicated a faster rate of clearance of the absorbed triglyceride by the germfree animals. These animals also exhibited elevated fasting plasma glucose levels and cleared glucose from the plasma more slowly than conventional rats. There were also indications that germfree rats had a decreased and delayed insulin secretion.

7641 TRANSMURAL MOVEMENTS OF ZINC, MANGANESE, CADMIUM AND MERCURY BY RAT SMALL INTESTINE.

(E.) Sahagian, B. M. (Washington U. Sch. Med., St. Louis, Mo.), I. Harding-Barlow and H. M. Perry Jr. *J Nutr* 93(3):291-300, 1967.

Characteristic uptake and transport patterns of Zn, Mn, Cd and Hg in segments of small intestine from male and female Wistar rats, weighing 250-350 g, were studied using a newly developed *in vitro* perfusion method adapted for the use of the radioisotopes of these metals. Individual solutions containing Mn, Cd, Hg or Zn ions (10^{-4} M) were introduced to the mucosal side of the intestinal segment and the hourly rate of accumulation of each metal in the solution bathing the serosal side was measured. Only small amounts of Cd, Hg, and Zn were transported during the first hour of perfusion, but Mn was transported rapidly. The rates of transmural passage of these metals increased in the following order: Mn, Cd, Hg and Zn. The tissue metal uptake or the amounts of metal taken up or retained by the same segment of intestine perfused for the same length of time increased in the order Zn, Hg, Cd and Mn. Studies of the effects of pharmacologic agents on the uptake and transport of these metals demonstrated that Mn transport was enhanced in the presence of EDTA and enhanced slightly less by dehydroascorbate and iodoacetate. Zn transport was enhanced in the presence of EDTA, dehydroascorbate, phlorhizin and ascorbate, whereas iodoacetate depressed Zn transport. Cd transport was increased to a greater extent by ascorbate than by dehydroascorbate or by EDTA, while phlorhizin slightly depressed it and iodoacetate had no effect on Cd transport. Hg transport was depressed by dehydroascorbate, iodoacetate and markedly so by EDTA. Studies of the competitive effect of one metal on the transport of another demonstrated that Mn transport is enhanced greatly by Cd and depressed by g, Cd transport is enhanced by Zn and Hg, Hg transport enhanced by Cd, and Zn transport is enhanced by Cd.

642 **EFFECT OF RATION UPON THE INTESTINAL DISTRIBUTION OF Ca, Mg, Na, K AND N IN ALVES.** *(E.)* Perry, S. C. (Agric. Res. Lab., U. Tennessee, ak Ridge), R. G. Cragle and J. K. Miller. *J Nutr* 93(3):283-90, 1967.

he effect of extremely different diets on the electrolyte disribution in the intestinal contents was studied in 11 Holstein alves. The calves were divided into 3 groups: group A (4 alves) received a semipurified ration; group B (4 calves) reeived a concentrate ration consisting of 76.5% ground corn, % alfalfa meal, 12% cottonseed meal, 5% molasses and 0.5% ach of bone meal, dicalcium phosphate and trace mineralized alt; and group C (3 calves) received a ration containing the elements of the concentrate mixture and alfalfa hay. A net ecretion of Ca, Mg, Na, K and N occurred in the upper portion of the small intestine and the absorption was greatest in passge through the small intestine. Ca, Mg and N showed only negligible changes in their intestinal concentration during passge through the cecum and large intestine. Minimal values of Na and K secretion and absorption were calculated for the ower intestines. Differences attributed to the various effects of the different diets were seen in the ruminal concentrations of Mg and K, especially in the animals who received the semipurified ration. The diet had very little if any effect on the concentration differences in the small intestine.

7643 **DETERMINATION OF $^{14}CO_2$ IN THE EXPIRED AIR FOR COMPARING THE UTILIZATION OF LONG-, MEDIUM-, AND SHORT-CHAIN FATTY ACIDS.** *(Fr.)* Metais, P. (Fac. Med., Strasbourg, France), A. Bach and J. Warter. *C R Soc Biol (Paris)* 161(1):1372-1376, 1967.

Using a stomach tube, fasting rats were given 0.12 ml/100 g body weight of olive oil containing 0.5 μC of ^{14}C-labeled palmitic (C_{16}), lauric (C_{12}), or caprylic (C_8) acids or their triglycerides. All of these compounds had ^{14}C-labeled carboxyl groups. Curves for the cumulative percentage of $^{14}CO_2$ exhaled as a function of time showed that utilization of these fats and fatty acids increased with decreasing chain length. The rate at which $^{14}CO_2$ appeared in the expired air also increased with decreasing chain length. Free fatty acids were absorbed and utilized more rapidly than their corresponding triglycerides.

7644 **THE EFFECT OF BILE ON VITAMIN A ABSORPTION IN THE RAT.** *(E.)* Gagnon, M. Royal Free Hosp., London, England) and A. M. Dawson. *Proc Soc Exp Biol Med* 127(1):99-102, 1968.

Two groups of 11 Sprague-Dawley rats, both fitted with thoracic duct fistulas and one also fitted with bile fistulas, were fed vitamin A-^{14}C in corn oil. A poor total recovery of vitamin from the animal tissue and gastrointestinal contents of both groups of animals was noted, being less in the bile fistula rats where less vitamin A was absorbed; this was attributed to the degradation of lipid-soluble vitamin A into water-soluble meabolites by bacteria in the large intestine. In the control aninals, more radioactivity was recovered from the intestinal lymph than from the liver and other tissues, suggesting that most of the absorbed vitamin A was transported by the lymphatic route. In the absence of bile, however, when the absorption of vitamin A was grossly impaired, there was a tendency for more to be recovered from the liver than the lymph, suggesting that an alternate route of transport of the absorbed vitamin was used.

7645 **IRON ABSORPTION IN MAN.** *(E.)* Kuhn, I. N. (Dept. Med., U. Washington, Seattle), E. R. Monsen, J. D. Cook and C. A. Finch. *J Lab Clin Med* 71(5): 715-721, 1968.

7646 **ROLE OF VITAMIN E IN IRON DEFICIENCY ANEMIA.** *(E.)* Fujii, T. (Saiseikai Utsunomiya Hosp., Tokyo, Japan), Y. Matsuki and M. Hasegawa. *Keio J Med* 17(3):207-216, 1968.

7647 **INFLUENCE OF FRUCTOSE ON IRON ABSORPTION FROM THE DIGESTIVE SYSTEM OF HEALTHY SUBJECTS.** *(E.)* Brodan, V. (Inst. Human Nutrit., Prague, Czechoslovakia), M. Brodanová, E. Kuhn, V. Kordač and J. Válek. *Nutr Dieta (Basel)* 9(4):263-270, 1967.

7648 ***IN VIVO* METHOD FOR THE STUDIES ON PENETRATION PHENOMENA THROUGH INTESTINAL MEMBRANE.** *(E.)* Nakano, S. (Jikei U. Sch. Med., Japan), T. Sato and K. Fujii. *J Physiol Soc Japan* 30(6):415-416, 1968.

7649 **AMINO ACID TRANSPORT: FACTORS INFLUENCING UPTAKE IN KIDNEY AND INTESTINE.** *(E.)* Scriver, C. R. (McGill U.-Montreal Child. Hosp. Res. Inst., P.Q., Canada). *Bibl Paediat* 87:72-92, 1968.

7650 **DIGESTION AND ABSORPTION OF SUGARS.** *(E.)* Semenza, G. (Inst. Biochem., U. Zurich, Switzerland). *Bibl Paediat* 87:32-47, 1968.

7651 **DIGESTION AND ABSORPTION OF FATS.** *(E.)* Borgström, B. (Chem. Ctr., U. Lund, Sweden). *Bibl Paediat* 87:48-55, 1968.

7652 **INTESTINAL HYDROLYSIS OF STARCH.** *(E.)* Auricchio, S. (Inst. Child. Welfare, U. Naples, Italy), F. Ciccimarra, D. Della Pietra and A. Vegnente. *Bibl Paediat* 87:22-31, 1968.

7653 **THE PASSAGE OF EXOGENOUS PEROXIDASE FROM BLOOD CAPILLARIES INTO THE INTESTINAL EPITHELIUM.** *(E.)* Hampton, J. C. (Northwestern U. Med. Sch., Chicago, Ill.) and B. Rosario. *Anat Rec* 159(2): 159-170, 1967.

7654 **PARTITION OF LIPIDS BETWEEN EMULSIFIED OIL AND MICELLAR PHASES OF GLYCERIDE-BILE SALT DISPERSIONS.** *(E.)* Borgström, B. (Dept. Physiol. Chem., U. Lund, Sweden). *J Lipid Res* 8(6):598-608, 1967.

7655 **INTESTINAL ABSORPTION OF Ca^{47} WITH RELATION TO ADMINISTRATION OF PARATHYROID HORMONE EXTRACT IN NEPHROPATHIC SUBJECTS.** *(It.)* Matassi, L. (Inst. Spec. Med. Path., U. Firenze, Italy), G. Buzzelli, R. Sassi, N. Marra and P. Chiarini. *Settimana Med* 55(20):1193-1197, 1967.

See also: 7594,7811,7915,8043,8134,8238,8239,8377

7656 PHARMACOLOGICAL ACTIONS OF 9-[(1N-METHYL-3-PIPERIDYL)METHYL]-THIOXANTHENE HYDROCHLORIDE (METHIXENE) ON THE FUNCTION OF THE GASTROINTESTINAL TRACT. *(Jap.)*

Shiraishi, T. (Tokyo Med. Coll., Japan) and K. Morita. *J Physiol Soc Jap* 30(1):30-36, 1968.

The effects of methixene, a parasympatholytic agent with a marked antitremorin effect, on the motility and secretory activity of the gastrointestinal tract were investigated *in vivo* in mice and Wistar rats. An excellent inhibitory effect on the gastrointestinal propulsion of a charcoal meal was noted in mice, similar to the effects of atropine. The inhibitory effect on the secretion of gastric acid in rats was proven by Schild's method. The potency of methixene was 114.8% of that of atropine. The compound also had an inhibitory effect on the formation of gastric ulcers in Shay rats, with a potency somewhat greater than that of atropine.

7657 POSTOPERATIVE INTESTINAL MOTILITY: AN EXPERIMENTAL STUDY ON DOGS. *(E.)*

Baker, L. W. (Royal Infirm., Aberdeen, Scotland) and D. R. Webster. *Brit J Surg* 55(5):374-378, 1968.

In the first part of a 2-fold study, 9 dogs with Thiry-Vella loops, Roux-en-Y loops with external fistulas, or both were used to confirm the validity of Thiry-Vella pressure recordings as reflections of parent intestinal activity. The introduction of a 2-cm balloon into a tonic but inactive bowel produced a spasm which was released over a 9-min period; inflation of the balloon increased bowel tone and activity, which ceased and did not return following deflation. Simultaneous and individual recordings from Thiry-Vella loops and Roux-en-Y fistulas 2-4 hr after their formation were similar and showed quiescent periods with occasional bursts of segmental activity and changes in tone; similar activity was noted 8 days postoperatively, although quiescent periods were less frequent but often longer. Radiography used in conjunction with pressure studies confirmed the findings. Atropine (0.2 mg i.v.) produced inhibition in both the Thiry-Vella loop and the parent intestine, which was less marked if the intestine contained food. Morphine increased the rate and force of activity initially at both sites, followed by a decrease in the contraction rate. These observations showed that Thiry-Vella loops do reflect parent intestinal activity and can be used to measure intestinal activity after surgery. In the second part of this investigation, intestinal activity was measured in 4 dogs with established Thiry-Vella loops after cholecystectomy, Billroth I gastrectomy, and transthoracic vagotomy. Activity was observed as early as 10 min after surgery and was similar in all respects to that found preoperatively; indicating that early postoperative activity could be normal. These findings on dogs support the observations in humans that normal or near-normal intestinal motility is evident after uncomplicated abdominal surgery.

7658 ELECTRICAL ACTIVITY OF THE GASTRODUODENAL JUNCTION AND ITS RELATION TO THE ELECTRICAL ACTIVITY OF THE ANTRUM AND DUODENUM. *(It.)*

Lanfranchi, G. A. (Inst. Spec. Med. Path., U. Bologna, Italy), L. Barbara and M. Bortolotti. *Rass Fisiopat Clin Ter* 40(1):5-17, 1968.

The electrical activity of the pyloric antrum and duodenum w studied in 5 dogs, each of which had 5 electrodes implanted i the gastric fundus, anterior wall of the antrum, pylorus, duod nal bulb, and the antimesenteric edge of the duodenal wall. Basal activity in the antrum consisted of diphasic, or occasionally triphasic, waves lasting about 3-4 sec followed by a restin stage lasting about 16 sec. The frequency of these waves was more variable than that of duodenal waves and their shape depended on how deeply the electrodes were implanted and how close they were to the pylorus. In the duodenum the basal rhythm consisted of diphasic, or occasionally triphasic, waves with a mean frequency of 17.8/min. This rapid electrical activity was superimposed on slow waves which were almost always followed by contractions of smooth muscle fibers. Basa activity in the pylorus was characterized by the simultaneous presence of both the slow gastric rhythm and the duodenal rh thm. Sometimes one of these components predominated and sometimes the other, depending upon the predominating functional state of the pylorus. This was demonstrated by i.v. administration of serotonin which eliminated the gastric rhythm. Pyloric and duodenal rhythms were then the same except for bursts of spikes which were of greater amplitude and duration in the duodenum. Neostigmine and acetylcholine produced effects similar to that of serotonin. Pyloric activity was almos completely suspended by i.v. administration of epinephrine, morphine, and atropine.

7659 5-HYDROXYTRYPTAMINE PARTICIPATION I THE VAGAL INHIBITORY INNERVATION OF THE STOMACH. *(E.)*

Bülbring, E. (Dept. Pharmacol., U. Oxford, England) and M. D. Gershon. *J Physiol* 192(3):823-846, 1967.

Guinea-pig and mouse stomachs were isolated with the vagus and sympathetic nerves attached and intraluminal pressure was recorded by a balloon technique. The response to vagal stimul tion, which consisted of an excitatory and an inhibitory component, resembled the response to 5-hydroxytryptamine (5-HT which has no direct action on the muscle but acts on intrinsic excitatory and inhibitory ganglia. In the presence of hyoscine, the effects of vagal stimulation, nicotinic compounds and 5-HT were all purely relaxant. Competitive block of ganglionic receptors for acetylcholine reduced the vagal relaxation without antagonizing 5-HT. Specific desensitization of ganglionic recep tors for 5-HT reduced the vagal relaxation without antagonizing nicotinic compounds. During the early phase of the blocking action of nicotine, responses to vagal stimulation and to 5-HT were both abolished. As the nonspecific antagonism changed t the later phase of specific antagonism to acetylcholine, the inhibtory (but not the excitatory) component of the vagal response recovered partially, in parallel with the recovery of the relaxant effect of 5-HT. The vagal inhibitory effect was completely abolished only when competitive block of acetylcholine receptors was combined with desensitization of 5-HT receptors. Stimulation of the mouse stomach in the presence of hyoscine caused the release of 5-HT; this release was blocked by tetrodotoxin. These results, together with previous observations that 5-HT is contained within preganglionic nerve fibers in the myen teric plexus, are consistent with the hypothesis that 5-HT, with tylcholine, may be a neurotransmitter in the vagal inhibitory ervation of the stomach.

0 ELECTROPHYSIOLOGIC AND CINEFLUOROGRAPHIC OBSERVATIONS OF BILE FLOW CHANISM OF MAN. *(E.)* Ono, K. (Hirosaki U. Fac. Med., an), K. Suzuki, H. Tsuchida and M. Abo. *Bull Soc Int Chir* 3):191-196, 1968.

mechanism of bile flow into the duodenum was studied in patients treated surgically for cholelithiasis. Action poten- s from the sphincter of Oddi and the duodenum, detected pairs of electrodes sewn in these respective muscles, and ary radiographs obtained by infusing contrast medium into -tube in the common bile duct were recorded simultaneously film. Bile flow was interrupted at the terminal portion of common bile duct when electrical activity was observed in muscle of Oddi, but bile poured into the duodenum when electrical activity was observed. Electrical activity of the odenal wall muscle was not associated with interruption of flow. These observations indicate that the muscle of Oddi a sphincter action which operates independently of duodenal iscle activity.

61 THE EFFECT OF DIMETHOXANATE ON CONTRACTIONS IN THE ISOLATED GALL-ADDER OF THE GUINEA PIG. *(Fr.)* Guerrin, F. (Fac. ed. Pharm., Lille, France), J. C. Paris and J. Paris. *C R Soc ol (Paris)* 161(1):1322-1323, 1967.

concentrations of 10^{-7}-10^{-4} M, the antitussive agent di- ethoxanate had no detectable effect on pressure within the olated guinea-pig gallbladder or on spontaneous changes in ne at its base. Contractions produced in guinea-pig gall- adders by 10^{-8} M acetylcholine were reduced 75-80% by x 10^{-6} and 10^{-5} M dimethoxanate. Contractions disappeared mpletely when higher concentrations of dimethoxanate were ed. After a lag of 20-40 sec there was a period of rapid re- xation followed by a period of slower relaxation. At a con- ntration of 10^{-5} M dimethoxanate completely eliminated gall- adder contractions produced by 10^{-4} M barium chloride in 12 of 15 cases. This relaxation differed somewhat from that seen after acetylcholine-induced contraction. In many cases it was preceded by a slight increase in pressure with a shorter period of rapid relaxation and a longer period of slow relaxation. These results show that dimethoxanate has an antispasmodic action similar to that of atropine and papaverine.

7662 THE EFFECTS OF METOCLOPRAMIDE ON STOMACH MOTILITY IN THE RAT. *(Fr.)* Guerrin, F. (Fac. Med. Pharm., Lille, France), G. Milbled, P. Merveille and B. Hequet. *C R Soc Biol (Paris)* 161(1):1318-1322, 1967.

Stomach motility was measured by a pressure method in Wistar rats under pentobarbital anesthesia before and after administration of 10 mg/kg of metoclopramide s.c. The amplitude of stomach contractions increased rapidly after administration of metoclopramide, doubling in 15 min and tripling in 30-45 min. The frequency of these contractions remained unchanged. A dose of 5 mg/kg of metoclopramide did not have a consistent effect on gastric motility while doses larger than 10 mg/kg increased mortality because metoclopramide synergizes anesthesia. EEG tracings were made on rats before and after i.v. administration of 10-40 mg/kg of metoclopramide. Results suggest that metoclopramide produces functional changes in structures of the CNS and probably in the reticular formation.

7663 EFFECTS OF ANGIOTENSIN, BRADYKININ AND OXYTOCIN ON ELECTRICAL AND MECHANICAL ACTIVITIES IN THE TAENIA COLI OF THE GUINEA PIG. *(E.)* Ohashi, H. (Dept. Pharmacol., U. Hokkaido, Sapporo Japan), Y. Nonomura and A. Ohga. *Jap J Pharmacol* 17(2):247-257, 1967.

7664 GENERAL PHARMACOLOGY, CHOLERETIC AND SPASMOLYTIC ACTIVITY OF METHYL-4-OMBELLIFERONE. *(Fr.)* Fontaine, L. (Licha-Oberval Res. Ctr., Lyon, France), M. Grand, D. Molho, J. Chabert and E. Boschetti. *Therapie* 23(1):51-62, 1968.

See also: 7679,7894,7981,7986,8051,8662

7665 THE EFFECTS OF HYPOPHYSECTOMY ON THE DIGESTIVE GLANDS OF THE MOUSE.

(E.) Koerker, B. M. (U. Michigan Med. Sch., Ann Arbor). *Amer J Anat* 121(3):571-600, 1967.

The effect of hypophysectomy on the secretory cells of the pancreas, stomach and salivary glands was investigated in adult male ICR Swiss mice 8-10 weeks after the operation. In general, the digestive gland cells which secrete proteins or mucopolysaccharides or are involved in ion transport or release were most severely affected by hypophysectomy. Marked involutional changes occurred in gastric chief and parietal cells; secretory granules were reduced in number or absent, basophilia was diminished and mitochondria were shorter and fewer in the former, while the intracellular canalicular system became greatly contracted in the latter. The gastric mucous cells were greater in number and smaller in size than in controls and contained less secretory material; a colloidal iron-positive variety predominated. In the pancreas, decreases in acinar cell diameter and size, number of secretory granules, amount of cytoplasmic RNA, number and size of mitochondria, reductions in nuclear size and condensation of the Golgi apparatus were observed. Hypophysectomy also induced parotid acinar cell atrophy, similar to but not as severe as that in the pancreas; basophilia was moderately reduced and mitochondria were thinner. Partial involution of the salivary ducts of the parotid and sublingual glands was observed. Submandibular and sublingual mucous acinar cells showed loss of cytoplasmic basophilia and secretory material. Serous cells of the submandibular tubules were markedly involuted with depletion of zymogenic granules and increased numbers of glycogen granules. These observations are discussed in relation to findings for other mammalian species. It is concluded that, as in the rat and man, the protein- and mucopolysaccharide-secreting cells of the mouse digestive system, as well as those involved in ion transport, depend on the pituitary hormones for maintenance of their structure and function.

7666 THE RELATION BETWEEN THE CLEARANCE OF IODIDE AND PERTECHNETATE IN HUMAN PAROTID SALIVA AND SALIVARY FLOW RATE.

(E.) Harden, R. McG. (Western Infirm., Glasgow, Scotland) and W. D. Alexander. *Clin Sci* 33(2):425-431, 1967.

A linear relation between parotid salivary iodide clearance and salivary flow rate was demonstrated in 4 subjects studied at flow rates ranging from 0.03-4.1 ml/min. In 26 subjects, the parotid salivary iodide clearance was studied at 3 flow rates and ranged from 4.1 ± 0.57 ml/min under resting conditions to 14.9 ± 1.43 ml/min during lemon juice stimulation. The clearance of pertechnetate (TcO_4^-) was measured at the same time as the iodide (I^-) clearance. As the flow rate increased so did the TcO_4^- clearance, values ranging from 2.2 ± 0.28 ml/min under resting conditions to 7.1 ± 0.63 ml/min during lemon juice stimulation. The ratio of I^- clearance to TcO_4^- clearance did not vary with flow rate and at all flow rates the TcO_4^- clearance was about half the I^- clearance. These results are consistent with TcO_4^- and I^- sharing a common transport mechanism. In any study of salivary I^- or TcO_4^- clearance, flow rate of saliva must be considered. Variations in the saliva/plasma ratios at resting flow rates are significantly greater than variations in the clearance rate.

7667 COMPARATIVE ANALYSIS OF CELL RENEWAL IN THE GASTROINTESTINAL TRACT OF NEWBORN HAMSTER.

(E.) Lipkin, M. (Bellevue Hosp., New York, N. Y.) and E. Deschner. *Exp Cell Res* 49(1):1-12, 1968.

Comparative rates of cell proliferation in different areas of the gastrointestinal tract of newborn hamsters were estimated by observing the appearance of repetitive waves of ^{3}H-thymidine-labeled mitosis. The proliferation of cells was also studied by constructing theoretical models to simulate the appearance in time of the repetitive waves. In each model, the cycle durations of individual cells or groups of cells in the proliferating population, and the fractional contribution of each group to the total population were known. The contributions to the population elements were summed, to indicate the descriptive characteristics of the repetitive proliferative cycles. The data indicated more rapid proliferation in small intestine than in stomach or colon, and suggested additional similarities and perturbations in the proliferative cycle of cells in these areas.

7668 SALIVARY SECRETION AND AMYLASE DIGESTION IN MAN DURING THE MASTICATION OF PRODUCTS CONTAINING FLOUR.

(Fr.) Dechezlepretre, S. (Fac. Med., Paris, France) and A. Guilbot. *Arch Sci Physiol (Paris)* 34(3):285-297, 1967.

A study was made of the amount of saliva secreted when fresh and freeze-dried bread and toast from which the crusts had been removed were chewed by 2 subjects for 20 sec. The course of starch hydrolysis in the masticated product was then measured at 37C by determining alcohol-soluble sugars in ethano ethanol extracts by the anthrone method. The percentage of saliva found in the dry product after chewing differed considerably in these 2 subjects, was inversely proportional to the dry weight of the toast, and was slightly higher in bread allowed to ferment for longer periods. The baking time had no effect on the quantity of saliva absorbed or secreted. Starch was hydrolyzed more rapidly in fresh bread than in freeze-dried bread or toast. The amylase activity of the saliva in 1 subject was 2 times that in the other and was inversely related to the quantity of saliva secreted. When starch digestion was allowed to proceed for 10 min, it occurred 5 times more rapidly in a dilute medium *in vitro* than *in vivo*. The chain length of oligosaccharides was longer in *in vitro* digestion than in *in vivo* digestion in which only maltotetraose, maltotriose, maltose, and glucose were found, the products depending upon the length of the reaction.

7669 CYTOKINETIC STUDIES ON THE EPITHELIUM OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY AND DIGESTIVE TRACTS OF THE MOUSE. I. CYTOKINETICS OF THE STRATIFIED SQUAMOUS EPITHELIUM.

(Jap.) Ohno, T. (Sapporo Med. Coll., Japan). *Sapporo Med J* 31(4/5):195-201, 1967.

7670 CYTOKINETIC STUDIES ON THE EPITHELIUM OF MUCOUS MEMBRANE OF THE UPPER RESPIRATORY AND DIGESTIVE TRACTS OF THE MOUSE. 2. CYTOKINETICS OF THE PSEUDO-STRATIFIED CILIATED EPITHELIUM.

(Jap.) Ohno, T. (Sapporo Med. Coll., Japan). *Sapporo Med J* 31(4/5):202-206, 1967.

71 **CONDITIONED SALIVARY RESPONSE AND ITS SIGNIFICANCE.** *(E.)* Blumberger, W. (Max nck Inst. Nutrit. Physiol., Dortmund, Germany) and H. atzel. *Nutr Dieta (Basel)* 10(2):123-132, 1968.

72 **MECHANISM OF THE PILOCARPINE-STIMULATED INCREASE IN RAT SUBMAXILLARY AND AMYLASE LEVEL.** *(E.)* Chignell, C. F. (Natl. Heart Inst., NIH, Bethesda, Md.). *Biochem Pharmacol* 17(10):2225-2227, 1968.

7673 **CHOLESTEROL SYNTHESIS IN THE SQUIRREL MONKEY: RELATIVE RATES OF SYNTHESIS IN VARIOUS TISSUES AND MECHANISMS OF CONTROL.** *(E.)* Dietschy, J. M. (U. Texas Southwestern Med. Sch., Dallas) and J. D. Wilson. *J Clin Invest* 47(1):166-174, 1968.

See also: 7629,7643,7946

7674 THE MECHANISM OF PEPSIN SECRETION IN THE ISOLATED PERFUSED CANINE STOMACH.

(E.) Dritsas, K. G. (Surg.-Med. Res. Inst., U. Alberta, Edmonton, Canada) and K. Kowalewski. *Canad J Surg* 11(1):85-92, 1968.

Using a previously described preparation in which the isolated, perfused stomach from 1 dog is supported by the circulation of a second dog, the relation between pepsin secretion and the pH of gastric secretion was investigated. The perfusion circuit was modified by removing the stomach of the supporting dog and anastomosing the esophagus to the duodenum so that the only source of blood pepsinogen was the perfused stomach. Only the corpus of the stomach was isolated and perfused, using an airtight perfusion chamber exposed to intermittent positive pressure of 9/6 cm H_2O at a physiologic rhythm to preserve the effect of respiration on the intraabdominal pressure variation. In isolated stomachs stimulated with histamine (0.006-0.012 mg/min), pepsin secretion correlated with the pH of gastric secretion, decreasing at pH values above or below the optimal range of 1.4-2.4. Pepsin output increased continuously when the pH was maintained in this range during perfusion for up to 11 hr. Stomachs which were perfused, but not stimulated with histamine, produced gastric secretion with high pH and little pepsin. Instillation of HCl into the gastric lumen during the resting period was not as effective in increasing pepsin output as instillation of glycine buffer (pH 5.6) during histamine stimulation. The rate of restoration of blood pepsinogen to normal levels was slower with stomachs that produced gastric pepsin (histamine-stimulated, or HCl- or glycine-instilled) than with resting stomachs that did not produce gastric pepsin, indicating an inverse relation between pepsinogen diffused into the blood stream and that released into the gastric lumen. This is also supported by the observed drop in blood pepsinogen either immediately before or concomitant with the increase in gastric pepsin.

7675 EFFECT OF MEDICAL AND SURGICAL VAGOTOMY ON GASTRIC RESPONSE TO GRADED DOSES OF PENTAGASTRIN AND HISTAMINE.

(E.) Konturek, S. J. (Med. Sch., Krakow, Poland), A. Wysocki and J. Oleksy. *Gastroenterology* 54(3):392-400, 1968.

The effect of surgical vagotomy and/or antisecretory agents on pentagastrin- and histamine-induced gastric acid secretion was studied in 26 duodenal ulcer patients (22-55 yr old; 63-76 kg), including 22 patients pretreated with 50 mg hexamethonium i.m. and 1 mg atropine s.c. (medical vagotomy), 15 patients subjected to surgical vagotomy, 11 patients given both medical and surgical vagotomy. Histamine and pentagastrin doses were 20-80 μg/kg/hr and 0.5-8 μg/kg/hr, resp. by i.v. infusion. Medical vagotomy was performed 1 hr before histamine or pentagastrin infusion and (in the 3rd group) 1 week before surgical vagotomy. Before medical vagotomy, the mean maximal acid output was 50.62 ± 4.38 mEq/hr for pentagastrin and 45.02 ± 4.11 mEq/hr for histamine; the mean basal acid secretion was 7.09 ± 0.84 mEq/hr. After medical vagotomy the mean maximal acid response to doses producing maximal responses before vagotomy was reduced by 37% for pentagastrin and by 39% for histamine. The relative drop decreased as the dose increased. After medical vagotomy, the mean basal acid secretion was 1.47 ± 0.75 mEq/hr. The mean basal acid output after surgical vagotomy was 2.37 ± 0.91 mEq/hr; the mean maximal acid responses to doses producing maximal responses before surgical vagotomy were reduced by about 30% for histamine and 53% for pentagastrin. The relative drop again decreased as the dosage increased. There was a strong correlation between the maximal responses to pentagastrin and histamine before and after both types of vagotomy, as well as between the prevagotomy maximal acid output to both stimulants and the percentage drop in gastric acid secretion following vagotomy. The correlation between the percentage drop in maximal acid secretion in those patients having both medical and surgical vagotomy was not significant for either histamine or pentagastrin.

7676 EFFECT OF PROLONGED INHIBITION OF 11β-HYDROXYLASE ON GASTRIC SECRETION ULCER PROVOCATION, AND PARIETAL CELL POPULATION OF THE DOG.

(E.) Van Nostrand, D. M. (Minneapolis Veterans Hosp., Minn.) and D. M. Nicoloff. *Surgery* 63(2): 301-305, 1968.

The effect of Metopirone on gastric secretion, the parietal cell population, and ulcer formation was determined in dogs. Eleve of 21 dogs in which the antrum was transplanted onto the tran verse colon as an antiperistaltic diverticulum and gastrointestina continuity was restored by gastrojejunostomy received Metopirone (100 mg/kg daily) orally; those that survived 60 days were sacrificed. Five additional dogs with vagally denervated gastric pouches were studied for volume, free acid, and total acid content of pouch secretions before and after a 20-day course of Metopirone (100 mg/kg daily). Six other dogs with vagally denervated gastric pouches stimulated with 1.0 mg/day s.c. of histamine had pouch secretions analyzed before and afte a 3-day course of Metopirone (100 mg/kg/day). Full-thickness gastric biopsies were obtained from 5 dogs before and after a 3-week course of Metopirone (100 mg/kg) in order to determin the mucosal thickness and parietal cell population. It was note that Metopirone reduced the incidence of ulceration from 80% to 9% in dogs with the ulcerogenic preparation. Acid secretion from vagally denervated pouches stimulated with a standard meal and histamine was decreased 75% and 51%, resp., by administration of Metopirone. The reduction in free acid secretio and ulcerogenesis was not accounted for by a decrease in pariet cell population. Although not proven, the decreased ulceration noted with Metopirone is most likely related to the decrease in acid secretion.

7677 A NEW PREPARATION FOR GASTRIC SECRETORY STUDIES IN DOGS: TRANSPLANTATIO OF THE AUTOLOGOUS AND HOMOLOGOUS TOTAL STOM ACHS.

(E.) Odaka, M. (U. Washington Sch. Med., Seattle), H. N. Harkins and L. M. Nyhus. *Arch Surg* 96(6):902-908, 1968.

Adult mongrel dogs were prepared for gastric secretory studies by autotransplantation of the intact stomach to the groin and by auto- and homotransplantation of total stomachs divided into antrum and corpus with whole-layer barriers. The operativ technique and experimental methods are described in detail. Histamine, gastrin, and antral irrigation with acetylcholine were sed as gastric stimuli. Histamine and antral irrigation with cetylcholine produced the same responses as seen in conscious nimals. Gastrin did not produce the same effects, but the easons for this difference are unknown. It is recognized that perative trauma plays a part in the arrest of gastric secretion, lthough in these experiments its effect did not last as long s had previously been reported. The results of this study sugest that this preparation which combines features of both ransplantation and isolated perfusion, may find a place as a ool in the study of gastric secretion.

678 INFLUENCE OF INTRAGASTRIC pH ON ELECTROLYTE SECRETION BY THE GUINEA-IG STOMACH. *(E.)* James, R. M. V. (Sch. Pharm., Leicester egional Coll. Tech., England). *J Physiol* 195(3):623-630, 968.

lectrolyte secretion by the gastric mucosa in anesthetized emale guinea pigs was assessed over a wide range of pH values 2.6-10.3). Intragastric pH was maintained at a constant level y the instillation of 20 ml saline containing either NaOH or ICl, with or without added glucose, via a gastric cannula. Seretion was also measured at pH 10 and 8 (with added glucose) fter acetazolamide (50-100 mg/kg i.v.). With saline and glucose nfusions, the secretion of hydrogen ions increased progressively ver the pH range 4.4-9.4, with greatly increased output at pH 0.0 (double that at pH 9.0) and pH 10.3 (4 times that at pH .0). Hydrogen ion secretion ceased below pH 4.4. Acid seretion was unaffected by atropine, 2 mg/kg i.v. Sodium and otassium secretion were not correlated with pH, but chloride ecretion into glucose increased at pH values above 8.0, alhough not paralleling hydrogen ion output. Bicarbonate seretion increased with a rise in pH over the range 7-10.3. Aceazolamide (100 mg/kg) significantly depressed hydrogen ion ecretion at pH 10.0 for up to 4 hr; 50 mg/kg of acetazolamide ad only insignificant effects. Neither dose altered hydrogen on secretion at pH 8.0. Alkali-stimulated acid output probably esults from increased carbonic anhydrase activity.

7679 EFFECT OF GASTRIN II ON GASTRIC EMPTYING AND SECRETION DURING A TEST MEAL. *(E.)* Hunt, J. N. (Guy's Hosp. Med. Sch., London, England) and N. Ramsbottom. *Brit Med J* 4(5576): 386-387, 1967.

Two male subjects (27 and 50 yr old) were given 3 test meals in succession with 10-min intervals between them. For the 10 min before and during the 2nd test meal, a continuous i.v. infusion (0.125-3.0 μg/min) of gastrin was given. The stomach was washed out with 250 ml of water before each meal, which consisted of 750 ml of sodium citrate (0.1 N) containing 30 ml of phenol red as a marker. The recovered gastric contents were analyzed for acid and volume secretion. It was found that the meal given during the infusion of gastrin left the stomach more slowly than the preceding or the succeeding meal. The minimal effective dose of gastrin was 0.5 μg/min and 2.0 μg/min, resp. In both subjects, the threshold for stimulating acid secretion in the 2nd meal was less than that required to slow emptying–0.125 and 0.5 μg, resp. With most doses, the slowing of emptying had worn off in the 10 min between stopping the infusion of gastrin and giving the 3rd meal. However, with larger doses of gastrin (0.75 and 2.0 μg, resp.), the acid secretion in response to the 3rd meal was greater than that in response to the 2nd. It is concluded that the rate of gastric emptying was slowed in proportion to the dose given, and the increased rate of gastric secretion outlasted the slowing of gastric emptying.

7680 EFFECT OF VAGOTOMY ON GASTRIC ACID SECRETION STIMULATED BY PENTAGASTRIN AND HISTAMINE. *(E.)* Payne, R. A. (North Middlesex Hosp., London, England), A. G. Cox, J. Spencer and F. C. Y. Cheng. *Brit Med J* 4(5577):456-457, 1967.

The maximal gastric responses to stimulation by histamine and pentagastrin were determined in 15 patients with uncomplicated chronic duodenal ulcer before and after total vagotomy and pyloroplasty. All patients showed a substantially reduced response to both agents after vagotomy. The mean reduction in the histamine response (66.4%) did not differ significantly from the mean reduction in the pentagastrin response (61.5%). This investigation shows that vagotomy reduces the maximal gastric acid secretory response to pentagastrin and to histamine by similar proportions; however, unlike histamine, pentagastrin has minimal side-effects and seems likely to supersede it in the measurement of gastric acid secretion.

7681 THE EFFECT OF ACUTE ETHANOL INTOXICATION ON THE AMOUNT OF HISTAMINE IN THE DIGESTIVE TRACT OF THE RAT. *(Fr.)* de Saint-Blanquat, G. (Fac. Sci., Toulouse, France) and R. Derache. *Therapie* 22(5):1045-1053, 1967.

Male Wistar rats were given 2 g/kg of alcohol, either alone or with an isocaloric solution of glucose. Fluorimetric analyses made on animals killed 15 sec to 8 hr after administration of alcohol showed that in all animals the histamine contents were higher in the stomach and duodenum than in the liver. The largest amounts of histamine were found in parts of the stomach where secretion is the greatest. There were no significant differences between the histamine contents of the stomach, liver, and duodenum in alcohol-treated rats and those in untreated controls. Alcohol significantly reduced the volume of gastric juice secreted and the total amount of histamine in gastric juice. Urinary excretion of histamine was increased, but not significantly, in alcohol-treated rats. These results indicate that the increase in gastric acid secretion produced by ethanol is not caused by increases in histamine production.

7682 SELF-REGULATED INHIBITION OF STIMULATED GASTRIC SECRETION. GASTROPHYSIOLOGICAL STUDIES ON DOGS WITH STOMACH FISTULAS. *(Ger.)* Lick, R. F. (Surg. Polyclin., U. Munich, Germany), R. Endriss, W. Hart and D. Balser. *Med Klin* 62(45):1763-1766, 1967.

Fistulas were made in the anterior wall of the nonisolated antrum, near the lesser curvature, in 2 dogs with Heidenhain pouches. The volume, pH, free acid, and pepsin activities of stomach secretion were determined with and without perfusion of the antrum with acid buffer solution (pH 1.2). Perfusion partially inhibited increases in gastric secretion and acid secretion produced by 0.05 mg/kg histamine s.c., 0.025 mg/kg neostigmine i.m., 7.5 g/kg tetragastrin i.m., 7.5 g/kg pentagastrin s.c., and 7.5 g/kg i.v. gastrin. In addition, perfusion of acid inhibited increases in pepsin secretion produced by tetragastrin and pentagastrin. It was not established whether antral self-regulation of gastric secretion operates by blocking basal liberation of gastrin or was produced by an inhibitory hormone.

7683 **STUDIES ON THE GASTRIC INHIBITORY SUBSTANCE IN HUMAN SALIVA (SALIVOGASTRONE). 1. EXPERIMENTAL STUDIES.** *(Jap.)* Miyoshi, A. (Fac. Med., Kyoto U., Japan), M. Moriga, M. Ohbayashi, N. Iwai, K. Kogawa, M. Kobayashi, T. Suyama, T. Imoto, H. Ando and M. Yamamoto. *Jap Arch Intern Med* 15(5/6):121-135, 1968.

7684 **EFFECT OF MEDICAL VAGOTOMY ON ACID AND PEPSIN RESPONSES TO HISTAMINE AND PENTAGASTRIN IN MAN.** *(E.)* Konturek, S. J. (Med. Sch., Krakow, Poland), Z. Szybinski and T. Horzela. *Scand J Gastroent* 2(4):311-315, 1967.

7685 **SECRETION IN THE HEIDENHAIN POUCH OF THE RAT STIMULATED BY RESERPINE.** *(E.)* Rees, J. R. (New York Hosp.-Cornell Med. Ctr., N. Y.), S. Bjornsson and B. Thorbjarnarson. *J Surg Res* 8(4):188-191, 1968.

7686 **SOME LATE EFFECTS OF A BILATERAL TRUNK VAGOTOMY IN THE RAT.** *(It.)* Vecchioni, R. (Inst. Gen. Surg. Clin., U. Padua, Italy), G. Perrino and C. Cordiano. *Acta Chir Ital* 22(Suppl. 1):69-72, 1966.

7687 **RADIOTELEMETRICAL STUDY OF THE STOMACH AFTER ADMINISTRATION OF SEVERAL THERAPEUTIC AGENTS.** *(Rus.)* Samson, E. I. (Chernovitskii Med. Inst., USSR). *Ter Arkh* 39(11):99-101, 1967.

7688 **GASTRIC SECRETION AFTER INTRAVENOUS ADMINISTRATION OF FATTY EMULSIONS.** *(It.)* Vecchioni, R. (Inst. Gen. Clin. Surg., U. Padua, Italy), D. D'Amico, C. Cordiano and G. Perrino. *Acta Chir Ital* 22(Suppl. 2):129-136, 1966.

7689 **THE EFFECTS OF INDOMETHACIN ON GASTRIC FUNCTION.** *(It.)* Lunghetti, R. (Inst. Gen. Clin. Med., U. Siena, Italy), G. Bianco, R. Marcolongo and A. Carcassi. *Settimana Med* 55(24):1339-1353, 1967.

See also: 7604,7656,7845,7854,7857,8059,8458

690 STUDY OF THE STIMULATING EFFECT ON EXOCRINE PANCREATIC SECRETION OF A
YNTHETIC PRODUCT WITH GASTRIN-LIKE ACTIVITY.

t.) Michetti, P. (Inst. Med. Path., U. Genoa, Italy) and G.
allo. *Epatologia* 13(6):1281-1288, 1967.

n 10 subjects with histamine-resistant achlorhydria, 0.5 IU/kg
f secretin i.v. reduced the volume of pancreatic secretion and
ie bicarbonate concentration and amylase activity of the pan-
reatic juice. When 6 μg/kg of pentagastrin were given i.m.
nmediately after administration of the same dose of secretin,
ie volume of pancreatic juice increased to normal values and
nylase activities were normal in all 10 subjects. The bicarbon-
te concentrations of the pancreatic juice were normal in 3 sub-
cts and reduced in 7. The effects of pentagastrin on pancrea-
c secretion were not as pronounced as those of gastrin, prob-
bly because the synthetic product lacks the complete molecu-
r sequence of gastrin.

7691 TESTS OF SECRETIN AND CHOLECYSTOKININ-PANCREOZYMIN AFTER INTRA-
VENOUS AND INTRA-INTESTINAL INJECTION IN RAB-
BITS.

(Ger.) Thorn, W. (State Inst. Chem., U. Hamburg,
Germany), B. Müldener and H. Rothe. *Arzneimittelforschung*
18(4):456-459, 1968.

Pancreatic juice was collected through a tube implanted in the pancreatic duct and hormones were infused through a catheter into the femoral vein or injected into the duodenum in 80 rabbits under Nembutal anesthesia. The normal amount of pancreatic juice, secreted during various stages of digestion, ranged from 96.0-2431.0 mg/hr with a mean of 678.0 mg/hr. Protein secretion ranged from 6.45-14.10 mg/hr with a mean of 3.21 mg/hr. Various doses of hormones were administered i.v. or intraduodenally over a 15-20 sec period. Since large doses (80 mg) of hormone concentrate administered i.v. or intraduodenally produced intestinal spasms, the secretion of pancreatic juice was not strictly dose-dependent. The effect produced by intraduodenal administration of hormones depended upon the functional state of the part of the duodenum in which the injection was made. Pregnancy apparently promoted pancreatic secretion. Continuous i.v. infusion of hormones produced more secretion than a single i.v. or intraduodenal injection.

7692 COMPARISON OF PANCREATIC EXOCRINE SECRETION AFTER STIMULATION WITH PANCREOZYMIN AND SECRETIN AND AFTER A TEST MEAL.

(It.) Fiore, E. (Inst. Spec. Med., U. Bologna, Italy), P. R. Dal Monte and M. Sasdelli. *G Clin Med* 48(11):1194-1203, 1967.

Determinations were made of the volume, trypsin and amylase activities, and bicarbonate concentration of duodenal juice from 10 normal subjects, aged 32-54 yr. These studies were performed before and after i.v. administration of 95 U of pancreozymin followed, 15 min later, by 95 U of secretin and after a test meal consisting of 45 g skimmed milk powder, 45 g corn oil, and 20 g glucose made up to 300 ml with water and homogenized. The mean volume of secretion, amylase activity and bicarbonate concentration were higher after pancreozymin and secretin stimulation while the mean trypsin activity was higher after the test meal. With the test meal the maximum enzyme and bicarbonate concentrations and the maximum volume of secretion were reached about 15 min later than with i.v. pancreozymin and secretin. Low amylase activities obtained after the test meal are probably due to the neutralization of some salivary amylase by gastric acid. Low bicarbonate concentrations after the test meal are probably due to the buffering effect of the meal and the greater bicarbonate stimulation produced by exogenous secretin. From these findings it is concluded that exocrine pancreatic function can be studied adequately by determining trypsin activities before and after administration of the test meal.

7693 EXOCRINE PANCREATIC SECRETION AFTER TEST MEALS IN NORMAL SUBJECTS AND SUBJECTS WITH PANCREATIC INSUFFICIENCY.

(It.) Dal Monte, P. R. (Inst. Spec. Med. Path., U. Bologna, Italy), E. Fiore, L. Lodi and R. Cremonini. *G Clin Med* 48(10):1054-1069, 1967.

The volume of pancreatic juice and its amylase and trypsin activities were determined on 31 patients with no pancreatic diseases and on 11 with pancreatic insufficiency due to chronic pancreatitis (6 cases), pancreatic cysts (2 cases), and cancer of the head of the pancreas (3 cases). Controls and patients were matched for age and sex. Pancreatic juice was collected from the duodenum through an Einhorn catheter before and at 30 min intervals after a test meal consisting of an emulsion of 45 g of skimmed milk, 45 g of corn oil, and 25 g of glucose made up to 300 ml with water. In normal subjects the volume of pancreatic juice was significantly higher than basal values in the first 2 samples taken after the test meal and did not decrease to its initial level in the 2 hr observation period. In patients with pancreatic insufficiency the volume of secretion increased in the first 30 min after the test meal, but then gradually decreased to reach basal values after 2 hr. Amylase and trypsin activities reached a maximum 30 min after the test meal in controls and 1 hr after in patients with pancreatic insufficiency. Differences between controls and patients with pancreatic insufficiency were most pronounced for trypsin.

7694 THE COLLECTION OF PANCREATIC SECRETION IN THE DOG: A SURGICAL TECHNIQUE.

(It.) Ricci, M. (Inst. Hydrol., U. Rome, Italy) and F. Tesio. *Epatologia* 13(6):1289-1293, 1967.

7695 EFFECT OF INTRAVENOUS ALCOHOL ON THE PANCREAS. [CASE REPORT]

(E.) Lowenfels, A. B. (Grasslands Hosp., Valhalla, N. Y.), B. Masih, T. C. Y. Lee and M. Rohman. *Arch Surg* 96(3):440-441, 1968.

7696 SYNTHESIS AND EXCRETION OF PANCREATIC HYDROLASES IN RAT IN WHICH PROTEIN MALNUTRITION IS FOLLOWED BY A BALANCED DIET.

(Fr.) Robberecht, P. (Fac. Med. Pharm., Free U. Brussels, Belgium), A. Vandermeers, M. H. Khayat, J. Rathe and J. Christophe. *Bull Soc Chim Biol (Paris)* 49(12):1735-1750, 1967.

7697 **EVIDENCE FOR SEPARATE EPINEPHRINE AND GLUCAGON RESPONSIVE ADENYL CYCLASE SYSTEMS IN RAT LIVER.** *(E.)* Bitensky, M. W. (New York U. Sch. Med., N. Y.), V. Russell and W. Robertson. *Biochem Biophys Res Commun* 31(5):706-712, 1968.

Liver homogenates from male Sprague-Dawley rats were incubated in media containing ^{14}C-ATP and ^{14}C-cyclic-AMP, synthesized by the liver enzymes, was isolated from the mixture of labeled products by descending chromatography on Ecteola thin layers. The response of whole-homogenate cyclase to glucagon *in vitro* was consistently less than that of repeatedly washed membrane particles derived from the same homogenate. Glucagon-responsive cyclase activity increased after washing (soluble phosphodiesterase was removed); however, responsiveness to epinephrine was lost in these particles after repeated washings. After dilution of the whole homogenate enzyme from 800 to 600 μg/20 μL, the epinephrine-responsive cyclase exhibited the same apparent stimulation of specific activity as did the glucagon-responsive cyclase. Greater dilutions resulted in rapid loss of epinephrine cyclase activity, and resuspension of the particles in high-speed supernatants from the whole homogenate did not restore the epinephrine-responsive adenyl cyclase activity. Epinephrine-responsive cyclase was more easily damaged by heating or sonication than was the glucagon-responsive cyclase. Following incubation at 37C for 18 min, response to epinephrine fell more than 80%, whereas the response to glucagon was unaffected, and after sonication the response to epinephrine fell more than 50%, whereas the response to glucagon fell only 15%. Differences were also noted in the sedimentation rates of the 2 systems. The existence of 2 completely separate adenyl cyclase systems in liver which respond independently to specific hormones is proposed.

7698 **MECHANISM OF THE DAILY RHYTHM IN HEPATIC TYROSINE TRANSAMINASE ACTIVITY: ROLE OF DIETARY TRYPTOPHAN.** *(E.)* Wurtman, R. J. (Dept. Nutr. Food Sci., Massachusetts Inst. Tech., Cambridge), W. J. Shoemaker and F. Larin. *Proc Nat Acad Sci USA* 59(3):800-807, 1968.

Both intact and hypophysectomized female Sprague-Dawley rats demonstrated a daily rise in hepatic tyrosine transaminase activity on an *ad libitum* Purina Chow diet. In neither type of experimental animal was the elevation preceded by a rise in plasma or liver tyrosine concentrations. Plasma and liver tyrosine tended to fall after the transaminase activity had risen, suggesting that more of the amino acid was being metabolized. Statistically significant decreases were observed in the hepatic tyrosine content of intact rats and in the plasma tyrosine concentration of hypophysectomized animals. When the hepatic tryptophan content and tyrosine transaminase activity were measured in rats killed at different times of day, the tryptophan concentration was lowest at 9 A.M. and by 2 P.M. it had risen to levels of 14.5 μg/g. It did not change significantly for 12 hr, after which it fell to the 9 A.M. levels. Tyrosine transaminase activity was lowest at 2 P.M.; it rose 5-fold during the next 10 hr. The increase in hepatic tryptophan levels thus preceded the daily elevation in tyrosine transaminase activity by several hours. The daily rhythm of hepatic tyrosine transaminase was rapidly abolished when the rat was deprived of dietary protein. It could be restored when the rat was given access to a mixture of amino acids, but only if this mixture included tryptophan. In the absence of other amino acids, dietary tryptophan alone produced gross oscillations in tyrosine transaminase activity, provided this amino acid was given in high concentrations. The tyrosine transaminase rhythm in the normal rat is ascribed mainly to the fact that the rat normally consumes significant quantities of food during the hours which precede the daily rise in tyrosine transaminase activity.

7699 **DIRECT MEASUREMENT OF THE RATES OF SYNTHESIS OF PLASMA PROTEINS IN CONTROL SUBJECTS AND PATIENTS WITH GASTROINTESTINAL PROTEIN LOSS.** *(E.)* Wochner, R. D. (Natl. Cancer Inst., NIH, Bethesda, Md.), S. M. Weissman, T. A. Waldmann, D. Houston and N. I. Berlin. *J Clin Invest* 47(4):971-982, 1968.

Instantaneous synthetic rates of the plasma proteins were measured in 7 control subjects, (all males), 1 patient with falling serum albumin (a male) and 5 patients (2 males and 3 females) with gastrointestinal protein loss by pulse-labeling the hepatic arginine pool with ^{14}C-bicarbonate, using a modification which enabled the specific activity of the urinary urea to be converted into simultaneous measurements for albumin, fibrinogen, and transferrin. The synthetic rates of albumin determined by this method were compared to those estimated from simultaneous studies with ^{131}I-albumin. The close agreement between these two methods supported the validity of the modification and suggested that there is relatively little fluctuation in the rate of albumin synthesis with time. The albumin synthesis rates in 6 control patients averaged 5.8 mg/kg/hr, compared to 7.2 mg/kg/hr in the 5 patients with gastrointestinal protein loss. In these patients there was relatively little acceleration of albumin synthesis in response to continued loss of plasma protein into the gastrointestinal tract. Fibrinogen synthesis rates averaged 1.9 mg/kg/hr in 5 control patients and 3.2 mg/kg/hr in 5 patients with gastrointestinal protein loss. Transferrin synthesis rates exhibited considerable individual variation in both groups and averaged 0.24 mg/kg/hr in 4 control subjects and 0.31 mg/kg/hr in 5 patients with gastrointestinal protein loss. This new method provides a direct measurement of protein synthesis rates and is applicable to the investigation of the physiological factors controlling the rates of synthesis of plasma proteins.

7700 **UREA CYCLE ENZYMES IN THE DEVELOPING HUMAN FETUS.** *(E.)* Colombo, J. P. (Children's Hosp., U. Bern, Switzerland) and R. Richterich. *Enzym Biol Clin* 9(1):68-73, 1968.

Carbamylphosphate synthetase, argininosuccinate cleavage enzym arginase and ornithine-carbamyl transferase were measured in the livers of human fetuses at various stages of development. Activity was measurable for all enzymes from the 50th day of pregnancy, and except for ornithine-carbamyl transferase, all enzymes showed 1 or 2 peaks of activity between the 50th and the 110th day. Carbamylphosphate synthetase showed an initial peak at 60 days and a higher peak at 90 days, between which times the concentration was in the normal adult range; the activity then decreased until the 200th day when it beg rise again, reaching the adult range at the end of pregnancy. he second peak of argininosuccinate cleavage enzyme activity as lower than the first, after which the concentration de- eased for about 2 months and then slowly rose toward the nd of gestation, although it never again regained the first level. rnithine-carbamyl transferase activity remained relatively con- ant throughout gestation, but increased markedly toward the nd when the values reached the lower limits of the adult range. rginase activity followed a pattern similar to that of cleavage nzyme; however, the adult range was not attained at the end f gestation. The carbamylphosphate synthetase and ornithine- arbamyl transferase concentrations observed are a strong argu- ent in favor of fetal urea synthesis.

701 **BILE SALTS AND LIPOLYSIS.** *(E.)* Štork, A. (1st Med. Clin., Charles U., Prague, Czechoslovakia), . Fabian and J. Šponarová. *Enzym Biol Clin* 9(2):137-142, 968.

he effect of sodium dehydrocholate (10 ml of 20% solution v.) on plasma free fatty acid levels was investigated in 10 ealthy fasted individuals with an average age of 45.2 yr. The asting free fatty acid level was 588 ± 128 μEq/1000 ml plasma; is fell to 308 ± 149 μEq 10 min after sodium dehydrocholate dministration. The free fatty acid level rose after 20 min and eturned to normal within 60 min. Heparin (50 mg i.v.) in- reased plasma fatty acids by 762.5 ± 378 μEq/1000 ml in 0 healthy subjects (average age 21.8 yr); if sodium dehydro- holate was administered before heparin, the increase was only 96 ± 291.7 μEq. Although the increase in free fatty acids fter dehydrocholate and heparin was lower than the increase fter heparin alone, this difference was not significant. Lipo- rotein lipase activity was 24.32 ± 3.63 U after heparin and 8.42 ± 4.31 U if dehydrocholate was administered before eparin; this decrease in activity was significant. In 7 subjects average age 52.2 yr) lipoprotein lipase activity after heparin as 21.41 ± 3.66 U; if dehydrocholate was added to the reac- ion mixture instead of injected i.v., the lipoprotein lipase activ- ty decreased to 11.25 ± 2.31 U. The decrease in plasma fatty cid levels after i.v. sodium dehydrocholate is therefore ascribed o inhibition of lipolysis. It is suggested that lipoprotein lipase hibition by sodium dehydrocholate may play an important ole in reducing the plasma free fatty acid levels in cholestasis.

702 **ELEVATION AND DEPRESSION OF HEPATIC TYROSINE TRANSAMINASE ACTIVITY BY EPLETION AND REPLETION OF NOREPINEPHRINE.** *(E.)* lack, I. B. (Natl. Inst. Mental Health, NIH, Bethesda, Md.) and . Axelrod. *Proc Nat Acad Sci USA* 59(4):1231-1234, 1968.

drenalectomized, female, Sprague-Dawley rats weighing 160- 80 g were used to demonstrate that depletion of norepine- hrine raises hepatic tyrosine transaminase activity and reple- ion of the catecholamine decreases enzyme activity. Tyrosine ransaminase activity was increased by 100-200% after adminis- ration of reserpine (2 mg/kg i.p.) and exhibited an inverse cor- elation with the level of brain norepinephrine. Reserpine caused he expected rise in tyrosine transaminase activity, whereas uromycin (50 mg/kg i.p.) reduced the enzyme activity to con- rol levels in rats pretreated with reserpine. The administration f α-methyl-p-tyrosine, an inhibitor of tyrosine hydroxylase hich is the rate limiting enzyme in norepinephrine biosynthe- is, increased tyrosine transaminase activity nearly 5-fold and reduced brain norepinephrine to approximately 10% of control values. L-DOPA significantly raised brain norepinephrine concentrations in rats previously treated with the tyrosine hydroxylase inhibitor, and reversed the effect of α-methyl-p-tyrosine in raising tyrosine transaminase activity. L-DOPA alone had no effect on the enzyme.

7703 **CHANGING PATTERNS OF HISTONE ACETYLATION AND RNA SYNTHESIS IN REGENERATION OF THE LIVER.** *(E.)* Pogo, B. G. T. (Rockefeller U., New York, N. Y.), A. O. Pogo, V. G. Allfrey and A. E. Mirsky. *Proc Nat Acad Sci USA* 59(4):1337-1344, 1968.

Male Sprague-Dawley rats weighing 250-280 g were used to study acetate incorporation and turnover in different histone fractions of normal and regenerating liver, comparing histone acetylation and RNA synthesis at different times. It was found that the patterns of histone acetylation are strikingly altered soon after partial hepatectomy, and that a peak of acetylation of the arginine-rich histones occurs before maximal rates of RNA synthesis are achieved. The kinetics of histone acetylation were followed by tracer techniques, measuring the specific activities of different histone fractions at different times following a single injection of acetic acid (^{3}H-methyl). Normal, sham-operated, and partially hepatectomized rats were given the tritiated precursor i.p. and killed after 15 and 60 min. The specific activity of the arginine-rich band in normal liver was plotted as a function of time and the peak specific activity was observed at 15 min, with a rapid fall after that time, so that at 60 min only about one third of the acetyl groups originally incorporated into the histones remained. This rapid turnover of histone acetyl groups was also noted in the sham-operated rats. The pattern of acetylation is dramatically altered in regenerating liver and changes are evident even at 1 hr after partial hepatectomy. An increase in the acetyl content of the arginine-rich histones occurs within 1 hr, and a peak of acetylation occurs at 3-4 hr. RNA polymerase assays showed that gene activation begins 1-2 hr after partial hepatectomy and reaches a plateau at about 6 hr. Rates of deacetylation are decreased, while rates of histone acetylation are increased. These findings correlate with the view that acetylation of histones modifies DNA-histone interactions, and that the subsequent changes influence the template activity of the chromatin for RNA synthesis.

7704 **EFFECTS OF DIET ON HEPATIC TRIGLYCERIDE SYNTHESIS.** *(E.)* Fallon, H. J. (U. North Carolina Sch. Med., Chapel Hill) and E. L. Kemp. *J Clin Invest* 47(4):712-719, 1968.

In studies on rats, the activity of hepatic L-α-glycerophosphate acyltransferase (GPAT) was correlated with the rate of triglyceride synthesis under various dietary conditions and the *in vitro* effects of changes in substrate and albumin concentration on reaction velocity were measured. The rate of incorporation of L-α-glycerophosphate into triglyceride was doubled when the diet contained 64% glucose as compared to a normal diet of chow. An 80% increase in hepatic GPAT was noted in rats fed high glucose diets, but no change was noted in either L-α-glycerophosphate or triglyceride content. High carbohydrate diets containing 64% fructose, 64% potato starch, or 32% sucrose and 32% starch resulted in increased triglyceride synthesis and GPAT activity. The diet containing 64% starch was significantly less effective than the other carbohydrate diets in increasing the rate of L-α-glycerophosphate incorporation into triglycerides. Rats fed 40% corn oil showed significantly higher levels of GPAT activity than those fed high glucose diets but the hepatic concentration of L-α-glycerophosphate was unchanged by the 40% corn oil diet. Increasing the albumin concentration also resulted in increased GPAT activity. Since the substrate concentrations for GPAT *in vivo* are always significantly less than those used to measure the maximum activity *in vitro*, it is likely that the reaction velocity of GPAT in liver is significantly less than the maximum.

7705 ALTERATION IN LIVER LIPID AND LIPID FATTY ACID COMPOSITION AFTER PARTIAL HEPATECTOMY IN THE RAT.

(E.) Glende, E. A. Jr. (Cleveland Metropolitan Gen. Hosp., Ohio) and W. S. Morgan. *Exp Molec Path* 8(2):190-200, 1968.

A significant increase in liver neutral lipids was observed in Sprague-Dawley rats as early as 4 hr after partial hepatectomy, reaching a maximum of 65-69 mg neutral lipid/g fresh liver at 18-24 hr and declining to sham-operated control levels within 72 hr. Hepatic phospholipid levels remained essentially unchanged throughout the observation period. The serum free fatty acid concentration was consistently significantly higher (about 1.3-1.5 fold) 9-72 hr after partial hepatectomy than in control animals, with a significant increase in the oleic and linoleic acid content and a decrease in stearic and arachidonic acids at 24 hr. Fatty acid analysis of total liver phospholipids showed marked increases in oleic and linoleic acids and significant decreases in stearic and arachidonic acids which were maximal at 24 hr after surgery. Similar, though less dramatic increases in the level of oleic and linoleic acids, with corresponding decreases in palmitic and stearic acids, were observed in the liver neutral lipid fraction. The fatty acid pattern of the triglycerides, the major constitutent of the neutral lipid, also revealed significant increases in oleic acid and a concomitant decrease in palmitic acid. Individual phospholipids separated by thin-layer chromatography (phosphatidyl inositol, lecithin, phosphatidyl ethanolamine) all showed fatty acid patterns similar to those observed in the total phospholipid fraction. Palmitic, oleic and linoleic acids formed the major fatty acids of the epididymal fat pad lipids in control rats and 24 hr after partial hepatectomy. These studies suggest that the fatty acids derived from adipose tissue lipid are the source of the fatty acids incorporated into the liver lipids after partial hepatectomy. Ergotamine (5.0 mg/kg s.c.), 3,5-dimethylpyrazole (15.0 mg/kg, i.p.) and nicotinic acid (200 mg/kg, i.p.) given at the time of the operation significantly reduced the accumulation of liver triglyceride 18 hr after partial hepatectomy.

7706 THE LOCALIZATION OF MITOTIC FIGURES IN REGENERATING MOUSE LIVER.

(E.) Melvin, J. B. (Div. Biol. Med. Sci., Brown U., Providence, R. I.). *Anat Rec* 160(3):607-617, 1968.

The uniformity of the mitotic index during hepatic regeneration was determined by counting mitotic figures from early prophase through telophase in livers of BU^B mice given CCl_4 s.c. (0.1 cc of 40% solution) and ^3H-thymidine (1.4 C/mM) i.p. and sacrificed 48 hr later at the period of peak mitotic activity. Highly significant variation was found among counts on consecutive days when no attempt was made to count the same regions of the slide, accounting for 6.14% of the total variance. However, variation among different unit areas of observation (oil-immersion microscopic field of 120 μ) accounted for 80.45% of the total variance, indicating a nonrandom distribution of mitotic counts. When larger areas were considered the variation was satisfactorily randomized. Autoradiographs of liver cells 40 hr after administration of CCl_4, at the period of peak DNA synthesis, demonstrated labeled cells outlining radial pathways which extended from the portal triads; this arrangement suggests that these cells were adjacent to the radial portal twigs extending from the branches of the portal vein into the adjacent lobules, conforming to the concept of the liver acinus. When injection of CCl_4 was followed by 0.1 mg colchicine (i.p.) 48 hr later to show the pattern of mitotic figures and by india ink (into the portal vein) at death to outline the portal vessels, the distribution of mitotic figures also appeared to be determined by the winding path of the terminal portal twigs. Mitotic figures also occurred adjacent to necrotic cells in the centrilobular zone of the classical lobule. These statistical and histologic studies indicate that the location of dividing cells in regenerating liver is correlated with the location of a rich blood supply and cannot be considered random. Such a nonrandom distribution of mitotic figures requires a reexamination of quantitative methods for determining the labeling index or mitotic index in regenerating liver.

7707 β-HYDROXYBUTYRATE DEHYDROGENASE ACTIVITY IN LIVER AND LIVER TUMORS.

(E.) Ohe, K. (Temple U. Sch. Med., Philadelphia, Pa.), H. P. Morris and S. Weinhouse. *Cancer Res* 27(8):1360-1371, 1967.

D(−)-β-Hydroxybutyrate dehydrogenase was assayed in rat liver under various dietary conditions and in a series of liver tumors of varying growth rates and degrees of differentiation. The liver enzyme activity in normal male CFN rats was about 16 U/g of tissue and was not affected appreciably by drastic dietar alterations. The enzyme was also present in a series of slow-growing, well-differentiated hepatic carcinomas, though at levels only 10-30% of that of normal liver, and was very low in severa rapidly growing, poorly differentiated tumors, including the Novikoff hepatoma. Like the normal liver enzyme, the tumor enzyme was tightly bound to mitochondria. Both were released in a particulate form capable of transferring electrons from substrate to exogenous nicotinamide adenine dinucleotide or its reduced form by sonic treatment or osmotic alteration, but could also be solubilized, though with considerable loss of activity, by treatment with 0.3% cholate. The soluble enzymes did not require lecithin and were not activated by this substance, in contrast with previous data on the soluble beef-heart dehydrogenase. They were eluted rapidly from Sephadex G-200 as a single band, indicating a molecular weight near 200,000. Both the soluble and particle-bound forms of the liver and tumor enzymes had similar kinetic properties, thermal stability, and pH optima, and it is assumed that they are identical.

7708 HORMONAL AND SUBSTRATE INDUCTION OF TRYPTOPHAN PYRROLASE IN REGENERATING RAT LIVER.

(E.) Seidman, I. (New York U. Sch. Med., N. Y.), G. W. Teebor and F. F. Becker. *Cancer Res* 27(9):1620-1625, 1967.

induction of tryptophan pyrrolase by hydrocortisone owing 70% hepatectomy was studied in adult male Sprague- vley rats. An alteration in the rate of induction was ob- ed in both sham-operated and 70% hepatectomized rats y in the postoperative period. Normal maximal values were ntually reached, however. Later in the postoperative period, uction patterns were similar to those observed in unoperated . During the period in which DNA synthesis is known to ur following 70% hepatectomy, the induction of tryptophan rolase by hydrocortisone was depressed in the 70% hepa- omized but not in the sham-operated rats. At this latter e period, induction of tryptophan pyrrolase by L-tryptophan not diminished in the regenerating liver. It has been sug- ed that alterations in enzyme control mechanisms, such as absolute or relative failure of tryptophan pyrrolase induc- by cortisone in minimal-deviation hepatomas, may repre- t a fundamental biochemical alteration in tumors. This ly shows that a depressed response to hormonal enzyme uction also occurs in nonneoplastic hepatic cells preparing livide.

9 **INFLUENCE OF SOME ALIPHATIC ALCOHOLS ON THE METABOLISM OF RAT LIVER SLICES.** Forsander, O. A. (Res. Lab. State Alcohol Monopoly, inki, Finland). *Biochem J* 105(1):93-97, 1967.

effect of some aliphatic alcohols on oxygen uptake, carbon ide production, acid formation and lactate and pyruvate con- rations was studied in liver slices from Wistar rats of both s. At the concentrations used, none of the alcohols influ- ed oxygen uptake. Of the alcohols that are not oxidized by alcohol dehydrogenase, methanol increased carbon dioxide luction, propan-2-ol decreased it, and 2-methylpropan-2-ol no effect. All the alcohols that are oxidized by the enzyme ngly decreased carbon dioxide production. The alcohols are not oxidized had no marked effect on the lactate/pyru- concentration ratio, whereas the other alcohols strongly in- sed the ratio. A highly significant correlation was found veen the effects of the alcohols on pyruvate concentration carbon dioxide production. It is assumed that a shift in the ox potential inhibits the function of the tricarboxylic acid e of the liver.

0 **FATTY ACID CHANGES IN LIVER AND PLASMA LIPID FRACTIONS AFTER SAFFLOWER OIL S FED TO RATS DEFICIENT IN ESSENTIAL FATTY IDS.** *(E.)* Johnson, R. R. (Dept. Nutrit. Sci., U. California, keley), P. Bouchard, J. Tinoco and R. L. Lyman. *Biochem J* (1):343-350, 1967.

ty acid patterns of liver and plasma triglycerides, phospho- ds and cholesteryl esters were determined at intervals during hr after rats deficient in essential fatty acids had been given feeding of linoleate (as safflower oil). Liver triglyceride, spholipid and cholesteryl ester fatty acid compositions did change for up to 7 hr after feeding. Between 7 and 10 hr, leic acid began to increase in all fractions, but arachidonic l did not begin to rise in the phospholipids until 14-19 hr r feeding. Oleic acid and eicosatrienoic acid in liver phospho- ds began to decline at about the time that linoleic acid in- ased, i.e. about 9 hr before arachidonic acid began to in- ase. Changes in linoleic acid, arachidonic acid and eicosatrie- c acid in phosphatidylcholine resembled those in the total phospholipids. Phosphatidylethanolamine had a higher percentage content of arachidonic acid before the linoleate was given than did phosphatidylcholine, and after the linoleate was given the fatty acid composition of this fraction changed little. The behavior of the plasma lipid fatty acids was similar to that of the liver lipids, with changes in linoleic acid, eicosatrienoic acid and arachidonic acid appearing at the same times as they occurred in the liver. The results indicated that linoleic acid was preferentially incorporated into the liver phospholipid at the expense of eicosatrienoic acid and oleic acid. The decline in these fatty acids apparently resulted from their competition with linoleic acid for available sites in the phospholipids rather than from any direct replacement by arachidonic acid.

7711 **BILIARY EXCRETION OF FOREIGN COMPOUNDS: SPECIES DIFFERENCES IN BILIARY EXCRETION.** *(E.)* Abou-El-Makarem, M. M. (St. Mary's Hosp., Med. Sch., London, England), P. Millburn, R. L. Smith and R. T. Williams. *Biochem J* 105(3):1289-1293, 1967.

The biliary excretion of injected $[^{14}C]$aniline, $[^{14}C]$benzoic acid, 4-aminohippuric acid and 4-acetamidohippuric acid was studied in 6 or 8 species of animal (rat, dog, hen, cat, rabbit, guinea pig, rhesus monkey and sheep). These compounds, with molecular weights in the range 93-236, were poorly excreted in the bile in all species examined, so that there was little significant species difference in the extent of their biliary excretion. Compounds of higher molecular weight (355-495) were also studied, namely succinylsulfathiazole, $[^{14}C]$stilbestrol glucuronide, sulfadimethoxine N^{1}-glucuronide, and phenolphthalein glucuronide. With these compounds, a clear species difference in the extent of biliary excretion was found, the rat, dog and hen being good excretors, the rabbit, guinea pig and monkey poor excretors, and the cat and sheep taking an intermediary position. There was a general trend for biliary excretion to be higher in all species when the compounds were of higher molecular weight. These results are discussed in their relation to species differences in drug metabolism.

7712 **BILIARY EXCRETION OF FOREIGN COMPOUNDS: SULPHONAMIDE DRUGS IN THE RAT.** *(E.)* Millburn, P. (St. Mary's Hosp. Med. Sch., London, England), R. L. Smith and R. T. Williams. *Biochem J* 105(3): 1283-1287, 1967.

The extent of biliary excretion of 15 sulfonamide derivatives was studied in female Wistar rats bearing bile fistulas. Most of the sulfonamides studied, with molecular weights from 172 (sulfanilamide) to 352 (N^{4}-acetylsulfadimethoxine) were poorly excreted in the bile (0-4% of the dose), except for sulfapyridine, sulfamethoxypyridazine and sulfadimethoxine. The last 3 were partly metabolized to glucuronides, whose molecular weights and polarities are such as to allow them to be excreted in the bile in appreciable amounts. Succinylsulfathiazole and phthalylsulfathiazole are polar and have molecular weights (355 and 403) of an appropriate order, and are excreted unchanged in the bile in appreciable amounts. Sulfadimethoxine N^{1}-glucuronide (molecular weight 487) is extensively excreted in the bile unchanged.

7713 **DEVELOPMENT OF GLUCONEOGENESIS IN NEONATAL RAT LIVER: EFFECT OF TRIAMCINOLONE.** *(E.)* Yeung, D. (Dept. Biochem., U. West.

Australia, Nedlands), R. S. Stanley and I. T. Oliver. *Biochem J* 105(3):1219-1227, 1967.

The normal development of the key enzymes of gluconeogenesis in rat liver, glucose 6-phosphatase, hexose diphosphatase, phosphopyruvate carboxylase and pyruvate carboxylase, was measured during the neonatal period. Glucose 6-phosphatase, hexose diphosphatase and pyruvate carboxylase were all present in the late fetal liver, but all the enzymes showed an increase in activity after birth. Phosphopyruvate carboxylase was not present in liver extracts from fetal rats, but activity appeared immediately after birth and increased rapidly over the first day and then more slowly to reach its maximum at the 4th postnatal day. When triamcinolone was administered to fetal rats at various gestation times by i.p. injection *in utero* and the animals were killed at intervals between 4 and 48 hr later, there was a slight depression of glucose 6-phosphatase and a more significant depression of hexose diphosphatase to about one-half its normal activity in fetal rat liver. Triamcinolone had no effect on pyruvate carboxylase activity and did not result in premature appearance of phosphopyruvate carboxylase in fetal rat liver. Pyruvate kinase and aspartate aminotransferase activities in fetal rat liver were both depressed by triamcinolone treatment, whereas phosphofructokinase activity was elevated. Tyrosine aminotransferase activity in fetal rat liver was markedly elevated in animals exposed to triamcinolone for 10 hr or more, but the effect was only observed in animals close to term. The results are discussed in relation to mechanisms involved in the initial synthesis of tissue-specific enzymes in developing tissues, and it is concluded that glucocorticoids do not initiate the synthesis of the gluconeogenic enzymes.

7714 **DEVELOPMENT OF GLUCONEOGENESIS IN NEONATAL RAT LIVER: EFFECT OF PREMATURE DELIVERY.** *(E.)* Yeung, D. (Dept. Biochem., U. West. Australia, Nedlands) and I. T. Oliver. *Biochem J* 105(3): 1229-1233, 1967.

An assay method for the determination of phosphopyruvate carboxylase activity is described in which improved sensitivity is obtained by separation of the enzyme from interfering pyruvate kinase by zone sedimentation. The molecular weight of rat liver phosphopyruvate carboxylase determined by zone sedimentation was about 68,000. Premature delivery of rat fetuses by uterine section resulted in the rapid appearance of phosphopyruvate carboxylase, but hexose diphosphatase and pyruvate carboxylase, already present in the fetal rat liver, were not significantly affected, and glucose 6-phosphatase activity was only slightly affected. The rate of incorporation of [^{14}C] pyruvate into glucose by liver slices was also greatly increased by premature delivery and there was a highly significant linear correlation between this process and the phosphopyruvate carboxylase activity.

7715 **BILIARY EXCRETION OF [^{14}C] SUCCINYLSULPHATHIAZOLE IN THE RAT AND RABBIT.** *(E.)* Abou-El-Makarem, M. M. (St. Mary's Hosp. Med. Sch., London, England), P. Millburn and R. L. Smith. *Biochem J* 105(3):1295-1299, 1967.

Experiments in female Wistar rats and New Zealand White rabbits, bearing bile fistulas, showed that after i.v. injection about 30% of the dose (20 mg/kg) of succinylsulfathiazole is excreted unchanged in the bile in 3 hr by the rat, whereas only about 1% is excreted by the rabbit. When the renal pedicles were ligated the biliary excretion of succinylsulfathiazole in the rat increased to about 80% of the dose, but in the rabbit under these conditions the biliary excretion was only 2% of the dose. In the rat, the sulfonamide readily enters the liver and biliary excretion occurs against a concentration gradient from liver to bile; further, the excretory process can be saturated, and can be depressed by the simultaneous administration of phenolphthalein glucuronide or bile salts. In the rabbit, these conditions were not found; succinylsulfathiazole did not readily ente the liver from the plasma, there was no transfer of the drug fro the liver cells to the bile against a concentration gradient, and no saturation or depression of the biliary excretion of succinylsulfathiazole was found. It is suggested that two factors respo sible, at least partly, for the low biliary excretion of succinylsulfathiazole in the rabbit are the poor entry of the sulfonamid into the liver in this species and a deficiency of the concentrative mechanism for its excretion in the bile.

7716 **THE EFFECT OF DIET ON CARBON TETRACHLORIDE METABOLISM.** *(E.)* Seawright, A. A. (Med. Res. Council Lab., Carshalton, Surrey, England) and A. E. M. McLean. *Biochem J* 105(3):1055-1060, 1967.

Blood and liver concentrations of CCl_4 were measured, at intervals after an oral dose, in male Wistar rats given stock and protein-free diets. The values did not correlate with the resistance to poisoning found in the rats on protein-free diets. The metabolism of CCl_4 to CO_2 *in vivo* and in liver microsomal preparations was depressed in animals given protein-free diets. Rats given a single dose of DDT [1,1,1-trichloro-2,2-bis-(p-chlorophenyl)ethane] were highly sensitive to CCl_4 poisoning. The livers of such animals had an increased microsomal protein content and greatly increased microsomal activity in the demethylation of Pyramidon (aminopyrine) and in the conversion of $^{14}CCl_4$ into $^{14}CO_2$. The incorporation of [^{14}C] leucine into protein by liver slices was also depressed by CCl_4. This effect was decreased by addition of SKF525A (2-diethylaminoethyl 2,2-diphenyl-2-propylacetate) and in slices from rats given protein-free diets. It is suggested that the toxicity of carbon tetrachloride is closely linked to its metabolism.

7717 **BILIARY EXCRETION OF FOREIGN COMPOUNDS: BENZENE AND ITS DERIVATIVES IN THE RAT.** *(E.)* Abou-El-Makarem, M. M. (St. Mary's Hosp. Med. Sch., London, England), P. Millburn, R. L. Smith and R. T. Williams. *Biochem J* 105(3):1269-1274, 1967.

The extent of the biliary excretion of benzene and 21 of its simple derivatives was studied in female Wistar rats bearing bile fistulas. Some 16 compounds of molecular weight less than 20 and including neutral molecules (benzene and toluene), aromati acids, aromatic amines and phenols, were injected i.p., after which metabolites in the bile were identified and estimated. Th extent of biliary excretion of these compounds was low, i.e. 0-10% of the dose in 24 hr, and most appeared in the bile main as conjugates. The biliary excretion of 6 conjugates of molecular weight less than 300, including 3 glycine conjugates, one sulfate conjugate, one glucuronic acid conjugate, and 2 acetyl derivatives, was low (less than 3% of the dose). It is concluded that simple benzene derivatives of molecular weight less than about 300 are poorly excreted in rat bile.

3 THE ROLE OF THE LIVER IN THE METABO-
LISM OF CHYLOMICRONS, STUDIED BY ELEC-
ON MICROSCOPIC AUTORADIOGRAPHY.

(E.) Stein, O.
dassah U. Hosp., Jerusalem, Israel) and Y. Stein. *Lab Invest*
4):436-446, 1967.

lomicrons labeled with glycerol-^{3}H, prepared by feeding
erol-^{3}H-triolein to donor thoracic duct fistula rats, were in-
ed into fed or fasted rats and their uptake by the liver was
lied for the following 5-10 min. Using electron microscopic
oradiography, the labeled chylomicrons were localized to the
soid lumen, the space of Disse, and intracellular vacuoles in
offer cells. The autoradiographic reaction present over the
enchymal liver cells was associated with endoplasmic reticulum
mitochondria, and cytoplasmic lipid droplets, up to 4 μ
liameter, not enclosed in vacuoles. It is concluded that while
offer cells take up intact chylomicrons by endocytosis, such
rocess is not evident in parenchymal cells into which the
el enters either as intact triglyceride molecules or as glycerol
ved from hydrolysis of chylomicron on the plasma mem-
ne, after which it is reesterified immediately.

9 BILIARY EXCRETION OF FOREIGN COM-
POUNDS. BIPHENYL, STILBOESTROL AND
ENOLPHTHALEIN IN THE RAT: MOLECULAR WEIGHT,
LARITY AND METABOLISM AS FACTORS IN BILIARY
CRETION.

(E.) Millburn, P. (St. Mary's Hosp. Med. Sch.,
ndon, England), R. L. Smith and R. T. Williams. *Biochem J*
(3):1275-1281, 1967.

extent of biliary excretion of biphenyl, tetralin, stilbestrol
phenolphthalein was studied in female Wistar rats bearing
fistulas. Biphenyl and its 4-hydroxy and 4,4′-dihydroxy
ivatives were extensively excreted in the bile as glucuronides
mounts increasing in order of molecular weight. Stilbestrol
its glucuronide were excreted almost quantitatively in the
, mainly as the monoglucuronide, as were phenolphthalein
its glucuronide. Tetralin was excreted to the extent of
ut 13% of the dose, mainly as *ac*-tetralyl glucuronides. The
lts and those of Abou-El-Makarem, Millburn, Smith &
liams (1967) are discussed and it is concluded that the extent
biliary excretion of foreign compounds in rats depends on
ir molecular weight and the presence of a strongly polar an-
ic group. There appears to be a minimum value of this mole-
ar weight below which little biliary excretion (i.e. not more
n 5-10% of the dose) occurs. There is some latitude in the
ice of this molecular weight, which is about 325 ± 50. The
essary molecular weight and polar group can be acquired by
tabolism. Above this minimum value, biliary excretion in-
ases with molecular weight. It is suggested that the mechanism
the biliary excretion of foreign compounds may be similar to
t of conjugated bile acids, which are highly polar and whose
lecular weights exceed 400.

20 INFLUENCE OF THYROXINE ON THE METAB-
OLISM OF ETHANOL AND GLYCEROL IN RAT
VER SLICES.

(E.) Rawat, A. K. (Inst. Biochem. A., U.
penhagen, Denmark) and F. Lundquist. *Europ J Biochem*
):13-17, 1968.

experiments on male Wistar rats, the rates of ethanol oxida-
n by liver slices from L-thyroxine treated, methyl-thiouracil
fed, and thyroidectomized animals were not significantly different from those in liver slices from normal animals. The production of lactate and pyruvate in the presence of ethanol was lower in liver slices from thyroxine-treated rats than in liver from normal and hypothyroid animals. The lactate/pyruvate ratio was not changed in hyperthyroid animals, but increased in hypothyroid rats. The rate of glycerol utilization by liver slices from normal and hypothyroid rats was inhibited about 50% in the presence of ethanol. This inhibition was not observed in thyroxine-treated rats. The concentration of α-glycerophosphate in liver slices incubated for 60 min was low in thyroxine-treated rats and significantly higher in hypothyroid rat livers. Addition of ethanol increased the concentration of α-glycerophosphate in liver slices to about twice the control values without ethanol in all groups of rats. Addition of ethanol and glycerol together produced very high concentrations of α-glycerophosphate in normal and hypothyroid but not in thyroxine treated animals. The glycerokinase activity of the liver did not change significantly after administration of thyroxine or thyroidectomy.

7721 EFFECTS OF PROFLAVINE ON ULTRASTRUCTURE AND RNA AND PROTEIN SYNTHESIS IN THE LIVER OF RATS.

(E.) Stenram, U. (Dept. Path., U. Uppsala, Sweden) and R. Willen. *Exp Cell Res* 50(3):505-514, 1968.

A combined study of the effects of proflavine on RNA and protein synthesis and the ultrastructure of the rat liver cell was performed in male Sprague-Dawley rats given 5 mg proflavine s.c. followed by 5 μC of ^{3}H-cytidine or ^{3}H-leucine. Electron microscopy revealed a decrease in size and coalescence of the nucleoli with a segregation of their ultrastructural components. Radioautography showed decreased RNA labeling over nucleoli and cytoplasm. Biochemical analysis of RNA synthesis indicated decreased synthesis of ribosomal RNA; protein synthesis showed no significant changes. It is concluded that the alterations caused by proflavine are similar to those caused by actinomycin D.

7722 EXPERIMENTAL STUDIES ON THE MECHANISM OF LIVER REGENERATION AFTER PARTIAL HEPATECTOMY IN NORMAL AND CIRRHOTIC LIVERS.

(It.) Marrano, D. (Inst. Spec. Surg. Path., U. Bologna, Italy). *Epatologia* 13(3):647-680, 1967.

The technique of Higgins and Anderson was used to perform partial hepatectomies on normal and cirrhotic albino rats. Cirrhosis was produced by daily inhalation of CCl_4 over periods ranging up to 8 months. In normal animals the volume of the remaining liver increased progressively until it reached or exceeded its preoperative weight 5-17 days after hepatectomy. The volume of the hepatic lobules did not increase, but the number of lobules did. First the portal and suprahepatic venous circulation went through a period of hemodynamic adaptation. This was followed by anatomical adaptation which consisted in an increase in the length and caliber of preexisting blood vessels and the development of new branches, particularly those of small and medium caliber. The biliary tract did not show signs of regeneration until 72 hr after surgery when parenchymal regeneration was almost complete. In precirrhotic animals with toxic liver damage regeneration after partial hepatectomy resulted in the complete disappearance of degeneration and necrosis and almost complete restitution of the parenchyma. Regeneration after hepatectomy reversed cirrhotic changes in animals with early stages of cirrhosis, but parenchymal and vascular changes were found, after partial hepatectomy symptoms of cirrhosis became more severe in animals with more advanced cirrhosis.

7723 THE EFFECT OF L(+)-ORNITHINE α-KETOGLUTARATE ON LIVER REGENERATION AND SERUM PROTEIN VALUES IN PARTIALLY HEPATECTOMIZED RATS.

(It.) Pannella, A. (Inst. Surg. Path., U. Pavia, Italy), S. Tinozzi and P. Ruozi. *Epatologia* 13(3):795-814, 1967.

The median and left lateral lobes of the liver were removed from 330 white female rats. A group of 110 were given 25 mg/100 g/day of L(+)-ornithine α-ketoglutarate i.p., and 110 others were given 50 mg/100 g/day. Untreated hepatectomized animals served as controls. The number of metaphases and anaphases in the livers of treated animals was greater than in untreated controls. More glycogen was found in the livers of treated animals, and the glycogen content was inversely proportional to karyokinetic activity. Total serum proteins were reduced and glyco- and lipoproteins increased, but to a lesser extent than in untreated animals. These results confirm previous reports that ornithine α-ketoglutarate promotes liver regeneration in hepatectomized rats.

7724 VASCULAR FACTORS IN LIVER REGENERATION.

(E.) Clarke, A. M. (U. Otago Med. Sch., Dunedin, New Zealand), R. Y. Thompson and G. J. Fraenkel. *Surg Gynec Obstet* 126(1):45-52, 1968.

The effect of arterialization of the portal vein on the rate of cell division in the regenerating liver was investigated in dogs. Partial hepatectomy (75%) was performed and portal vein arterialization was achieved by 2 procedures: 1) implantation of the splenic artery into the portal vein (15 dogs), and 2) creation of direct aorta-portal shunts with autogenous vein grafts, (9 dogs). Biopsies of the liver were obtained at the initial procedure and at laparotomy on the 3rd, 6th, and 9th postoperative day. No significant changes in the rate of cell division were noted with either shunt preparation as compared to sham-operated controls. It is concluded that an increase in tissue perfusion is unlikely to play a causative role in liver regeneration.

7725 EFFECT OF LARGE DOSES OF THE ORAL CONTRACEPTIVE ENOVID ON CHOLESTEROL METABOLISM IN THE RAT.

(E.) Aftergood, L. (Sch. Publ. Health, U. California, Los Angeles), H. J. Hernandez and R. B. Alfin-Slater. *J Lipid Res* 9(4):447-452, 1968.

The effects of oral administration of large doses of Enovid (1 mg/day of a mixture of norethynodrel and mestranol in a ratio of 250:1 for 7 days) were studied in adult male and female rats. At the end of the 7-day period, marked decreases in plasma cholesterol esters were noted accompanied by only slight elevations of hepatic cholesterol; cholesterol esters were also lower in the adrenals and ovaries while cholesterol-esterifying activity in the plasma was substantially increased. Enovid administration also affected the fatty acid composition of sterol esters remaining in the plasma, adrenals and ovaries: the concentration of linoleate and arachidonate was significantly decreased in plasma sterol esters, while the concentrations of arachidonate and docosatetraenoate in the adrenals and of docosatetraenoate in the ovaries were significantly lowered. All o these changes were more pronounced in the females than in the males. It is hypothesized that the decreased levels of cholestero ester found in the organs investigated, together with the increas in plasma cholesterol-esterifying activity, are associated with either changes in cholesterol biosynthesis and/or transport or with an increase in cholesterol excretion and transformation to other steroids.

7726 HEPATIC TRIGLYCERIDE SECRETION IN RELATION TO LIPOGENESIS AND FREE FATTY ACID MOBILIZATION IN FASTED AND GLUCOSE-FED RATS.

(E.) Baker, N. (VA Ctr., Los Angeles, Calif.), A. S. Garfinkel and M. C. Schotz. *J Lipid Res* 9(1):1-7, 1968.

Plasma triglyceride and free fatty acid concentrations were determined after feeding 2 ml 50% glucose to male Sprague-Dawle rats after a 22-hr fast. Acetate-^{14}C and glucose-^{14}C were also used to measure lipogenesis in liver slices, plasma free fatty acid concentrations and free fatty acid release from isolated epididymal fat pads, and Triton-induced hypertriglyceridemia. In rats receiving 3 feedings of glucose or water 1 hr apart, circulating triglyceride levels were 42% and 70%, resp., of those found in fasted controls. In the glucose-refed rats mobilization of free fatty acids from adipose tissue was impaired more rapidly than hepatic lipogenesis was restored from its low fasting level, indicated by a 50% fall in plasma free fatty acid concentration and an 84% decrease in free fatty acid release by isolated epididymal fat pads within 30 min after a single refeeding of glucose, whereas hepatic lipogenesis from either acetate-^{14}C or glucose-^{14}C was not restored even after 3 feedings of glucose at hourly intervals. The rate of Triton-induced hypertriglyceridemia was reduced after glucose refeeding in all but 1 experiment. This decreased secretion rate was sufficient to account for the nearly complete disappearance of triglyceride in very low density lipoproteins that occurred within 1 hr after glucose refeeding. It is concluded that refeeding a large load of glucose to fasted rats usually reduces the rate of hepatic triglyceride secretion promptly.

7727 QUANTITATIVE ASPECTS OF FREE FATTY ACID METABOLISM IN THE FASTED RAT.

(E.) Baker, N. (VA Ctr., Los Angeles, Calif.) and M. C. Schotz. *J Lipid Res* 8(6):646-660, 1967.

Palmitate-1-^{14}C was injected i.v. into unanesthetized, fasted rat and the disappearance of the tracer from plasma free fatty acid was studied. A large component of free fatty acid recycling wa directly demonstrated by reinjection experiments. The latter studies also indicated the existence of an unidentified, rapidly turning-over polar lipid in plasma which was synthesized from palmitate-^{14}C. The appearance of ^{14}C in hepatic and extrahepatic triglycerides, in other esters, and in respired CO_2 was also followed. The data were analyzed using a multicompartmental model and a digital computer. Only a small fraction of the triglycerides formed in liver was derived directly from plas free fatty acids. The major portion of net triglyceride formatio appeared to be by way of an intermediate nontriglyceride ester ol which turned over relatively slowly compared to plasma e fatty acids. Initial approximations were as follows (μmoles fatty acid/min/100 g body weight): net free fatty acid mo- zation (irreversible disposal) = 2.4; hepatic triglyceride forma- n directly from plasma free fatty acids = 0.1; total hepatic d formation from plasma free fatty acids = 0.5; oxidation of e fatty acids to CO_2 = 0.8; percentage of respired CO_2 from ect oxidation of fatty acids = 12%; extrahepatic triglyceride mation directly from fatty acids = 0.4; and total extrahepatic d formed directly from fatty acids = 1.2.

28 **EFFECT OF p-CHLOROPHENOXYISOBUTYRATE (CPIB) FED TO RATS ON HEPATIC BIOSYN- ESIS AND CATABOLISM OF UBIQUINONE.** *(E.)* kshmanan, M. R. (Food Drug Direct., Dept. Natl. Health Wel- e, Ottawa, Canada), W. E. J. Phillips and R. L. Brien. *J Lipid s* 9(3):353-356, 1968.

male Wistar rats fed a basal diet with or without 0.2% CPIB /w), and receiving either 10 μC of acetate-1-^{14}C after 15 days the diet or 2 μC of mevalonate-2-^{14}C after 35 days of the t, liver ubiquinone, hydrocarbon, and sterol content was de- mined 4, 24 and 72 hr after the tracer dose was given. *In ro* studies were also performed by incubating the radioactive cers with liver homogenates from rats maintained on the test ts for 21 or 150 days. In the *in vivo* experiments at the 4- interval, the results of acetate-1-^{14}C incorporation showed 2% inhibition of sterol specific activity in the CPIB-fed rats compared to controls; the incorporation into ubiquinone and drocarbon fractions was too low to be measured up to 72 hr. e rate of decrease in the specific activity of the liver sterols the 2 later time intervals was lower for the CPIB-fed group n in the controls. The results of mevalonate-2-^{14}C incorpora- n showed a consistently higher specific activity of the sterol ction in the CPIB-fed group at all time intervals. However, rate of fall in the 2 groups was similar up to 72 hr. The cific activity in the ubiquinone fraction was considerably ver than in the normal group at 4 hr, but significantly higher 24 and 72 hr. The specific activity in the hydrocarbon frac- n in CPIB-fed rats, although higher at 4 and 24 hr, dropped nost to the same level as in the controls after 72 hr. The *in ro* experiments confirmed the results of the *in vivo* experi- ents; it was noted that older rats showed lower incorporation es than younger rats with acetate-1-^{14}C. These results in- ate that CPIB feeding causes a block in the conversion of etate to mevalonate, thereby inhibiting over-all steroidogenesis. though liver ubiquinone synthesis was inhibited by CPIB, a ock in the catabolism of ubiquinone resulted in accumulation ubiquinone in CPIB-fed rats.

'29 **CHOLINE-DEFICIENCY FATTY LIVER: IM- PAIRED RELEASE OF HEPATIC TRIGLYCER- ES.** *(E.)* Lombardi, B. (U. Pittsburgh Sch. Med., Pa.), P. Pani d F. F. Schlunk. *J Lipid Res* 9(4):437-446, 1968.

ter i.v. injection of palmitate-1-^{14}C to rats fed a choline- ficient diet (CD) or choline-supplemented diet (CS) for 15- hr, liver triglycerides became labeled very rapidly (within min). In CS but not in CD rats, there was a considerable s, with time, of radioactivity from liver triglycerides. At the ne time, significantly less radioactivity appeared in plasma glycerides of CD rats than of CS animals. No difference was seen in the triglyceride content of microsomes isolated from the liver of rats fed the 2 diets. The lower radioactivity of CD rats was essentially due to a lower level and specific activity of very low density lipoprotein triglycerides. After i.v. injection of Triton and labeled palmitate, considerably less radioactivity accumulated in plasma triglycerides and phospholipids of CD rats than of CS animals. Post-Triton hyperphospholipidemia was also less pronounced in CD rats. It is concluded that the fatty liver observed in CD rats results from an impaired release of hepatic triglycerides into plasma.

7730 **EFFECT OF SPLENO-PORTAL ARTERIOVENOUS ANASTOMOSIS UPON HEPATIC REGENERATION AFTER PARTIAL HEPATECTOMY IN THE DOG.** *(E.)* Mavrantonis, S. (Sch. Med., U. Athens, Greece). *Bull Soc Int Chir* 27(3):183-190, 1968.

An arterio-portal shunt was performed simultaneously with 70% hepatectomy in 14 dogs by implanting the splenic artery into the portal vein. Autopsy 35-118 days following surgery showed patency of the shunt in 6 of 10 animals which survived the operation. The rate of hepatic regeneration was significantly higher in animals with a patent spleno-portal shunt (116 ± 8%) than in 4 dogs with occlusion of the shunt (82 ± 10%) or in 2 control dogs with 70% resection alone (74%). Except for scanty lymphocytic infiltration in the connective tissue and periportal spaces, some swelling of the cells and sinusoidal congestion, no significant histological differences were observed between the previously resected part of the liver and the regenerated tissue. In 1 animal, histologic changes of subacute interstitial hepatitis noted at hepatectomy disappeared following arterialization. In contrast, histologic changes of interstitial hepatitis, developing into cirrhosis, were observed in 1 dog with an obstructed shunt. One control animal developed areas of steatosis and the other showed slight fibrosis in the periportal and perilobular spaces. Portal pressure was 90 mm H_2O at laparotomy, 140 after 70% hepatectomy and 192 after arterio-portal shunt. Following a temporary increase in serum alkaline phosphatase level, normal levels were achieved 30-40 days postoperatively. Other liver function tests (BSP, serum bilirubin, cephalin flocculation, thymol turbidity, serum total proteins, prothrombin time) were not significantly altered after surgery. These findings suggest that spleno-portal anastomosis may find clinical application in cases of extensive trauma of the liver, in conditions necessitating major hepatectomy, and in the treatment of hepatic cirrhosis.

7731 **POLYPLOIDIZATION OF LIVER AFTER PARTIAL HEPATECTOMY IN THE DWARF MOUSE AND HYPOPHYSECTOMIZED RAT: EFFECT OF EXTENDED REGENERATIVE PERIODS.** *(E.)* Swartz, F. J. (U. Louisville Sch. Med., Ky.). *Exp Cell Res* 48(3):557-568, 1967.

Experiments with normal and pituitary-deficient mice and rats showed that the elevated levels of hepatic polyploid nuclei following partial hepatectomy largely disappear if minimal amounts of liver are removed and extended regenerative periods are permitted. In the normal rat, all excess polyploid classes disappear while in the hypophysectomized rat the percentage of polyploid nuclei remains below normal. The liver of the normal mouse shows a strong tendency for equilibration while that of the dwarf mouse remains in a state of "excess" polyploidization for at least a month after partial hepatectomy.

It is concluded that excess polyploid classes in the rat are not permitted permanently either in the presence or absence of the pituitary. A species difference is noted in the mouse, in that initial polyploid levels are higher and response to partial hepatectomy greater. The increased response to hepatectomy in the dwarf mouse liver is attributed to its greater potential for growth than the livers of the other experimental groups. It is assumed that equilibration in the dwarf mouse liver would have also occurred with time. The pituitary is not required for polyploidization during hepatic regeneration, but indirectly controls the general level of polyploidy through its control over organ growth. Other factors probably also come into play.

7732

CARBOHYDRATE SUPPLY AS A REGULATOR OF RAT LIVER PHOSPHOENOLPYRUVATE CARBOXYKINASE ACTIVITY. *(E.)* Shrago, E. (Inst. Enzyme Res., U. Wisconsin, Madison), J. W. Young and H. A. Lardy. *Science* 158(3808):1572-1753, 1967.

Administration of glucose, fructose, and glycerol to fasted rats produced a significant depression of liver phosphoenolpyruvate carboxykinase activity within 4-8 hr; galactose and ribose were much less effective. All these compounds yielded appreciable amounts of liver glycogen. The depression of phosphoenolpyruvate carboxykinase activity by glucose and glycerol was diminished by the concomitant administration of 2-deoxyglucose. The latter depressed glycogen formation from administered carbohydrate in muscle but not in liver. In rats made diabetic by alloxan, depression of elevated phosphoenolpyruvate carboxykinase activity by insulin was dependent upon a dietary source of carbohydrate. These results indicate that depression of certain gluconeogenic enzymes after carbohydrate ingestion is initiated by the metabolism of carbohydrate in some extrahepatic site.

7733

REGENERATION OF RAT LIVER: TRANSFER OF HUMORAL AGENT BY CROSS CIRCULATION. *(E.)* Moolten, F. L. (Massachusetts Gen. Hosp., Boston) and N. L. R. Bucher. *Science* 158(3798):272-274, 1967.

Carotid-to-jugular cross circulation between partially hepatectomized and normal Charles River female rats (37-42 days old), via polyethylene cannulas, stimulated incorporation of ^{14}C-thymidine into hepatic DNA in the normal partners when it was maintained for 19 hr at a flow rate of 2 ml/min. Cross circulation for 7 hr or less was ineffective. Cross circulation between normal rats produced only a low level of labeling of hepatic DNA. These findings suggest that control of liver regeneration resides in a humoral mechanism responsive to changes in effective liver mass.

7734

ANTIGEN DEVELOPMENT IN NEONATAL RAT LIVER. *(E.)* Raftell, M. (Wenner-Gren Inst., U. Stockholm, Sweden) and P. Perlmann. *Exp Cell Res* 49(2):317-331, 1968.

The antigens in rat liver between the 2nd and 15th day of postnatal development were studied using the antigen pattern of adult parenchymal liver cells and their subfractions as a reference. Detergent extracts of total homogenates and microsomal and cell sap fractions were analyzed for soluble antigens in gel diffusion experiments using rabbit antisera prepared against adult parenchymal cells or cell fractions and against total homogenates of 2-day-old liver. The localization of the antigens was also studied by fluorescent antibody staining of liver sections. Many of the adult-type antigens present in tota homogenates or cell sap were also present in the correspondin fractions of 2-day-old liver. In contrast, only 3 of 10 antigen seen in adult parenchymal microsomes were detected in microsomes of 2-day-old liver. The remaining microsomal antigens appeared progressively with development. When the immune precipitates were stained for enzyme activity, it was found th nonspecific esterases, chymotrypsin-like proteases and acid phosphatases were almost completely absent in 2-day-old liver extracts. Antigens with these activities appeared progressively and at different rates in the different fractions during the 1st 10 days of postnatal development. However, 2-day-old liver microsomes also contained an acid phosphatase, the activity of which decreased sharply with development. Fluorescent antibody staining of adult liver with serum prepared against parenchymal homogenate yielded cytoplasmic staining of the parenchymal cells. With this serum, 10-day-old liver showed nuclear fluorescence all over the sections. Sections of 2-day-old liver showed strong cytoplasmic staining of scattered grou of hematopoietic cells. These were also stained by serum prepared against 2-day-old liver, which stained sections of 10-day-old and adult livers only weakly. This suggests that antigens of the hematopoietic cells in 2-day-old liver are present in the parenchymal cells of adult liver as well. In contrast, other antigens typical for adult parenchymal cells seem to be absent in the neonatal liver. This confirms and extends the results obtained with agar-diffusion techniques.

7735

SYNTHESIS OF STAGE SPECIFIC SERUM PROTEINS AND TISSUE ANTIGENS BY EARI NEONATAL RAT LIVER. *(E.)* Raftell, M. (Wenner-Gren Inst., U. Stockholm, Sweden) and P. Perlmann. *Exp Cell Res* 49(2):332-339, 1968.

Two-day-old rat sucklings were injected with ^{14}C-labeled L-leucine, and after 1 hr of incubation a total homogenate was prepared of their livers. The incorporation of amino acids int liver and serum proteins was then analyzed by means of immu electrophoresis and double diffusion techniques in combinatio with autoradiography. Many of the serum proteins present in adult rat serum were also found in serum of 2-day-old rats. These serum proteins were actively synthesized by the neonat rats. The results suggested that some of the secretory functio typical of parenchymal cells of adult liver are already well developed at birth. However, serum from 2-day-old rats also co tained at least 4 antigens which were present neither in adult rat liver nor in serum. At least 1 of these was detected in serum of adult rats 24 hr after hepatectomy. All these components were actively synthesized by 2-day-old rats, but synthesis ceased rapidly as postnatal development proceeded. Ea postnatal liver also contained several (3-4) soluble tissue antig absent from serum and not detectable in livers from normal adults nor from hepatectomized animals. The biochemical nature and cellular origin of these antigens remains unknown.

7736

CELL PROLIFERATION IN THE REGENERATING LIVER AND THE EFFECT OF PRIOR CONTINUOUS IRRADIATION. *(E.)* Fabrikant, J. I. (Inst. Cancer Res., Surrey, England). *Radiat Res* 32(4):804-826, 196

Male August rats were exposed to 47 rads/day for up to 30 days in a study of the temporal patterns of proliferation in

parenchymal and littoral cell populations at frequent inter-
during the first 64 hr following partial hepatectomy. The
niques used involved the *in vivo* labeling of DNA with tri-
ed thymidine and high resolution autoradiography. Analysis
he cell population kinetics indicated that the rate of entry
parenchymal cells into synthesis (after an initial burst of
liferative activity) was an orderly phenomenon at 3-4%/hr.
st cells divided once, and a few twice, a large proportion
he cell deficit being replaced by 72 hr after the onset of
proliferation. The duration of the S period was 8 hr, and
of the G_2 + M/2 complex, approximately 3 hr. Littoral
proliferation was a response to growth of the parenchymal
population and the mean rate of entry of new cells into
thesis was greater than 4%/hr. Following continuous expo-
e at 47 rads/day, cell proliferation could be initiated and
ntained, albeit at a somewhat lower level, despite the pres-
e of large numbers of radiation-induced chromosome aber-
ons in parenchymal cells. There was a delay in the onset
DNA synthesis and mitosis, a decrease in the rate of entry
cells into synthesis and division, a slight speeding up of
enchymal cells through the S and G_2 + M/2 compartments,
ecrease in the number of cells taking part in the regenerative
cess, an increase in the overall period of cell proliferation,
a possible increase in the numbers of polyploid hepatocytes.
e number of radiation-induced chromosome aberrations was
endent on the duration of exposure and the accumulated
e. Despite the accumulation of radiation damage, viable
s in the liver remnant could still divide a few times to re-
ce the greatest proportion of the parenchymal and littoral
deficits within approximately 1 week.

37 NICKEL CARBONYL INHIBITION OF CORTISONE INDUCTION OF HEPATIC YPTOPHAN PYRROLASE. *(E.)*

Sunderman, F. W. Jr.
Florida Coll. Med., Gainesville). *Cancer Res* 27(9):1595-
99, 1967.

e effects of nickel carbonyl on induction of hepatic trypto-
n pyrrolase by cortisone or tryptophan was studied in non-
enalectomized, male, Sprague-Dawley Dublin Farms rats (3
nths old; 180-220 g) and in adrenalectomized, male, Sprague-
wley Charles River Farms rats (1 month old; 70-90 g). The
mals were given i.p. injections of either 4 ml L-tryptophan
25 M) in 0.9% NaCl at 0 and 3 hr, 10 mg cortisone acetate
1 ml 0.9% NaCl at 0 and 3 hr, or no injections, and were
rificed 6 hr after the initial injection. Nonadrenalectomized
perimental rats were injected i.v. with nickel carbonyl (2 mg
100 g body weight) at either 24, 48, 72, 120 or 168 hr be-
e sacrifice; adrenalectomized experimental rats were injected
hr before sacrifice only. Although nickel carbonyl inhibited
cortisone-stimulated induction of hepatic tryptophan pyrro-
e in both adrenalectomized and intact rats, it did not inhibit
ptophan induction. The average hepatic tryptophan pyrro-
e activity in cortisone-treated control rats was 36 ± 7 U,
mpared with 20 ± 4 U in similar rats receiving nickel car-
nyl 48 hr before sacrifice. Nickel carbonyl did not inhibit
ptophan pyrrolase *in vitro*. These findings suggest that the
ic effect of nickel carbonyl on enzyme induction is mediated
her by diminished synthesis or by denaturation of messenger
A.

738 FRUCTOKINASE, TRIOKINASE, AND ALDOLASES IN LIVER TUMORS OF THE RAT. *(E.)*

Adelman, R. C. (Temple U. Sch. Med., Philadelphia, Pa.), H. P. Morris and S. Weinhouse. *Cancer Res* 27(12):2408-2413, 1967.

Using newly developed spectrophotometric procedures, 3 enzymes involved in fructose metabolism were assayed in normal rat liver, regenerating liver, and a series of primary and transplantable liver tumors ranging widely in growth rate and degree of differentiation. Fructokinase was present in all, and triokinase (adenosine triphosphate: D-glyceraldehyde 3-phosphotransferase) was present in all except 3 of 14 transplantable, well-differentiated, slow-growing hepatic tumors, induced by chemical carcinogenesis. Both enzymes were also present in 6 primary, well-differentiated hepatocellular carcinomas induced by feeding 3′-methyl-dimethylaminoazobenzene. However, their levels were one third to one tenth those of normal liver. Comparison of aldolase activities toward fructose diphosphate and fructose-1-diphosphate indicated that the hepatic-type aldolase was retained largely or completely in the well-differentiated tumors, but was replaced largely or completely by the nonhepatic aldolase in the poorly differentiated tumors. Regenerating liver 24, 48 and 72 hr after partial hepatectomy retained the normal liver pattern of these 3 enzymes, although the activities were markedly decreased. Hepatic patterns of the 3 enzymes were retained in all of the well-differentiated tumors, despite moderate alterations in the moderate alterations in the chromosome number and the karyotype. However, marked changes in these chromosome characteristics were associated with loss in the hepatic enzyme pattern.

7739 SOME PROPERTIES OF β-GLUCURONIDASE ACTIVITY IN NORMAL RAT LIVER AND IN SEVERAL TRANSPLANTABLE RAT HEPATOMAS. *(E.)*

Wagner, R. L. (Div. Biol. Sci., U. Connecticut, Storrs) and J. S. Roth. *Cancer Res* 27(11):2053-2059, 1967.

The distribution and activity of β-glucuronidase in several transplantable tumors were compared with those in normal rat liver. In all the tumors studied, specific enzyme activity ranged from 20 to 50% that of liver. In rat liver, 5% of the total enzyme activity was recovered in the high-speed supernatant fraction, while in the tumors it ranged from 11 to 38%. The distribution of free and total enzyme in the four subcellular fractions of the Morris 5123 D and Novikoff hepatoma was also compared to rat liver. In addition to increased activity in the supernatant fraction, both tumors had a lowered percentage of total activity in the microsome fraction. The Novikoff hepatoma, in addition, had increased activity in the nuclear fraction and a decrease in the mitochondrial fraction. *In vitro* experiments in which mitochondrial fractions or homogenates were incubated or treated with various chemicals suggested that the stability of those hepatoma lysosomes studied varied markedly and also differed from that of normal rat liver.

7740 INTERACTION BETWEEN ETHIONINE AND RAT LIVER RIBONUCLEIC ACID AND PROTEIN *IN VIVO*. *(E.)*

Farber, E. (U. Pittsburgh Sch. Med., Penna.), J. McConomy, B. Franzen, F. Marroquin, G. A. Stewart and P. N. Magee. *Cancer Res* 27(10):1761-1772, 1967.

The interactions of ethionine and methionine with RNA and protein fractions were studied in acute experiments and under conditions leading to liver cancer. Following injection of L-ethionine-ethyl-1-^{14}C into rats, radioactivity was regularly found in RNA and protein of liver, pancreas, kidney, and spleen, but the highest by far was in liver RNA and pancreatic protein. The pattern of labeling was different than that following the injection of methionine-methyl-^{14}C, glycine, serine, acetate (C_1 and C_2), or ethanol (C_1 and C_2). The labeling pattern of the total liver RNA and protein was similar with L-ethionine-ethyl-1-^{14}C and L-ethionine-ethyl-1-^{3}H and different with L-ethionine-^{35}S. The most radioactive fractions of the liver following 7 daily injections of radioactive ethionine were soluble RNA and nuclear protein. Of the nuclear protein fractions studied, the highest specific activity was found in a fraction soluble in 0.14 NaCl. In contrast to ethionine, the injection of $^{14}CCl_4$ labeled total liver RNA to an insignificant degree and liver protein to a small degree. Acid hydrolysis of liver RNA labeled with L-ethionine-ethyl-1-^{14}C followed by chromatography on Dowex 50 revealed at least 4 peaks of radioactivity, only 1 of which (uridylic acid) coincided with a peak of ultraviolet absorption. By means of paper chromatography and paper electrophoresis, it was possible to separate all the radioactive components from ultraviolet-absorbing compounds. The labeling of RNA and protein fractions with radioactive ethionine or methionine were compared in animals fed control diets or diets containing ethionine, ethionine-methionine, or methionine for periods of 1, 2, 3, or 4 months. The patterns were generally similar at each of these time periods. The labeling of animals on the ethionine-methionine diet was, in most instances, less than in animals on the ethionine diet, but a considerable degree of labeling always occurred in the former group, even though they did not develop liver cancer. The possible relation between the labeling of RNA and protein and liver carcinogenesis is discussed.

7741 **BILIARY EXCRETION OF THE O-GLUCURONIDE OF N-HYDROXY-2-ACETYLAMINOFLUORENE BY THE RAT AND RABBIT.** *(E.)* Irving, C. C. (VA Hosp., Memphis, Tenn.), R. Wiseman Jr. and J. T. Hill. *Cancer Res* 27(12):2309-2317, 1967.

Quantitative data were obtained on the excretion of the O-glucuronide of N-hydroxy-2-acetylaminofluorene after administration of single doses of 2-acetylaminofluorene or of N-hydroxy-2-acetylaminofluorene to the rat and rabbit. Following administration of either 2-acetylaminofluorene-9-^{14}C or N-hydroxy-2-acetylaminofluorene-9-^{14}C to rats, approximately 50% of the radioactivity was excreted in the bile in 24 hr; 10.2% of the dose of 2-acetylaminofluorene-9-^{14}C and 21.1% of the dose of N-hydroxy-2-acetylaminofluorene-9-^{14}C were excreted in the bile in 24 hr as the O-glucuronide of N-hydroxy-2-acetylaminofluorene-9-^{14}C. Glucuronides of 3-, 5-, and 7-hydroxy-2-acetylaminofluorene-9-^{14}C and the sulfate of 7-hydroxy-2-acetylaminofluorene-9-^{14}C were virtually absent in rat bile after the administration of N-hydroxy-2-acetylaminofluorene-9-^{14}C. This was in marked contrast to the results obtained after administration of 2-acetylaminofluorene-9-^{14}C to the rat and suggests that the formation of the O-glucuronide of N-hydroxy-2-acetylaminofluorene is the major metabolic reaction of N-hydroxy-2-acetylaminofluorene in rat liver *in vivo* rather than its reduction or deacetylation. Even though appreciable amounts of the O-glucuronide of N-hydroxy-2-acetylaminofluorene were excreted in the urine of rabbits given either 2-acetylaminofluorene or N-hydroxy-2-acetylaminofluorene, significant amounts of this metabolite were not present in the bile of rabbits following the administration of 2-acetylaminofluorene or N-hydroxy-2-acetylaminofluorene. Only small amounts of other metabolites of these two compounds were excreted in rabbit bile. After administration of 2-acetylaminofluorene-9-^{14}C, an average of 5.7% of the radioactivity was excreted in rabbit bile in 24 hr. The relevance of these studies to the carcinogenicity of 2-acetylaminofluorene and N-hydroxy-2-acetylaminofluorene in the rat and the rabbit is considered.

7742 **PATTERNS OF BILIRUBIN CONJUGATION IN THE NEWBORN.** *(E.)* Jacobsen, J. (Dept. Biochem. A, U. Copenhagen, Denmark), R. Brodersen and D Trolle. *Scand J Clin Lab Invest* 20:249-251, 1967.

Concentrations of unconjugated bilirubin and bilirubin diglucuronide (measured as alkali-hydrolyzable bilirubin conjugate were determined in serum from umbilical cord blood and cap lary blood, taken daily during the 1st 5 days after birth in 4 healthy mature infants. Bilirubin diglucuronide concentratio were higher than adult values in all infants, probably indicati inhibition of excretion. In most infants, unconjugated biliru concentrations were high, possibly the result of slow irreversi conjugation, or, if an equilibrium of conjugation is present, t result of the elevated concentration of conjugate. In the 3 cases which showed aberrant patterns, unconjugated bilirubin was lower than the rest of the series, being in the adult rang this may possibly be due to an early development of the conjugating enzyme or to a high uridine diphosphoglucuronic acid/uridine diphosphate ratio in the liver.

7743 **BILIRUBIN CONJUGATION IN THE HUMAN FETUS.** *(E.)* Brodersen, R. (Protein Lab., U. Copenhagen, Denmark), J. Jacobsen, H. Hertz, H. Rebbe and B. Sorensen. *Scand J Clin Lab Invest* 20(1):41-48, 1967.

Quantitative determinations of bilirubin (unconjugated, by chloroform extraction), bilirubin diglucuronide (by the isotop derivative method), and albumin were performed in 26 specimens of amniotic fluid from cases suspected of hemolytic disease *in utero*. A hypothetical scheme of fetal bilirubin metabolism is proposed, presuming unidirectional transplacen elimination of unconjugated bilirubin. On the basis of theoretical considerations of the mechanism of conjugation, it is thought possible that bilirubin in the fetal liver is in chemical equilibrium with its diglucuronide. Both pigments are in equ librium with the pigments in the amniotic fluid. Equilibratio takes place through the fetal plasma. The ratio of the conce trations of bilirubin and bilirubin diglucuronide in the amnio fluid is determined by the following 3 factors: the relative affinities of the binding of the 2 pigments to albumin, the ra of the concentrations of uridine diphosphoglucuronic acid an uridine diphosphate (UDPGA/UDP) in the liver, and the stan ard free energy of conjugation. The slow glycogen synthesis in the fetal liver may be responsible for a high UDPGA/UDP ratio and hence for a high proportion of conjugated bilirubin in the amniotic fluid. The experimental findings were in agreement with this hypothesis, offering an alternative to the generally accepted concept of low transferase activity and impaired hepatic excretion of conjugate before and shortly afte birth.

7744 **TRYPTOPHAN AND TYROSINE LOADING I DANBOLT'S ACRODERMATITIS ENTEROPATHICA.** *(Ger.)* Feuerstein, M. (Dermatol. Clin., Friedrich Schiller U., Jena, Germany) and N. Sönnichsen. *Klin Wschr* 45(20):1055-1056, 1967.

ncentrations of tryptophan and its metabolites were deter-
ned in 24-hr urine samples from 3 children with acroderma-
is enteropathica and from the parents of 1 patient before
d after oral loading with 0.1 g/kg of tryptophan and in nor-
l controls before and after loading with hydroxyquinoline.
patients with acrodermatitis enteropathica urinary excretion
kynurenin, kynurenic acid, xanthurenic acid, and 3-hydroxy-
thranilic acid was in the lower normal range. Indican excre-
n was abnormally low, probably because of long-term hy-
oxyquinoline therapy. The results of this study and a pre-
us study of tyrosine loading indicate that oxidative break-
wn of amino acids in the liver is reduced in acrodermatitis
teropathica.

745 **PROTEIN CONSTITUENTS OF BILE FROM THE RABBIT GALLBLADDER IN RELATION**
THE SPECIFIC SYNTHETIC ACTIVITY OF THE BILIARY
ITHELIUM. *(Fr.)* Chişiu, N. S. (Dr. I. Cantacuzino Hosp.,
icharest, Romania). *Rev Roum Med Intern* 4(6):433-438,
67.

le, taken from the gallbladders of 100 rabbits, was subjected
agar immunoelectrophoresis and the various fractions studied
th horse and pigeon anti-bile immune sera conjugated with
iorescein-isothiocyanate. Fractions identified included serum
oumin, IgG, IgA, β_1- and β_2-globulins, α_2-globulins, and at
ist 2 specific bile antigens. The bile antigen present in the
rgest quantity moved parallel to the IgA fraction but was
finitely separate from it. Using conjugated anti-bile sera
iich had first been incubated with excess rabbit blood serum,
ese bile antigens were found only in cells of the biliary mu-
sa where they appeared as fluorescent granules. This is
terpreted as indicating that at least some specific bile antigens
e synthesized in the biliary tract mucosa.

746 **ISOCITRIC DEHYDROGENASE ACTIVITY IN THE IMMATURE LIVER (ITS RELATIONS**
O NEONATAL ACIDOSIS). *(It.)* Cordone, G. (G. Gaslini
ediat. Clin., U. Genoa, Italy), G. Gemme and R. Gatti.
inerva Pediat 19(27):2336-2338, 1967.

he activity of isocitric dehydrogenase was determined in
aterial obtained by needle biopsy of the liver shortly after
eath in 36 infants. These infants (18 males and 17 females)
ere born between 5.5 and 9 months after conception and all
ut 1 had birth weights under 2500 g. Most of these infants
ed of complications of prematurity. Enzyme activities were
ormal in almost all cases, but this may be because they were
etermined under optimum experimental conditions which are
ot necessarily present *in vivo*. However, it is unlikely that iso-
tric acid or other acids of the Krebs cycle are responsible
r physiological acidosis in the newborn.

747 **THE EFFECT OF A MODIFIER OF CAPILLARY PERMEABILITY, BENZARONE, ON BILIARY**
UNCTION IN THE RAT. *(Fr.)* Vanlerenberghe, J. (Fac.
led. Pharm., Lille, France), N. Trupin, C. Bel, R. Cassaigne
nd L. Adenis. *Therapie* 22(5):1021-1028, 1967.

enzarone, in doses of 10, 25, and 50 mg, was added to 90
l of medium with which rat livers were being perfused. A
ioderate decrease in bile secretion which lasted for 30 min
ccurred with 10 mg of benzarone; larger doses caused greater and more long-lasting decreases. At doses of 25 and 50 mg the content of dry solids was higher than in the controls. Benzarone reduced the concentrations of bilirubin and iodine in the bile, but had little effect on cloride and reducing sugar concentrations. The concentration and output of BSP and neutral red were significantly reduced by benzarone. These effects can be attributed either to the enzyme-inhibiting action of benzarone or to its reduction of capillary permeability.

7748 **EFFECT OF ACETYLSALICYLIC ACID ON THE LIVER RESPONSE TO ENDOTOXIN.** *(E.)* Solomon, L. A. (U. Oklahoma Med. Ctr., Oklahoma City) and L. B. Hinshaw. *Proc Soc Exp Biol Med* 127(1):332-335, 1968.

The mechanism of the canine liver response to endotoxin was studied in experiments on an *in situ* liver preparation with controlled hepatic arterial and portal venous blood flows. In 12 dogs, acetylsalicylic acid (50 mg/kg) dissolved in 90% ethanol (150 mg/ml) was administered i.v., followed in 7 dogs by and LD_{80} dose of endotoxin (0.4 mg/kg); 5 animals received no endotoxin and served as controls, while in 7 other animals, alcohol and saline were administered i.v. followed by endotoxin. Administration of acetylsalicylic acid prior to endotoxin prevented the decrease in mean systemic arterial pressure and obliterated the constriction observed in both the hepatic artery and hepatic vein in animals given endotoxin alone. The mechanism of this action of acetylsalicylic acid is unknown.

7749 **RELATION OF ETHANOL INHIBITION OF HEPATIC FATTY ACID OXIDATION TO ETHANOL-INDUCED FATTY LIVER.** *(E.)* Zakim, D. (Fitzsimons Gen. Hosp., Denver, Colo.) and J. Green. *Proc Soc Exp Biol Med* 127(1):138-142, 1968.

The quantitative importance of the inhibition of hepatic fatty acid oxidation for the pathogenesis of alcoholic steatosis was investigated in perfused rat livers. Since octanoate is known to be poorly esterified in rat liver, by using octanoate the effects of ethanol on hepatic fatty acid esterification could be separated from effects on fatty acid oxidation. Comparison of the effect of ethanol perfusion on the metabolism of octanoate-(1-^{14}C) and palmitate-(1-^{14}C) indicated that ethanol decreased fatty acid oxidation to CO_2 for both acids. However, in contrast to the palmitate data, inhibition of the oxidation of octanoate to CO_2 did not lead to an increase in the recovery of ^{14}C from octanoate in the liver. Ethanol inhibition of CO_2 production from fatty acids did not lead *per se* to an accumulation of fatty acids in the liver. Thus, the data suggest that ethanol inhibition of CO_2 production from fatty acids does not contribute to the accumulation of fatty acids (in the form of triglycerides) seen in livers from ethanol-treated rats.

7750 **ORTHOPTIC LIVER HOMOTRANSPLANTATION IN THE DOG. IMMUNOSUPPRESSIVE THERAPY WITH ANTILYMPHOCYTE SERUM.** *(Fr.)* Mikaeloff, P. (Fac. Med., Lyon, France), R. Pichlmayr, J. P. Rassat, K. Messmer, J. Bomel, G. Tidow, M. Étiennemartin, J. Malluret, P. Belleville, A. Jouvenceau, J. Falconnet, J. Descotes and W. Brendel. *Presse Med* 75(40):1967-1970, 1967.

Antilymphocyte serum (0.5 ml/kg b.i.d.), prepared by immunizing horses with dog lymphocytes, was given to 10 dogs who had undergone orthoptic liver transplantation performed by Starzl's procedure. In all but 2 of these animals therapy was begun 4 days before surgery. Complications occurring after surgery included intussusception (3 cases), intercurrent infections (4 cases), mild anaphylactic reactions (4 cases), and rejection (6 cases). Of these animals 5 survived 20 days or longer (up to 138 days) while none of the untreated controls survived longer than 10 days. No evidence was found that antilymphocyte serum had hemolytic activity, probably because it had been repeatedly adsorbed on dog erythrocytes. Only 1 animal developed anemia. Antilymphocyte serum increased the number of leukocytes, particularly polynuclear ones, and reduced the number of peripheral lymphocytes and adult lymphoid cells in the lymph nodes and spleen. Infiltrates of lymphocytes and plasmocytes were found in the livers of 5 of the 6 dogs developing signs of rejection. Increased doses of antilymphocyte serum were given to 1 dog showing signs of rejection. During the 3rd week liver function tests returned to normal, and no histological signs of liver rejection were seen 1.5 months after transplantation. It is possible that in this case the signs of rejection regressed spontaneously. No evidence was found for the mechanism of action of antilymphocyte serum.

7751 **THE EFFECT OF PREDNISONE AND 4-CHLOROTESTOSTERONE ACETATE ON TISSUE PHOSPHATASES IN EXPERIMENTAL FATTY LIVER IN RATS.** *(Ger.)* Petzold, H. (Med. Clin., Karl Marx U., Leipzig, Germany) and A. Ziegler. *Z Ges Inn Med* 22(21):654-657, 1967.

Wistar rats were given 0.1 mg/100 g body weight/day of 4-chlorotestosterone acetate i.m. or 0.04 mg/100 g body weight/day of prednisone i.m. beginning 3 days before resection of 2/3 of the liver. In untreated controls signs of fatty liver appeared 2 days after hepatectomy and were accompanied by increases in acid and alkaline phosphatase and ATPase activities. After 10 days fat deposits were present only in the peripheral and intermediate zones. Administration of prednisone for 3 days produced fat deposits and reduced the activities of all 3 phosphatases. Diffuse fat deposits were found and alkaline phosphatase activities were lower than in controls 2 days after hepatectomy. After 10 days there were only traces of alkaline phosphatase although fat deposits were still present. Administration of 4-chlorotestosterone acetate for 3 days produced small diffuse fat deposits in the liver. However, 2 days after hepatectomy there were no differences between treated and control animals. Ten days after hepatectomy fat deposits were present only in the periphery, while in controls they extended into the intermediate zone. Increases and decreases in phosphatase activities paralleled the extent of fat deposits, and the drug did not inhibit the activities of these enzymes. Thus, 4-chlorotestosterone acetate does not inhibit deposition of fat in the liver, but promotes fat transport.

7752 **EXPERIMENTAL RESULTS AND THE THERAPEUTIC ACTION OF A DEPROTEINIZED LIVER EXTRACT IN HEPATOBILIARY DISEASES OF SURGICAL INTEREST.** *(It.)* Boselli, G. (Gen. Surg. Clin., U. Milan, Italy). *Minerva Med* 58(79):3725-3733, 1967.

Experimental animal studies showed that deproteinized liver extract stimulated liver regeneration in hepatectomized rats, reduced BSP retention in male guinea pigs with acute CCl_4 intoxication, had a protective action against chronic CCl_4 intoxication and acute NH_4Cl intoxication in male rats, and reduced blood lipid levels in rabbits on a high-fat, high-cholesterol diet. Although the nucleoside content of this preparation m have a role in producing these effects, it cannot account for of the activity of deproteinized liver extract. In clinical stud deproteinized liver extract (5 and 10 ml/day i.v. for 4 weeks was given to 68 patients, including 48 with cholelithiasis and biliary stasis, 13 with chronic progressive liver disease, and 7 with choledocholithiasis. Only 2 patients developed mild sig of intolerance (erythema at the site of injection, mild frontal hemicrania). Deproteinized liver extract tended to normalize liver serum enzymes, abnormal serum protein, and blood ammonia levels found in chronic liver disease, produced subjecti improvement, reduced moderate increases in blood bilirubin levels, and normalized prothrombin levels. Similar, but not extensive, improvement was obtained more slowly with a cor bination of vitamins and lipotropic agents on 40 other patien with hepatobiliary diseases.

7753 **ENDOCRINE AND METABOLIC CONTROL MECHANISMS FOR ENZYME SYNTHESIS AND ENZYMATIC ACTIVITY.** *(Ger.)* Englhardt, A. (2nd Med. Clin., U. Dusseldorf, Germany). *Med Welt* 18(1):11-19, 1968.

7754 **CURRENT DATA ON THE ENZYMATIC ACTIVITY OF BILE.** *(Fr.)* Pappo, A. (Inst. Inter Med., Bucharest, Romania) and I. Apostolesco. *Rev Roum M Intern* 4(3):163-167, 1967.

7755 **THE EFFECT OF BACTERIAL LIPOPOLYSAC CHARIDES ON HEPATIC REGENERATION.** *(Hun.)* Főris, G. (State Inst. Publ. Hlth., Budapest, Hungary) M. Keresztes, Z. Bihary, C. Sepp and P. Kertai. *Kiserl Orvost* 19(3):254-257, 1967.

7756 **THE HEPATOTROPIC ACTION OF THE PINE GLAND.** *(Fr.)* Milcou, S. M. (Inst. Endocrin., Acad. Soc. Rep. Romania, Bucharest). *Rev Roum Med Intern* 4(6):381-386, 1967.

7757 **ACTIVATION OF DOPA-DECARBOXYLASE AND OTHER CYTOPLASMIC LIVER ENZYM BY INDUCERS OF ENDOPLASMIC RETICULUM.** *(Ger.)* Palm, D. (Pharmacol. Inst., U. Frankfurt/Main, Germany), C. Ernzerhoff and P. Holtz. *Naunyn Schmiedeberg Arch Exp P* 258(3/4):252-371, 1967.

7758 **EFFECT OF STARVATION ON THE *IN VIVO* METABOLISM AND EFFECT OF DRUGS IN FEMALE AND MALE RATS.** *(E.)* Kato, R. (Nat. Inst. Hy Sci., Tokyo, Japan) and A. Takanaka. *Jap J Pharmacol* 17(208-217, 1967.

7759 **INACTIVATION OF PENICILLINS BY THE ISOLATED RAT LIVER.** *(E.)* Kind, A. C. (U Washington Sch. Med., Seattle), H. N. Beatty, L. F. Fenster and W. M. M. Kirby. *J Lab Clin Med* 71(5):728-735, 1968.

7760 **THE EFFECT OF PHYSOSTIGMINE AND NE STIGMINE ON THE CONCENTRATION OF GLYCOGEN IN THE LIVER OF THE RAT.** *(E.)* Varagić, V. M. (Med. Fac., Belgrade, Yugoslavia), M. Terzić and B. B. Mršulja. *Naunyn Schmiedeberg Arch Exp Path* 258(3/4):229 237, 1967.

61 GLYCOLYTIC SUBSTRATES IN CIRRHOSIS AND IN QUANTITIATIVE UNDERNUTRITION THE RAT. *(E.)* Henley, K. S. (U. Michigan Med. Sch., n Arbor). *J Lab Clin Med* 71(2):183-191, 1968.

62 STUDY OF THE FUNCTIONAL BEHAVIOR OF THE LIVER PARENCHYMA AFTER CROSS- GATION OF THE BILIARY AND PORTAL ELEMENTS. *)* Forti, D. (Inst. Surg. Path., U. Milan, Italy), A. Russo l C. Oldani. *Epatologia* 13(6):1255-1268, 1967.

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NDER THE INFLUENCE OF ADRENALIN. *(Hun.)* Fövényi, (János Hosp., Budapest, Hungary), E. Góth, I. Koncz and Zétény. *Magy Belorv Arch* 21(5):238-242, 1968.

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7808 **THE EFFECT OF TREATMENT WITH ARGININE GLUCOSE-1-PHOSPHATE ON LIVER REGENERATION IN ANIMALS WITH ALCOHOLIC INTOXICATION.** *(It.)* Goi, A. (A. Fleming Res. Inst., SPA, Milan, Italy), F. Rognoni, G. Bevilacqua and G. Gallioli. *Epatologia* 13(3):785-789, 1967.

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See also: 7596,7934,7946,8442,8485,8487,8495,8539,8540,8543,8546,8548,8577,8581,8631,8649,8657

7810 **THE ROLE OF BILE SALTS IN CONTROLLING THE RATE OF INTESTINAL CHOLESTEROGENESIS.** *(E.)* Dietschy, J. M. (U. Texas Southwest. Med. Sch., Dallas). *J Clin Invest* 47(2):286-300, 1968.

In order to identify the constituent of bile responsible for inhibition of intestinal cholesterogenesis, to determine its biochemical and anatomic sites of action, and to define the kinetics of this inhibition, various experiments were carried out in female Sprague-Dawley rats. Four different diets were used: 1) a low-cholesterol control diet; 2) a high-cholesterol diet prepared by dissolving 1.5 g of cholesterol in 5 g of hot triolein and mixing this with 100 g of ground rat chow; 3) a 3 or 5% cholestyramine diet; and 4) liquid diets for intragastric feeding. Initial experiments provided evidence suggesting that bile salt is the constituent of bile responsible for the specific inhibition of intestinal cholesterogenesis in the intact animal. Cholestyramine, a resin which binds bile salts intraluminally, was nearly as effective as biliary diversion in enhancing intestinal sterol synthesis, and an inverse relation was found down the length of the small bowel between the rate of cholesterogenesis and the intraluminal bile salt concentration. Experiments identified the cells of the intestinal crypt as the anatomic site within the intestinal wall where feedback inhibition of the rate of sterol synthesis takes place. The point of feedback inhibition can be localized to one of the few enzymatic steps between the condensation of acetyl-CoA to form acetoacetyl CoA and the reduction of hydroxymethylglutaryl-CoA to mevalonate, and probably involves the reaction mediated by hydroxymethylglutaryl reductase. Kinetic experiments demonstrated that inhibition of intestinal cholesterogenesis by bile salt is characterized by a marked decrease in the maximum rate of incorporation of acetate into sterol/unit weight of tissue, without any significant change in the substrate concentration at which these maximal velocities are achieved.

7811 **TRANSFER OF LOCALLY SYNTHESIZED CHOLESTEROL FROM INTESTINAL WALL TO INTESTINAL LYMPH.** *(E.)* Wilson, J. D. (U. Texas Southwest. Med. Sch., Dallas) and R. T. Reinke. *J Lipid Res* 9(1):85-92, 1968.

Male rats fed a diet containing 0.5% cholesterol for 1-3 weeks (to inhibit hepatic cholesterol synthesis) were utilized for 2 types of experiments designed to study the movement of cholesterol synthesized in the intestinal wall to intestinal lymph. Intestinal perfusion studies were performed on rats fitted with intestinal lymph duct cannulas, in which catheters were sewn into the duodenum and terminal ileostomies were constructed. A solution containing 2.5% mannitol, 2.5% glucose and electrolytes was infused into the duodenal cannula (1-2 ml/hr). Oleic acid (1 ml) was also introduced into the duodenum every 3-4 hr, and in some animals, egg yolk (containing 1 mg/ml cholesterol) was also added. Acetate-2-^{14}C was given i.v., and lymph and intestinal contents were sampled and analyzed at intervals for 48 hr. In another series of experiments, lymph ducts cannulations were performed, cannulae were placed in the stomach, and high bile duct cannulations were also performed. After administering acetate-2-^{14}C, either high or low cholesterol-containing diets were given through the stomach tube, and feces were examined along with intestinal tissue for labeled cholesterol during a 48-hr study period. The results showed that locally synthesized cholesterol is excreted bidirectionally, approximately half going into the lymph and half going into the intestinal lumen. Little of the luminal cholesterol appeared to be reabsorbed, suggesting that the direct transfer from wall to lymph is the principal route for entry of this endogenously produced cholesterol into lymph and ultimately into the blood stream. Ligation of the bile duct demonstrated that bile is required for this transfer of cholesterol from wall into lymph as well as for the absorption of dietary cholesterol.

7812 **CELLULAR DYNAMICS IN THE JEJUNUM OF ESSENTIAL FATTY ACID DEFICIENT MICE.** *(E.)* Snipes, R. L. (Div. Biol. Med. Sci., Brown U., Providence, R. I.). *Anat Rec* 159(4):421-430, 1967.

The population dynamics of the jejunal epithelium of mice maintained on a semisynthetic diet deficient in essential fatty acids (EFA) were investigated, using colchicine to collect mitotic figures for the determination of mitotic indices. Twenty-five crypts in longitudinal section were counted per animal and the mean mitotic index was computed as the number of mitos per total number of crypt cells. EFA-deficient animals had a mean mitotic index of 36.56 as compared with 26.51 and 16.7 for the linoleic acid-supplemented and chow-fed control animal resp. Counts of the number of epithelial cells on 1 side of a villus and crypt indicated that the height of the villi, but not that of the crypts, was significantly lower in the EFA-deficient animals than in either control group. In the EFA-deficient animals, the mean percentage of villi having extrusion zones exceeded that in both control groups. Radioautography demonstrated that 36 hr after injection of tritiated thymidine the majority of villi in the EFA-deficient animals were labeled at their tips, whereas in control samples labeling was restricted to the lower portions of the villus. Thus, EFA-deficiency induces increased rates of mitosis, migration and sloughing, resulting in alterations in villous architecture.

7813 **CELL NUMBER AS A MEASURE OF DISTRIBUTION AND RENEWAL OF EPITHELIAL CELLS IN THE SMALL INTESTINE OF GROWING AND ADULT RATS.** *(E.)* Altmann, G. G. (Dept. Anat., McGill U., Montreal, Canada). *Amer J Anat* 121(2):319-336, 1967.

Based on DNA estimations and differential cell counts, with or without colchicine treatment (0.1 mg/100 g) 4 hr before sacrifice, the absolute numbers of villus epithelial cells, and of dividing and nondividing crypt epithelial cells, were calculated in 10 successive segments of the small intestine (pylorus to cecum) of 16-18 day-old (weanling), 36-39 day-old (young) and 85-90 day-old (adult) Sherman rats. The number of crypt cells per segment was relatively constant regionally (averaging 17, 103 and 182 million in every segment of the 3 age groups, resp.) and the proportion of mitotic crypt cells was also similar throughout the small intestine (3.7%, 5.3% and 6.4% for the 3 age groups, resp.). As expected, the calculated number of epithelial cells produced per 4 hr showed no significant regional variation (1.87, 15.2 and 29.9 million per segment for the 3 age groups, resp.). The duodenal epithelium of weanling nd young animals differed from the rest of the epithelium in hat the number of crypt epithelial cells was significantly above verage and the mitotic index was below average. In adults, he number of villus epithelial cells per segment decreased radually from 270 million in the duodenum to 127 million n the ileum. It was deduced that the turnover time of the illus epithelium (1.1 days for the entire intestine) gradually lecreased, but that that of the crypt epithelium (1.1 days for he entire intestine) remained constant along the length of the mall intestine. With increasing age, the calculated number of pithelial cells produced daily rose from 112 (weanling) to 1796 nillion (adult). However, of the total number of epithelial ells present in the small intestine of weanling, young and adult ats, 21, 40 and 48%, resp. were found to enter mitosis daily o produce new cells. The extent of this acceleration of renewal is also demonstrated by the mean turnover time of the ntestinal epithelium, which decreased from about 22 days in veanling rats to 2.6 and 2.1 days in young and adult rats, esp., indicating a 10-fold increase in renewal rate.

814 CELL PROLIFERATION STUDIES IN THE INTESTINAL EPITHELIUM OF THE RAT: RESPONSE TO CONTINUOUS IRRADIATION. *(E.)*

Cairnie, . B. (Inst. Cancer Res., Surrey, England). *Radiat Res* 32(2): 40-264, 1967.

hanges in cell proliferation kinetics in the jejunal crypts under he stress of continuous irradiation at 350 rads/day and the eturn to the normal condition after irradiation were studied n August strain rats, weighing 100-120 g. In experiment 1, he distributions of labeled and mitotic cells were studied in xposure to 350 rads/day for 0,1,2, or 5 days and following or 2 days of recovery after 5 days of irradiation. In experiments 2 and 3, the first and last 2-day periods covered in experiment 1 were studied in more detail. In experiment 4, an nalysis was made of the cell cycle, based on the percentage of abeled mitoses, and of the transit time on the villus in animals continuously exposed to radiation for 5-7 days. Results from xperiment 1 showed that after irradiation for 1 day, there was marked reduction throughout the crypt in the percentage of abeled cells. The maximum labeling index was below 40%, compared with about 56% in the controls. Mitotic activity was ery low in the bottom of the crypt and below normal in the nidregion. After 2 days the labeling index had recovered to give a peak level of almost 50%. There was little change between 2 and 5 days, the interval when the intestine is in a steady-state conditon. One day after irradiation was discontinued the peak labeling index was about 66%, while 2 days after cessation or irradiation the proliferative activity was returning toward normal. In experiment 2, the number of labeled cells/crypt column reached a minimum, which was less than 40% of the initial value, after irradiation for 18 hr and then recovered to near normal by 36 hr. Mitotic activity fell for 24 hr to a minimum and then recovered gradually. At the end of irradiation the distributions of labeled and mitotic cells in the crypts of rats in experiment 3 were very similar to those seen in some animals in experiment 1. The labeling index was increased by 6 hr, giving a pattern close to normal which indicated a considerable augmentation, during this time, of cells beginning DNA synthesis. At 12 hr there was still some deficit in the lower part of the crypt, but many more labeled and dividing cells were found in the upper part of the crypt than in controls. The pattern changed little during the next 24 hr, and at the end of 42 hr repopulation of crypt and villus had begun. In experiment 4 in both normal and irradiated animals, the turnover time of the villus epithelium was 24 hr after allowing for the delay before labeled cells reach the base of the villus.

7815 COMBINATIONS OF RADIOPROTECTIVE COMPOUNDS AND THE MIGRATION OF CRYPT CELLS ALONG VILLI IN THE MOUSE DUODENUM. *(Fr.)*

Maisin, J. R. (Dept. Radiobiol., Ctr. Study Nucl. Energy, MOL, Belgium) and M. Lambiet-Collier. *C R Soc Biol (Paris)* 161(1): 1134-1138, 1967.

BALB/c mice were given 16 mg of reduced glutathione by stomach tube and 15 and 20 min later, resp., 5 mg of 2-β-aminoethylisothiourea bromide HBr and 1 mg of 5-hydroxytryptamine i.p. The duration of the cell cycle and the rate at which crypt cells migrated along the duodenal villi were determined autoradiographically with ^{3}H-thymidine. Thymidine was incorporated more slowly into DNA and the cell cycle was longer in treated mice than in untreated controls. The G_2 phase and DNA synthesis were longer and the G_1 phase shorter in treated animals. The migration of crypt cells along the duodenal villi was slower in treated animals, particularly when repeated doses of radioprotective agents were given.

7816 INHIBITION OF INTESTINAL CHOLESTEROGENESIS *IN VITRO*. *(E.)*

Cayen, M. N. (Ayerst Res. Lab., Montreal, P. Q., Canada) and D. Dvornik. *Proc Soc Exp Biol Med* 127(1):117-120, 1968.

The effect of several inhibitors of hepatic cholesterol synthesis on the incorporation of acetate-(2-^{14}C) and DL-mevalonate-(^{3}H) into cholesterol was studied in sections of everted rat ilea. The agents AY-9944, SKF 525-A, triparanol, and 22, 25-diazacholestanol suppressed the incorporation of acetate-(2-^{14}C) and mevalonate-(^{3}H) into cholesterol. Clofibrate had no effect on the conversion of acetate-(2-^{14}C) into cholesterol. The results indicate the presence in rat intestine of enzyme systems involved in the saturation of the Δ^{24}-bond in lanosterol and its metabolites, the conversion of 7-dehydrocholesterol into cholesterol, and in those reactions inhibited by SKF 525-A. Hence, the pathway of intestinal cholesterol synthesis is similar to that in the liver.

7817 THE ROLE OF THE COLON IN THE DUALITY OF FECAL EXCRETION IN THE RABBIT.

(Fr.) Bonnafous, R. (Inst. Physiol., Toulouse, France) and P. Raynaud. *Arch Sci Physiol (Paris)* 34(3):261-270, 1967.

Rabbits were sacrificed at different hours of the day and night, and stool samples were collected from 6 parts of the colon for dry weight and nitrogen determinations. A sharp decrease in the nitrogen content from 43.7% to 26.1% occurred near the middle of the proximal colon. The loss of water occurred very gradually with the highest rate of loss in the distal colon. Removal of moderately long segments of the colon resulted in the production of stools which had nitrogen contents intermediate between those of "day" and "night" stools. However, 15 days after surgery 2 kinds of stools appeared again. Autopsies, performed 6 months after surgery, showed that the part of the intestine adjacent to the segment which had been removed had increased in length.

7818 **ATPASE ACTIVITY OF GLYCERINATED MUSCLE HOMOGENATES PREPARED FROM PIG INTESTINAL SMOOTH MUSCLE. 2. EFFECTS OF SOME CHELATING AGENTS AND MICROSOMAL FRACTION.** *(E.)* Taneda, A. (Sapporo Med. Coll., Japan). *Sapporo Med J* 31(2/3):110-115, 1967.

7819 **COMPENSATORY REACTIONS IN INTESTINAL CRYPT CELLS AFTER 300 ROENTGENS OF COBALT-60 GAMMA IRRADIATION.** *(E.)* Lesher, S. (Div. Biol. Med. Res., Argonne Natl. Lab., Ill.). *Radiat Res* 32(3): 510-519, 1967.

7820 **PREVENTION OF 5-FU INDUCED NADPH-D TRANSLOCATION IN COLONIC EPITHELIUM BY ANTIBIOTICS AND THE EFFECT ON THE INTESTINAL BACTERIAL FLORA.** *(E.)* Floch, M. H. (Yale U. Sch. Med., New Haven, Conn.), H. J. Kaufmann and H. M. Spiro. *Scand J Gastroent* 2(3):186-191, 1967.

7821 **BIOSYNTHETIC ORIGIN OF SERUM CHOLESTEROL IN THE SQUIRREL MONKEY: EVIDENCE FOR A CONTRIBUTION BY THE INTESTINAL WALL.** *(E.)* Wilson, J. D. (U. Texas Southwestern Med. Sch., Dallas). *J Clin Invest* 47(1):175-187, 1968.

See also: 7597,7626,8226,8258

GENERAL

22 **APPEARANCE AND BEHAVIOR OF VENO-VASOMOTOR REACTIONS IN THE LIVER RCULATION.** *(Ger.)* Lutz, J. (Physiol. Inst., U. Wurzburg, rmany) and E. Bauereisen. *Pflueger Arch Ges Physiol*)(4):311-325, 1968.

23 **THE EFFECTS OF SPICES ON DIGESTIVE FUNCTIONS.** *(Ger.)* Glatzel, H. (Max Planck t. Nutrit. Physiol., Dortmund, Germany). *Materia Med rdmark* 20(5):245-255, 1968.

24 **BILE CONVECTION IN A NORMAL GALL-BLADDER DURING CHOLECYSTOGRAPHY.** *is.)* Krugliakov, I. O., L. D. Lindenbraten, Ie. Z. Poliak l E. G. Sterebrianyi. *Sovet Med* 31(3):53-57, 1968.

25 **HYDROGEN AND METHANE PRODUCTION IN MAN.** *(E.)* Levitt, M. D. (Boston City Hosp., ss.) and F. J. Ingelfinger. *Ann NY Acad Sci* 150(1):75-81, 68.

26 **THROUGH THE ALIMENTARY CANAL WITH GUN AND CAMERA.** *(E.)* Chappell, G. S. w York, Dover, 1967. pp. 114.

27 **DISTRIBUTION OF ALCOHOL IN URINE AND SWEAT AFTER DRINKING.** *(E.)* Pawan, L. S. (Middlesex Hosp. Med. Sch., London, England) and Grice. *Lancet* 2(7576):1016, 1968.

28 **LIVER BLOOD FLOW IN MAN STUDIED WITH THE XENON METHOD.** *(E.)* Darle, N. (Dept. g., U. Göteborg, Sweden), N. G. Kock and D. H. Lewis. *l Soc Int Chir* 27(3):203-206, 1968.

29 **LARGE-SCALE GEL-FILTRATION IN THE PURIFICATION OF HUMAN LIVER AND ALL INTESTINE ALKALINE PHOSPHATASES.** *(E.)* Smith, J. K. (Royal Infirm., Edinburgh, Scotland), R. J. Eaton, L. G. Whitby and D. W. Moss. *Anal Biochem* 23(1):84-96, 1968.

7830 **THE GASTROINTESTINAL EPITHELIUM AND ITS AUTOCHTHONOUS BACTERIAL FLORA.** *(E.)* Savage, D. C. (Rockefeller U., New York, N. Y.), R. Dubos and R. W. Schaedler. *J Exp Med* 127(1):67-75, 1968.

7831 **THE EFFECTS OF HYPERCAPNIA AND HYPOXIA ON THE DISTRIBUTION OF CAPILLARY BLOOD FLOW IN THE DENERVATED INTESTINAL VASCULAR BED.** *(E.)* Svanvik, J. (Dept. Physiol., U. Göteborg, Sweden), J. Tyllström and I. Wallentin. *Acta Physiol Scand* 74(4):543-551, 1968.

7832 **ANTIBODIES TO INTESTINAL MICROVILLOUS MEMBRANES. I. CHARACTERIZATION AND MORPHOLOGIC LOCALIZATION.** *(E.)* Kopp, W. L. (Madison VA Hosp., Wisconsin), J. S. Trier, O. L. MacKenzie and R. M. Donaldson Jr. *J Exp Med* 128(2):357-373, 1968.

7833 **SOME HYALURONIDASE-PRODUCING ENTEROCOCCI AND COLIFORM BACILLI.** *(Rum.)* Bîrzu, A. (Inst. Hygiene, Iasi, Romania), I. Huţu, M. Antonescu, V. Dobre, E. Bailicenco and V. Vîrlan. *Rev Medicochir Iasi* 72(1):141-145, 1968.

7834 **THE ROLE OF ORGANIC AND MINERAL COMPONENTS OF MINERAL WATER IN THE MECHANISM OF THEIR EFFECT ON DIGESTIVE ORGANS.** *(Rus.)* Vasilenko, Yu. K. (Inst. Balneology Physiother., Pyatigorsk, USSR). *Vop Kurort Fizioter* (1):27-31, 1968.

7835 **ADVANCEMENTS IN GASTROENTEROLOGY.** *(Pol.)* Koziolowa, H. *Pol Arch Med Wewnet* 40(5):653-659, 1968.

DIAGNOSTIC PROCEDURES

7836 **SERUM CATALASE IN ACUTE PANCREATITIS.** *(Ger.)* Gülzow, M. (Med. Clin., U. Rostock, Germany) and G. Möller. *Deutsch Z Verdau Stoffwechselkr* 27(5/6):175-183, 1967.

Serum catalase and amylase determinations were made on 40 patients, aged 20-80 yr, with acute pancreatitis. Biliary tract disease was present in 29 cases and 28 had cholelithiasis. At the onset of symptoms, catalase and amylase activities were elevated in 29 and 36 of these 40 patients, resp. Amylase activities consistently returned to normal more rapidly than catalase activities. No relation was observed between the catalase and amylase activity and the severity of the pancreatitis, but increases in catalase activity persisted longer in patients with severe forms of pancreatitis. Increased catalase activities were also found in 6 of 22 patients with malignant blood diseases and in 8 of 8 patients with Pyrasid-induced fevers. Some moderate increases in catalase activity occurred during various stages of viral hepatitis. Normal catalase activities were repeatedly obtained on 2 patients with chronic pancreatitis and low values were obtained in a patient with a carcinoma of the head of the pancreas. Because of its lack of specificity, it is concluded that serum catalase activity is of little value in the diagnosis of acute pancreatitis.

7837 **THE DIAGNOSTIC VALUE OF LEUKOCYTE ALKALINE PHOSPHATASE IN HEPATOBILIARY DISEASES.** *(Ger.)* Pexa, H. (Empress Elisabeth Hosp., Vienna, Austria) and A. Neumayr. *Acta Hepatosplen (Stuttgart)* 14(6):348-357, 1967.

The activity index for leukocyte alkaline phosphatase was determined at weekly intervals in 50 normal controls and 31 patients with diseases of the liver and biliary tract (10 with extrahepatic obstructive jaundice, 11 with acute viral hepatitis, 2 with chronic hepatitis, and 8 with cirrhosis). Normal values ranged from 15-82 with a mean of 43.4 ± 18.87. Significant increases in alkaline phosphatase were found in 6 patients with benign and 4 with malignant obstructive jaundice. Marked increases in activity occurred after surgery or during an inflammatory complication. In patients with acute viral hepatitis the activity index for alkaline phosphatase was in the low-normal range or just below it. Activities were above normal in hepatitis patients with gastritis and stomach ulcers and in those who took more than 25 mg/day of prednisolone. Of the 2 patients with chronic hepatitis, 1 with acute gastroenteritis had elevated alkaline phosphatase activities and the other, who was on long-term therapy with 10 mg/day of prednisolone, had activities in the normal range. The activities were elevated or in the high-normal range in patients with cirrhosis. Even higher activities were noted in febrile intercurrent infections and liver coma. Very low values were found in a patient with an exacerbation of cirrhosis. No correlations were established between leukocyte alkaline phosphatase activities and serum bilirubin and transaminase levels or serum protein fractions. It is concluded that leukocyte alkaline phosphatase activities are of little diagnostic value for chronic hepatitis and cirrhosis.

7838 **DIFFERENTIAL DIAGNOSIS OF THE SPRUE SYNDROME.** *(Ger.)* Ammann, R. (Med. Polyclin., U. Zurich, Switzerland). *Therapiewoche* 18(10):397-408, 1968.

After the review of techniques used in the differential diagnosis of sprue, fecal chymotrypsin activities are compared in 100 controls and 113 patients with various diseases of the pancreas. There were good correlations between fecal chymotrypsin activity and the results of the pancreozymin-secretin test in patients with severe and moderately severe exocrine pancreatic insufficiency, but only a poor correlation in patients with mild insufficiency such as that found after acute pancreatitis. Very good correlations between these 2 tests were found in a group of 19 patients with carcinoma of the head of the pancreas and in 34 with chronic pancreatitis. Only slight differences in the results of repeated determinations of fecal chymotrypsin activity were found in 23 patients with chronic pancreatitis. Low chymotrypsin activities were obtained after 1-2 yr in 2 of 3 patients who initially had normal activities. Determination of fecal chymotrypsin activity is recommended if facilities are not available to perform the pancreozymin-secretin test.

7839 **COMPARISON OF GASTRIC ACID DETERMINATIONS WITH THE HEIDELBERG CAPSULE AND RESULTS OF HISTOLOGICAL STUDIES OF THE GASTRIC MUCOSA AND THE MAXIMAL HISTAMINE TEST.** *(Ger.)* Ewe, K. (2nd Med. Clin., U. Saar, Homburg, Germany) and H. Weis. *Deutsch Med Wschr* 93(29):1389-1393 1968.

The time required for the stomach contents to become acid again after ingestion of 5 ml of 1 N bicarbonate was measured in 533 patients using a Heidelberg capsule. Anomalous results, obtained in 33 cases, were excluded. Results of this test showe that 221 patients had normal gastric acidity, 58 had hypochlorhydria, and 80 had achlorhydria. Histological examination of mucosa obtained by aspiration biopsy revealed that all patients with achlorhydria had gastritis (85% with chronic atrophic, 2.5 with acute, and 12.5% with superficial gastritis). Gastritis, including 11 cases of the chronic atrophic form, was also found in 20 of 29 patients with hypochlorhydria. None of the patien with hyperchlorhydria had atrophic gastritis. Results of the alkali test were within the normal range in 3 patients with histologically diagnosed atrophic gastritis. These included 2 patien who still had functioning glandular tissue and 1 with severe but localized gastritis. Two patients with atrophic gastritis develope bleeding from the stomach after aspiration biopsies. Duodenal ulcers were detected radiologically in only 3 patients with norm gastric acidity (2.6%) and in 25 (27.1%) of those with hyperchlorhydria. Maximum acidity after histamine stimulation was determined in 15 patients with duodenal ulcers. On the whole, these results agreed with those of the alkali test, but there was no strict correlation between the 2 tests in individual cases.

7840 **SERUM IRON DETERMINATION AS A LIVER FUNCTION TEST.** *(Ger.)* Vaništa, J. (Pediat. Fac., Charles U., Prague, Czechoslovakia), J. Tesařová, J. Kocián and B. Jirsíková. *Ther Gegenw* 107(7):910-918, 1968.

Serum Fe levels were determined by a modification of the Vannotis method on 263 patients, including 177 with acute viral hepatitis, 16 with other liver diseases, 60 with biliary tract disease, and 10 controls. Increased serum Fe levels were found during the first week of viral hepatitis in 82.5% of the 57 children and 93.3% of the 120 adults. There was no correlation

tween increases in serum Fe levels and the course of the
;ease or increases in serum bilirubin values. Repeated deter-
nations on 143 patients showed that serum Fe levels remained
vated even after jaundice disappeared and returned to normal
the same time as SGPT activities in 54%. Increased serum
levels were found in 19 patients with abortive and anicteric
patitis; in 6 they were the only biochemical indication of liver
mage. Serum Fe and SGPT levels increased simultaneously
1 patient in the prodromal stage; in another, serum Fe levels
creased 13 days before SGPT levels. Increased serum Fe levels
re found in 9 of the 16 patients with other liver diseases: 4
th chronic hepatitis and cirrhosis, 3 with jaundice due to a
imary liver tumor, 1 with liver damage caused by pyrazinamide,
d 1 with chronic posthepatitic hyperbilirubinemia. No in-
eases occurred in 3 patients with hepatic coma. Serum Fe
vels were normal in 7 patients with "silent" cholelithiasis and
vated in 43% of those with simple biliary colic. Elevations
re more common among patients with cholestasis and gall-
adder infiltration. Low values were found in most patients
th pancreatitis. These findings indicate that serum Fe levels
e of diagnostic value in abortive hepatitis if serum transaminase
lues are within normal limits, as well as to distinguish between
renchymal and cholestatic jaundice and for the detection of
mochromatosis.

41

**DIAGNOSIS OF BILIARY ATRESIA: RELATIVE ACCURACY OF PERCUTANEOUS LIVER
IOPSY, OPEN LIVER BIOPSY, AND OPERATIVE CHOLAN-
IOGRAPHY.** *(E.)* Hays, D. M. (U. Southern California
h. Med., Los Angeles), M. W. Woolley, W. H. Snyder Jr.,
B. Reed, J. L. Gwinn and B. H. Landing. *J Pediat* 71(4):
)8-607, 1967.

ne accuracy of percutaneous liver biopsy, open liver biopsy
d operative cholangiography as diagnostic procedures was
alyzed in 132 children (3-13 weeks old) with obstructive
onatal jaundice seen at the Children's Hospital of Los Angeles.
the 25 infants in whom percutaneous liver biopsies were
rformed, the interpretation was substantiated by the ultimate
agnosis in 15, was indecisive or equivocal in 4, and was in
ror in 6. Of the total group of diagnoses following open liver
opsy, 46 were regarded as correct, 22 were regarded as equi-
ocal or not distinguishing in regard to the disease processes
biliary cirrhosis and/or bile retention, and 7 were regarded
incorrect. Operative cholangiograms usually indicate the
rrect surgical management, but in approximately 20% of the
atients studied the procedure suggested a diagnosis ultimately
own to be incorrect.

342

**HETEROGENEITY OF SERUM γ-GLUTAMYL TRANSPEPTIDASE IN DIFFERENT INTERNAL
ISEASES STUDIED BY STARCH GEL ELECTROPHORESIS
ND SEPHADEX FILTRATION.** *(E.)* Kokot, F. (3rd Int.
is. Clin., Silesian Acad. Med., Katowice, Poland) and J. Kuska.
ızym Biol Clin 9(1):59-67, 1968.

arch gel electrophoresis and Sephadex filtration were used
investigate γ-glutamyl transpeptidase (GGTP) activity in the
ra of 15 healthy subjects, 88 patients with hepatobiliary dis-
ses, and 64 patients with other diseases. No distinct distribu-
n curves of GGTP activity were obtained by either method
r any disease except liver cirrhosis. The heterogeneity of
serum GGTP is concluded to be unsuitable for the differential diagnosis of hepatic and extrahepatic diseases. Further investigations are needed, however, into substrate specificity physicochemical properties, susceptibility to activators and inhibitors, and the causes of GGTP heterogeneity.

7843

SIGNIFICANCE OF AN ELEVATED SERUM AMYLASE. *(E.)* Adams, J. T. (U. Rochester Med. Ctr., N. Y.), J. A. Libertino and S. I. Schwartz. *Surgery* 63(6): 877-884, 1968.

Of 154 patients with abdominal pain and hyperamylasemia, nearly 75% had isolated or associated pancreatitis, while in the remainder there was no apparent pancreatic lesion. In general, the serum amylase level was inversely related to the severity of pancreatic disease. When the amylase was over 1000 Somogyi U, there was usually a surgically correctable lesion, most frequently acute calculous disease of the biliary tract, while pancreatitis, if present, was of minimal or moderate extent. In contrast, when the amylase activity was between 200 and 500 Somogyi U, the majority of patients had isolated idiopathic or "alcoholic" pancreatitis; all instances of necrotizing or hemorrhagic pancreatitis were seen in this amylase group. The serum amylase is thus an important adjunctive measure in the management of a patient with abdominal pain. Markedly elevated levels, coupled with continued clinical manifestations, should prompt early operative intervention, while minimally elevated levels suggest that, unless clinical considerations dictate otherwise, the operation should be delayed.

7844

HEPATIC VEIN VISUALIZATION BY INTRAVENOUS CARBON DIOXIDE INJECTION. *(E.)* Collins, J. R. (VA Hosp., Nashville, Tenn.) and V. A. Vix. *Radiology* 89(4):864-867, 1967.

In an attempt to visualize the hepatic veins radiographically, carbon dioxide was injected i.v. in 22 patients with suspected pericardial effusion and 48 noncardiac patients (25 with hepatic disease and 23 controls). The hepatic veins wre visualized in 78% of patients without hepatic disease, but in only 8% of patients with hepatic disease (mainly cirrhosis). The diameter of the hepatic vein ranged from 5-15 mm. Visualization of the right atrium was improved by placing the patient in a 15° head-down tilt. In the cirrhotics, a pressure-flow phenomenon, rather than anatomical narrowing, was responsible for nonvisualization. The failure to demonstrate the hepatic veins regularly in cirrhotics places a limitation on the clinical usefulness of this procedure.

7845

THE RADIOLOGICAL ASSESSMENT OF GASTRIC ACID OUTPUT. *(E.)* Moghadam, M. (Henry Ford Hosp., Detroit, Mich.), R. Gluckmann and W. R. Eyler. *Radiology* 89(4):888-892, 1967.

Maximum histalog stimulation (MHS) and barium meal studies were undertaken in 400 consecutive cases in order to determine the correlation between mucosal patterns and gastric acid output. The rugal patterns of the stomach and duodenum were scored separately. There was a good correlation between MHS results and the appearance of the mucosal folds; rugal atrophy was consistently associated with low acidity or achlorhydria, and the coarser the rugae, the higher the gastric acid output.

Since the majority of patients in whom an MHS test is recommended are likely to have had a barium meal study, the gastric acid secretion can be easily and fairly accurately estimated according to the mucosal pattern of the stomach and duodenum, thus eliminating the need for gastric intubation.

7846 **ROENTGEN EVALUATION OF THE DILATED STOMACH AND ITS CONTENTS IN THE DIFFERENTIAL DIAGNOSIS OF PYLORIC OBSTRUCTION.** *(E.)* Bryk, D. (Maimonides Med. Ctr., Brooklyn, N. Y.) and K. B. Robinson. *Radiology* 89(4):893-895, 1967.

The roentgen appearance of the dilated stomach and its contents was evaluated in 40 cases of compensated pyloric obstruction, including 26 cases due to pyloroduodenal ulcer and 14 due to neoplasm of the distal stomach. In association with the hypersecretion in obstructing ulcer, the gastric rugae tend to persist despite gastric dilatation, while in neoplastic obstruction, which is usually associated with hyposecretion, the rugae tend to be obliterated. In peptic ulcer disease, the retained fluid tends to be roentgenographically homogeneous with precipitation and layering of the barium, while in neoplastic obstruction, the retained fluid tends to be nonhomogeneous with less layering of barium. These findings are felt to be useful in differential diagnosis as a supplement to the roentgen features of the obstructing lesion.

7847 **A CRITICAL EVALUATION OF SELECTIVE CELIAC AND SUPERIOR MESENTERIC ANGIOGRAPHY IN THE DIAGNOSIS OF PANCREATIC DISEASES, PARTICULARLY MALIGNANT TUMOR: FACTS AND "ARTEFACTS".** *(E.)* Nebesar, R. A. (Harvard Med. Sch., Boston, Mass.) and J. J. Pollard. *Radiology* 89(4):1017-1027, 1967.

A series of 300 selective arteriograms of the celiac axis, superior mesenteric artery, and their branches was analyzed for findings consistent with the changes of pancreatic carcinoma described in the literature. Such changes were found in 32 patients without evidence of tumor, indicating that at least 10% of patients without pancreatic carcinoma will show false-positive changes suggesting pancreatic neoplasm. The selective celiac and superior mesenteric angiograms of 47 patients studied for suspected pancreatic neoplasm were subjected to preoperative and retrospective analysis. Among 17 cases of proven malignant tumor there were 8 false negatives, and among 15 proven normal cases, there were 5 retrospective false-positives. The effects of pseudocysts could frequently be seen, but pancreatitis could rarely be diagnosed. This study suggests that pancreatic angiography as currently performed must be interpreted cautiously and regarded with some skepticism.

7848 **RADIOISOTOPE "SUBTRACTION" SCANNING FOR PANCREATIC LESIONS.** *(E.)* Eaton, S. B. (Harvard Med. Sch., Boston, Mass.), M. S. Potsaid, H. H. Lo and E. Beaulieu. *Radiology* 89(4):1033-1039, 1967.

An isotope subtraction system designed to eliminate "liver overlap" during pancreatic scanning is described. Gamma radiation from colloidal ^{198}Au in the liver and ^{75}Se-selenomethionine in the liver and pancreas is simultaneously detected by the scintillation detector and processed by dual pulse-height analyzer:ratemeter complexes. The ratio of counts from ^{198}Au and ^{75}Se over the right lobe of the liver is determined, and the apparatus is then operated so that no photoscan image is formed where this ratio is detected. Where pancreatic tissue is present, however, the ^{198}Au:^{75}Se ratio does not conform to that determined over liver alone, and therefore photoscan image formation proceeds whether or not any liver tissue is anatomically superimposed upon the pancreas. The system speeds and simplifies the scanning procedure and produces scans of superior quality. It is concluded that isotope subtraction scanning represents a significant advance in pancreatic visualization.

7849 **FINDING THE BOUNDARY BETWEEN THE CORPUS AND ANTRUM BY GASTROSCOPY.** *(Ger.)* Ottenjann, R. (Med. Clin., U. Erlangen, Germany), H. Peters and K. Elster. *Klin Wschr* 45(19):1006-1008, 1967.

A gastroscope technique is described which can be used to mar off the boundary between the gastric corpus and antrum befor an antrectomy is performed in patients with peptic ulcers. After neutralization of acid by instillation of 100 ml of 5% NaHC NaHCO$_3$ and aspiration of the stomach contents, 40 ml of a 0.3% Congo red solution are introduced through a stomach tub At a pH of 3.0 or less the indicator turns blue-black, thus mark ing off acid-secreting cells in the body of the stomach from the antral mucosa, which is stained red. Stimulants of acid secretion are given parenterally to hasten the process. The boundar between the antrum and corpus is then marked off by injecting a few drops of dilute India ink into the submucosa through a needle attached to a gastroscope. The location of this boundar was confirmed histologically in 15 cases.

7850 **QUANTITATIVE EVALUATION OF PERISTALSIS IN THE DIAGNOSIS OF STOMACH CANCE** *(Rus.)* Maiskii, V. B. (M. F. Vladimirskii District Sci.-Res. Inst. Moscow, USSR). *Vestn Rentgen Radiol* 42(5):42-49, 1967.

After ingestion of a barium cocktail, the amplitude and frequency of peristaltic waves on part of the lesser curvature of the stomach were measured by a radiocinematographic method. Harmonic analyses of the data were performed on a computer, using Fourier's series. Analysis of the frequency spectrum of peristaltic waves in normal subjects showed that it was very stable and did not change with age. Only the amplitude change when drugs were administered to stimulate peristalsis. In normal subjects the spectrum was made up of low-frequency harmonics. Use of this method on 22 patients with different form and stages of stomach cancer showed that there was a larger percentage of high-frequency impulses compared to healthy controls. There was a direct proportionality between the percentage of these waves and the extent of the cancer, so that the largest numbers of high-frequency impulses were seen in patients with invasive forms of cancer. The increase in the percentage of high-frequency harmonics in cancer is probably due to sclerosis of the muscular layer which causes the wall of the stomach to lose its ability to undergo active contractions.

7851 **LIVER FUNCTION DIAGNOSIS WITH WOFAVERDIN.** *(Ger.)* Hambsch, K. (Med. Polyclin. Inst., Karl Marx U., Leipzig, Germany), D. Reinelt, H. J. Klugmann and H. Häntzschel. *Z Ges Inn Med* 22(18): 560-563, 1967.

Determinations were made of the half-life of Wofaverdin after 10 min and the half-life of BSP after 45 min on 38 subjects with no evidence of liver disease and 47 patients with clinically

l histologically confirmed liver disease. Abnormal BSP and
ofaverdin half-lives were found in a total of 6 controls. Liver
ease was ruled out in 5 of these 6 patients. BSP half-lives
re abnormal in only 27 of the 47 patients with liver disease,
ile Wofaverdin half-lives were abnormal in 35. The use of
th methods showed that dye retention was abnormal in 38
these 47 patients. The Wofaverdin test was more sensitive
an the BSP test in the diagnosis of individual liver diseases.
was also shown to be more sensitive than protein lability
ts, serum protein fractions, and serum enzymes in the diag-
sis of liver diseases.

52 **THE EFFECT OF AGE ON THE NORMAL VALUE OF THE BROMSULFALEIN TEST.**
r.) Skaunic, V. (1st Med. Clin., Charles U., Hradec Králové,
echoslovakia) and V. Nerad. *Deutsch Z Verdau Stoffwechselkr*
(1/2):21-26, 1967.

P retention 30 min after injection was determined on 50 men,
d 18-90 yr, with no history of liver disease. The upper limit
the normal range was 6.8% among subjects 35-40 yr old.
is is below the 10% that is usually considered normal. In
5th decade BSP retention begins to increase progressively
h increasing age. For subjects over 35 the upper normal
it can be expressed by the formula 0.2 x age in yr. BSP
ention at 30 min was also determined on a randomly selected
up of 30 patients with clinical or histological signs of liver
ease. When 10% was considered the upper normal limit, 12
l retention values within normal limits. When the age factor
s taken into consideration, only 9 had normal BSP retention
ues.

53 **BLOOD GLUCOSE AND AMYLASE VALUES AFTER GLUCOSE LOADING IN**
SEASES OF THE EXOCRINE PANCREAS: COMPARISON
TH THE SECRETIN TEST. *(It.)* Bertero, R. (Inst. Gen.
d. Clin., U. Turin, Italy), S. Gabasio, A. Pera and M. De La
rre. *Minerva Gastroent* 13(2):36-42, 1967.

od glucose and amylase values were determined on 66 sub-
ts before and 30, 60, 90, and 120 min after ingestion of
5 g/kg of glucose. Parallel increases in blood glucose and
ylase were found in all 12 normal subjects and in 10 of 15
ients presumed to have diseases in which the exocrine pan-
as were not involved. Curves characteristic of nonobstructive
ncreatic disease were found in the remaining 5 patients who
l silicosis, duodenitis, neuroautonomic dystonia, biliary dys-
esia, and diencephalic syndrome with epilepsy. Abnormal
od glucose and amylase curves were found in 2 patients
h possible secondary involvement of the pancreas. These
luded 1 with rectocolitis and 1 with a duodenal ulcer dating
k 20 yr. Amylase curves were flat or diverged from glucose
ves in 23 of 25 patients with chronic pancreatitis. Glucose
ves were diabetic in 5 cases and prediabetic in 6. Amylase
ves were normal in 3 patients with pancreatic neoplasms, and
cose curves were diabetic in 2. The secretin test, performed
17 of the 25 patients with chronic pancreatitis, showed that
ximum bicarbonate concentrations were abnormally low in
cases and the total quantity of bicarbonate was low in 13
es. The total quantity of pancreatic juice secreted was
normally low in 9 cases. The most abnormal secretin tests
re seen in 3 patients with calcareous pancreatitis.

7854 **DETERMINING THE PEPTIC ACTIVITY OF THE GASTRIC JUICE WITH ^{131}I-LABELED ALBUMIN.** *(Fr.)* Quoidbach, A. (Cantonal Hosp., Geneva, Switzerland), B. Eggli and R. Busset. *Gastroenterologia (Basel)* 108(5):275-282, 1967.

A study was made of the effect of reagent concentrations and experimental conditions on the sensitivity of Klotz and Duvall's method for determining pepsin activity in gastric juice. The percentage of free iodine in a preparation of ^{131}I-labeled human serum albumin remained constant for at least 1 month and did not increase when incubated for 1 hr at 37C in a 6% albumin solution at pH 2. Maximum pepsin activity was observed between pH 1.5 and 3. At concentrations between 0.1 and 4 mg/ml, pepsin activity did not increase linearly with the pepsin concentration, but results were reproducible when the same preparation of ^{131}I-labeled albumin was employed. The quantity of albumin digested did not depend on its concentration when 0.1 mg/ml of pepsin was used. Optimum activity was obtained with a 6% albumin preparation. The reproducibility of this method was good (coefficient of variance 2.8%). In several hundred determinations performed on adults the pepsin concentration was never higher than 4 mg/ml. After stimulation of gastric secretion with Kay's test higher values were found in several children with hiatus hernias.

7855 **THE ROLE OF GASTROCAMERA IN THE DIAGNOSIS OF MARGINAL ULCER.** *(E.)* Benfield, J. R. (U. Hosp., U. Wisconsin, Madison), Y. Tanaka and J. Morrissey. *Arch Surg* 95(4):609-614, 1967.

The gastrocamera was used a total of 31 times in 17 patients (16 men and 1 woman, 41-70 yr old) with demonstrable marginal ulcers following gastrectomy. Fourteen ulcers were either on the stomal rim or in the jejunum. Two cases in which the gastrocamera contributed significantly to management are presented in detail. It is felt that gastrocamera photography is a valuable adjunct in the detection of marginal ulcers. It does not replace X-ray examination for the initial evaluation of patients suspected of marginal ulceration, but it usually makes follow-up X-rays unnecessary. The technique probably can and should replace older gastroscopic methods.

7856 **A NEW METHOD FOR DETECTION AND MEASUREMENT OF INTRINSIC-FACTOR ANTIBODIES.** *(E.)* Ashworth, L. A. E. (Dept. Med., Stanford U., Palo Alto, Calif.), J. M. England, J. M. Fisher and K. B. Taylor. *Lancet* 2(7527):1160-1164, 1967.

A new *in vitro* technique is described which utilizes homogenates of guinea pig small intestinal mucosa to detect both of the known types of antibody to Castle's intrinsic factor found in pernicious anemia. The technique involves the use of ^{57}Co-labeled vitamin B_{12}, charcoal assay of gastric juice vitamin B_{12} binding, and titration with goat antisera, pernicious anemia sera, and guinea pig small-intestinal mucosal homogenate. Preliminary results indicate that the technique is sensitive and the results are reproducible. This should provide a useful means of determining the course of development of the 2 types of intrinsic antibody in pernicious anemia and thus help to increase the understanding of this disease.

7857 **APPRAISAL OF THE APPLICATION OF ^{99m}Tc IN THE ASSESSMENT OF GASTRIC**

FUNCTION. *(E.)* Irvine, W. J. (Royal Infirm., Edinburgh, Scotland), A. G. Stewart, G. P. McLoughlin and P. Tothill. *Lancet* 2(7517):648-653, 1967.

The secretion of ^{99m}Tc into the gastric juice was studied in 5 patients with pernicious anemia, 2 with latent pernicious anemia but no actual anemia, 11 with thyroid anomalies, and 1 healthy control. Following a 12-hr fast, gastric acid secretion was collected for 1 hr before and 1 hr after s.c. injection of histamine acid phosphate (0.04 mg/kg) and analyzed with regard to volume, pH, acid content and intrinsic factor content. A tracer dose of up to 500 μC ^{99m}Tc-pertechnetate i.v. was given at the beginning of the basal collection of gastric juice. Although the ^{99m}Tc content of the gastric aspirate correlated significantly with each measure of gastric function in the post-histamine hr, the correlation was not significant in the basal hour. The isotope was also secreted in the saliva. Gastric uptake at 1 hr ranged from 1.8-13.3 (% of the dose), with ^{99m}Tc uptake increasing with increasing acid output. Although gastric scans showed 10.2% isotope uptake after 2 hr and 3.7% after 1 hr without histamine stimulation and without gastric aspiration, the uptake at 2 hr (4.7%) was the same as that at 1 hr (4.5%) with histamine stimulation. With gastric aspiration the isotope content was 5.6 and 11.7% for the pre- and post-histamine hours, resp., but only 6.2% after 2 hr. The average half-life of ^{99m}Tc in the blood was 2-7 hr. In 1 patient the isotope uptake was completely blocked by previous oral ingestion of 200 mg perchlorate. The results suggest that after histamine stimulation, much of the gastric juice may be lost via the duodenum if aspiration is not carried out.

7858 **CLINICAL INVESTIGATION OF THE PORTACAVAL SHUNT. III. THE DIAGNOSIS OF ESOPHAGEAL VARICES.** *(E.)* Peternel, W. W. (Dept. Med., U. California, Los Angeles), A. E. Dagradi, A. I. Rogers, H. M. Nadal, E. B. Perrin and F. C. Jackson. *JAMA* 202(12):1081-1084, 1967.

In 83 patients with esophageal varices (due in at least 78 cases to hepatic cirrhosis), an esophagogram after administration of barium sulfate, an endoscopic examination of the esophagus, and a portogram after percutaneous injection of an opaque material into the spleen were compared for diagnostic value in demonstrating esophageal varices. The diagnosis was established by esophagoscopy in 81, by splenoportography in 61, and by barium esophagogram in 55. Large varices were frequently missed by barium esophagograms as well as by splenoportograms. The mean splenic-pulp pressure was similar when patients with endoscopically demonstrated varices and normal portograms were compared with those whose portogram showed varices. Moreover, there was no apparent relation between portal pressure and variceal size. A time lapse between studies of as long as 2 months had no apparent influence on the incidence of positive diagnoses obtained.

7859 **DIAGNOSIS OF DIARRHOEA IN GENERAL PRACTICE: BACTERIOLOGICAL "SELF-HELP".** *(E.)* Knox, J. D. E. (191 Crewe Road North, Edinburgh, Scotland), A. R. Laurence, G. MacNaughtan and A. A. Robertson. *Lancet* 2(7531):1392-1394, 1967.

By inoculating media on MacConkey plates at the time of consultation and incubating the plates overnight, 4 family doctors examined 637 specimens from 589 episodes of diarrhea. It was noted that 2/3 of the specimens were of no pathologic significance. Of the remainder, 50% were shown by a bacteriological laboratory to be associated with shigella (106 cases) or salmonella (1 case). The ready availability of such a simpl technique has significantly increased the rate of detection of cases of bacillary dysentery.

7860 **UMBILICAL VEIN PORTOGRAPHY: ITS VALUE IN THE DIAGNOSIS OF EXTRAHEPATIC PORTAL VEIN OBSTRUCTION AND IN OTHER APPLICATIONS.** *(E.)* Pochaczevsky, R. (Jewish Hosp. Brooklyn, N. Y.), W. S. Calem and R. M. Richter. *Radiology* 89(4):868-873, 1967.

The technique and advantages of umbilical vein portography are presented. A case of duodenal bulb varices with extrahepatic portal obstruction, in which umbilical vein portograpl played an important diagnostic role, is discussed. Examples of other patients in whom this procedure was used are also presented. It is suggested that umbilical vein portography be performed whenever duodenal varices are found, in order to determine whether extrahepatic portal obstruction is present.

7861 **EXTERNAL GUIDED PASSAGE OF AN INTESTINAL INTUBATION TUBE.** *(E.)* Hanafee W. N. (UCLA Sch. Med., Los Angeles, Calif.) and M. Weiner. *Radiology* 89(4):1100-1102, 1967.

A guided catheter system such as those currently in use for arteriography is presented for use in intestinal intubation. The Muller guided system is described as useful for traversing the pyloric canal and duodenal loop. Using arterial catheterization techniques, a Seldinger-type guide wire is then passed through the Muller catheter and an exchange is made for eith a duodenal drainage tube, a decompression tube for intestinal obstruction, or an intestinal mucosal biopsy instrument. Thes systems can be used to reduce the time of performing the procedure and minimize discomfort to the patient during diagnostic, as well as therapeutic, intestinal tube placement. No complications have occurred to date.

7862 **QUANTITATIVE VARIATIONS IN SERUM GLYCOPROTEINS DURING PATHOLOGICAL PROCESSES: DETERMINATION OF ACID α_1-GLYCOPROTEIN, Gc, AND α_2-MACROGLOBULIN BY RADIAL IMMUNODIFFUSION.** *(Ger.)* Cleve, H. (Inst. Human Genet., U. Marburg/ Lahn, Germany) and G. Strohmeyer. *Klin Wschr* 45(20):1051-1054, 1967.

Radial immunodiffusion was used to determine acid α_1-glycoproteins, group specific serum components (Gc), and α_2-macroglobulins in serum from 169 patients with 42 different diseases These included 42 with liver diseases, 35 with gastrointestinal carcinomas, 15 with inflammatory diseases of the large intestine, and 3 with miscellaneous gastrointestinal diseases. Acid α_1-glycoprotein concentrations exceeded 200 mg% in most of the patients with carcinomas, but were also elevated in other conditions so that this is of little diagnostic value. The low values for acid α_1-glycoproteins found in cirrhosis indicate that this protein is probably synthesized in the liver. Group-specific serum components were reduced in patients with severe impairments in liver synthesis, particularly in those with cirrhosis, and might be used as a criterion for evaluating parenchymal ver damage. Serum concentrations of α_2-macroglobulin were evated in patients with Zollinger-Ellison syndrome and in some ncer patients and patients with ulcerative colitis. In contrast, rrhotics had normal or increased α_2-macroglobulin concentra- ons, suggesting that this protein is not synthesized in the liver. eterminations of the α_2-macroglobulin fraction are of little iagnostic value since this protein fraction is increased in a eat variety of diseases.

863 NONSURGICAL TEST FOR THE DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE.

(E.) Tobon, . (Baltimore City Hosps., Maryland), N. C. R. W. Reid, J. L. albert and M. M. Schuster. *New Eng J Med* 278(4):188-194, 968.

manometric technique is described for the diagnosis of irschsprung's disease without the necessity of deep-muscle rec- l biopsy. Separate pressure recordings were obtained from the ternal and external anal sphincters by a double balloon de- ce and from the rectum by a 3rd balloon. Simultaneous elec- omyographic activity was recorded from the external sphincter y a concentric needle electrode. In 34 normal subjects, tran- ent distension of the rectal balloon produced reflex relaxation f the internal sphincter (smooth muscle) and contraction of e external sphincter (striated muscle), with a simultaneous in- ease of electrical activity in the external sphincter. In 10 pa- ents with Hirschsprung's disease, there was contraction of the ternal sphincter instead of relaxation; external sphincter re- onses were normal. In 31 patients with idiopathic megacolon, oth internal and external sphincter responses were normal, in- icating that the abnormal contractile response found in irschsprung's disease is characteristic of this disorder and not f megacolon in general. This contractile response may contri- ute to the obstructive symptoms in Hirschsprung's disease.

864 EVALUATION OF PHENYL ACETATE ESTERASE ACTIVITY AS AN INDEX OF LIVER DAM- GE.

(E.) Takahashi, Y. (Osaka U. Med. Sch., Japan), I. oyama, F. Ito and Y. Yamamura. *Clin Chim Acta* 18(1):21- 2, 1967.

he phenyl acetate esterase activity was determined in the serum nd liver homogenates from male dd0 mice treated with various hemical toxins and in serum from 380 randomly selected pa- ients. Fractionation studies of mouse liver homogenates howed the highest phenyl acetate esterase activity in the micro- omal fraction; during starch block electrophoresis, this activity nigrated with the same mobility as serum albumin. The nor- nal specific phenyl acetate esterase activity was 25.0 ± 1.0 Eq NaOH/mg protein in mouse liver homogenates and 69.8 ± 5.8 μEq in human serum. The homogenate value was reduced o 11.7 μEq 24 hr after administration of CCl_4 (1 ml/kg i.p.) nd 9.9 μEq after allyl formate (0.1 ml/kg i.p.), but ethiona- nide (0.25 g/kg i.p.) had no effect. Of various agents tested, nly glutathione was able to counteract CCl_4 damage and othing was able to counteract the effect of allyl formate. The nean serum phenyl acetate esterase activity was sharply reduced to 35.1 ± 8.8 μEq) in cirrhosis, but ranged from 52.4 to 66.1 Eq in patients with other diseases, including hepatitis. Values elow 40 μEq were obtained in only 19 of the 380 patients, ncluding 8 of 10 cirrhotics and only 2 of 27 with hepatitis. lthough phenyl acetate esterase may therefore be useful for he diagnosis of cirrhosis, it showed a poor correlation with ther liver function tests.

7865 ANGIOGRAPHY FOR PREOPERATIVE DIAGNOSIS OF PANCREATIC DISEASES.

(Ger.) Bedacht, R. (Surg. Clin., U. Munich, Germany) and H. Zimmermann. *Bruns Beitr Klin Chir* 215(3):257-274, 1967.

Five case reports are presented for patients subjected to angiographic examination of the celiac trunk and the superior mesenteric artery. These include 4 patients with pancreatic neoplasms in which a definite diagnosis could not be established by clinical or routine radiological methods. Although simultaneous angiography of the celiac trunk and superior mesenteric artery usually gives the best results, this was not true in 2 of these cases. In 1 patient with an inoperable tumor of the head of the pancreas, angiography of the celiac trunk showed only changes in the wall of the common hepatic artery, indicating the probable presence of a tumor in the hepatic portal. However, angiography of the superior mesenteric artery revealed the presence of collateral circulation through the gastroduodenal artery to the proper hepatic artery which was not seen with simultaneous angiography. In another patient suspected of having a pancreatic cyst, angiography of the celiac trunk showed changes characteristic for tumors although the site could not be definitely established. In 1 case angiography was used to rule out a possible tumor in a patient with pancreatitis.

7866 THE BSP TEST: THE PRESENCE OF JAUNDICE AS A DISTINGUISHING FACTOR IN THE DIFFERENTIAL DIAGNOSIS OF LIVER DISEASES.

(It.) Barbi, G. (Hosp. Inst., Milan, Italy), D. Sardini and G. Tallone. *Minerva Med* 58(79):3922-3926, 1967.

A comparison was made of semilogarithmic curves for BSP blood levels determined on 2 patients who both had liver metastases of gastrointestinal tumors and identical clinical symptoms, but 1 had jaundice and the other did not. The slope of the curve was almost normal for the anicteric patient and only a presumptive diagnosis was made before autopsy, while the jaundiced patient had definitely abnormal slopes and the diagnosis was established on the reciprocal relations between them. A similar comparison of BSP curves was made for patients with: 1) preascitogenous hypertrophic cirrhosis without jaundice, 2) ascitogenous cirrhosis, hepatic coma, but no jaundice, and 3) ascitogenous cirrhosis with jaundice and a relatively favorable prognosis. Slopes were abnormal only for (3). It is concluded that the BSP test is most reliable in patients who have jaundice.

7867 THE HOYT-MORRISON TEST AND TRANSAMINASE VALUES COMPARED IN HUMAN VIRAL HEPATITIS AND IN THE HOSPITAL POPULATION.

(It.) Passoni, M. (Munic. Hosp., Pordenone, Italy) and N. Delendi. *G Mal Infett* 19(12):922-925, 1967.

The Hoyt-Morrison test was run and serum transaminase activities were determined on 53 patients with acute icterigenic hepatitis, 23 normal controls with histories of acute hepatitis, and 257 normal controls with no histories of acute viral hepatitis. The Hoyt-Morrison test was positive only in 9 hepatitis patients and in 3 controls with histories of hepatitis but in 59 controls with no histories of hepatitis. Serum transaminase activities were normal in all but 1 of the controls. No data are given for the transaminase activities in hepatitis patients. These results indicate that the Hoyt-Morrison test is of little value in the diagnosis of viral hepatitis.

7868 **INTRAVENOUS PANCREATOGRAPHY WITH ERYTHROSIN B IN CLINICAL PRACTICE. STUDY OF 30 PRELIMINARY OBSERVATIONS.** *(Fr.)* Ledoux-Lebard, G. (Cochin Hosp., Paris, France), F. Heitz, A. Béhar, J. Rosier, L. Leger and F. Siguier. *Presse Med* 75(39):1943-1946, 1967.

Pancreatic tomograms were obtained from 15 controls and 15 patients with pancreatic diseases after injection of 800-1000 ml O_2 into the retroperitoneal space, 200-300 ml O_2 into the peritoneum, and 2 mg of erythrosin B in a 10% buffered solution i.v. In controls the contrast medium became visible after 20 min, maximum opacification appeared after 40 min, and opacity disappeared and the duodenum appeared after 65 min. Tomograms from 5 patients with chronic calcareous and noncalcareous pancreatitis showed very few morphological changes, but opacification occurred much sooner, reaching a maximum after 20 min and disappearing after 40 min. Pancreatic stumps also became opaque after 20 min in tomograms from 6 patients who had undergone partial pancreatectomies. The pancreatic stump showed little or no opacity in patients with pancreaticojejunal anastomoses which were functioning well, but after 40 min the jejunal loop did become opaque. Other abnormalities demonstrated by this procedure included a hydatid cyst, a pseudocyst, and pancreatic trauma.

7869 **CYTOLOGICAL STUDY OF TUMOR CELLS IN THE PERIPHERAL BLOOD: REPORT OF MALIGNANT CELLS IN THE PERIPHERAL BLOOD OF A PATIENT WITH CARCINOMA OF THE STOMACH.** *(Jap.)* Takeuchi, M. (Sinkori Hosp., Hirakata, Japan) and M. Mashimo. *Med J Mutual Aid Ass* 17(3):93-94, 1968.

7870 **BEHAVIOR OF SOME LIVER FUNCTION TESTS IN EXPERIMENTAL LIVER INJURY.** *(Pol.)* Kuźmicka, J. (Inst. Lab. Diag., Acad. Med., Lodz, Poland), W. Torzecka, Z. Torzecki, A. Legiewski and I. Cyperling. *Pol Arch Med Wewnet* 39(4):437-442, 1967.

7871 **FIBEROPTIC EXAMINATION OF THE SMALL INTESTINE IN A CASE OF FAMILIAL TELANGIECTASIA. [CASE REPORT]** *(E.)* Sweeting, J. G. (Harkness Pavillion, New York, N. Y.). *Gastroint Endosc* 14(3): 152-153, 1968.

7872 **GASTROSCOPY IN MENETRIER'S DISEASE. [CASE REPORT]** *(E.)* Baker, W. G. (Cornell U. Med. Coll., New York, N. Y.), M. Kolodny, J. L. Colker, and H. Goldin. *Gastroint Endosc* 14(4):209-213, 1968.

7873 **THE IMPACT OF ENDOSCOPY IN ACUTE UPPER GASTROINTESTINAL HEMORRHAGE IN A SMALL HOSPITAL.** *(E.)* Jenkins, D. W. (USAF Acad. Hosp., Colo.). *Gastroint Endosc* 14(4):217-219, 1968.

7874 **POLAROID ENDOPHOTOGRAPHY.** *(E.)* Boyce, W. Jr. (Walter Reed Gen. Hosp., Washington, D. C.) and D. Bishop. *Gastroint Endosc* 14(4):186-189, 1968.

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7876 **COPROLOGICAL DIAGNOSIS OF SCHISTOSOMIASIS: EVALUATION OF QUANTITATIVE TECHNIQUES.** *(E.)* Katz, N. (René Rachou Res. Ctr., Caixa Brazil) and G. Chaia. *Rev Inst Med Trop S Paulo* 10(5):295-298, 1968.

7877 **COLOUR-SCANNING AS AN AID IN THE DIA NOSIS OF LIVER DISEASES.** *(E.)* Pedroso de Lima, J. J. (Radioisotope Lab., U. Mozambique, Lourenco Marques), A. S. Rego, F. Antunes Dias and A. Forjaz. *Ann Trop Med Parasit* 61(3):360-364, 1967.

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7879 **OPERATIVE CHOLANGIOGRAPHY.** *(E.)* Boy C. E. (Shreveport, La.). *J Abdom Surg* 10(3):87 91, 1968.

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7881 **A MODIFICATION OF THE ROY-CHOUDHUR CAPSULE FOR INTESTINAL BIOPSY.** *(Por.)* Castro, L. P. (Fac. Med., Fed. U. Minas Gerais, Belo Horizont Brazil). *Arq Gastroent* 5(1):29-31, 1968.

7882 **CLINICAL AND RADIOLOGICAL DIAGNOSIS OF PEPTIC ULCERS IN CHILDREN OF PRESCHOOL AND SCHOOL AGE.** *(Rus.)* Gerlants, A. I. (1st Munic. Clin. Hosp., Odessa, USSR). *Pediatriia (Moskva)* 46(1 65-66, 1967.

7883 **BILIRUBIN AND ITS FRACTIONS DURING VIRAL HEPATITIS IN CHILDREN.** *(Rus.)* Mashina, L. Ia. (Leningrad Inst. Pediat. Med., USSR). *Pediatr (Moskva)* 46(12):39-42, 1967.

7884 **EXPERIMENTAL STUDIES ON THE INTERPRETATION OF THE RESULTS OF WIRSUNGOGRAPHY.** *(Fr.)* Mallet-Guy, P. (Ed. Herriot Hosp., Lyon, France), J. Feroldi, J. Murat, A. Ray and F. Abboud. *Bull So Int Chir* 27(2):114-118, 1968.

7885 **PREOPERATIVE ENDOSCOPIC CHROMOGRAPHY OF THE CORPUS-ANTRUM BORDER.** *(Ger.)* Ottenjann, R. (Med. Clin. Polyclin., U. Erlangen-Nuremberg, Germany), H. Peters and K. Elster. *Materia Med Nordmark* 20(5):264-269, 1968.

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rcus, T. Popesco, I. Vasiliu and M. Vladareanu. *Rev Roum
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mania), M. Gidaly, M. Marina and M. Farcasanu. *Sem Hop
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mania) and N. Parau. *Rev Roum Med Intern* 4(3):175-185,
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gers, France), F. Joubaud, J. Cheguillaume, A. Malinovsky,
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AGNOSIS AND EVALUATION OF THE COURSE OF VAR-
US JAUNDICE SYNDROMES.** *(Rus.)* Sedlovets, M. P.
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R MOTILITY.** *(Rus.)* Kitaiev, V. V. (I. M. Sechenov 1st
scow Med. Inst., USSR). *Sovet Med* 31(3):66-69, 1968.

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sp., London, England). *Actualities Hepatogastroent Hotel
eu* 4(4):A217-A231, 1968.

96 **THE COMPLEX FUNCTIONAL, MORPHOLOGICAL, CLINICAL AND ROENTGENOLOGICAL
VESTIGATION OF THE STOMACH.** *(Rus.)* Britsis, F. A.,
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ıda, B. A. (Riga Med. Inst., USSR) and S. A. Berzin.
irurgiia (Moskva) 44(3):35-37, 1968.

98 **TRANSCUTANEOUS-TRANSHEPATIC CHOLANGIOGRAPHY.** *(Rus.)* Bragin, F. A. (P. Lumumba U. Friendship Peoples, Moscow, USSR). *Khirurgiia (Moskva)* 44(3):68-71, 1968.

7899 **PHENYLACETYLGLUTAMINE EXCRETION IN LIVER DISEASES.** *(Rus.)* Mardashev, S. R. (I. M. Sechenov 1st Moscow Med. Inst., USSR), S. S. Debov and A. P. Khokhlov. *Sovet Med* 31(3):27-30, 1968.

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7902 **CLINICAL EXAMINATION OF A VOMITING INFANT.** *(Fr.)* Mallet, R. *Rev Prat* 19(1):8-14, 1969.

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7904 **INTRADERMAL REACTION WITH ESCHERICHIA COLI ALLERGEN IN PERSONS WITH DISEASES OF THE LIVER AND BILIARY TRACT.** *(Rus.)* Kirilenko, V. A. (N. I. Pirogov Med. Inst., Vinnitsa, USSR). *Sovet Med* 31(6):108-109, 1968.

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7906 **EXPERIMENTAL *IN VIVO* PHOTOGRAPHIC MAGNIFICATION ANGIOGRAPHY OF THE CANINE KIDNEY AND BOWEL.** *(E.)* Bosniak, M. A. (Boston U. Med. Ctr., Mass.), M. H. Farmelant, C. Bakos, C. A. Hasiotis and L. F. Williams Jr. *Invest Radiol* 3(2):120-127, 1968.

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Kowalski, W. (Munic. Hosp., Piotrkow Tryb., Poland). *Pol Przegl Radiol* 32(3):339-342, 1968.

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7912 EVALUATION OF THE ROENTGENOANATOMICAL INJECTION METHOD FOR INVESTIGATING THE BLOOD SUPPLY OF THE STOMACH. *(Rus.)* Druzhkov, B. K. (Kazan Inst. Postgrad. Med., USSR). *Khirurgiia (Moskva)* 43(12):62-66, 1967.

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7916 GASTROCAMERA AND GASTROFIBERSCOPIC (GTF) EXAMINATIONS ON 823 CASES. *(E.)* Wang, T. H. (Coll. Med., Natl. U. Taiwan, China) and J. L. Sung. *J Formosa Med Ass* 67(10):404-409, 1968.

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7919 THE USE OF RADIOISOTOPES IN STUDIES OF THE GASTROINTESTINAL TRACT. *(E.)* Shapiro, B. *J Albert Einstein Med Ctr* 15(3):263-269, 1967.

7920 SEROLOGICAL TESTS IN AMOEBIASIS. *(E.)* Powell, S. J. (Amebiasis Res. U., U. Natal, South Africa). *Indian Pract* 21(10):646-648, 1968.

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See also: 7692,7693,7824,8171,8217,8229,8305,8365,8366,8392,8427,8447,8474,8593,8813

GASTROINTESTINAL DISEASES

ESOPHAGUS

7982 **THE ANTRUM CARDIACUM IN INFANCY: ITS SITE AND SHAPE IN NORMAL INDIVIDUALS AND IN HIATUS HERNIA.** *(E.)* Holthusen, W. (Children's Hosp., Hamburg, Germany). *Ann Radiol* 10(7/8):529-535, 1967.

Examples of the radiological appearance of the gastroesophageal junction are presented for both 179 normal infants and 58 infants suffering from hiatus hernia (1st and 2nd degree). It was shown that in infancy, the gastroesophageal junction is normally situated just above the hiatal pinchoock. The 'antrum cardiacum', interpreted as a small tube-like or cone-shaped upper portion of the stomach, identical with Scharzki's 'empty segment' and normally lying within the hiatel channel, was found to be a useful landmark for the diagnosis, classification, and prognosis of hiatus hernia.

7983 **ADJUNCTIVE TECHNIQUES IN THE OPERATIVE MANAGEMENT OF SLIDING ESOPHAGEAL HIATUS HERNIA.** *(E.)* Smith, R. S. (Boise, Idaho) and G. E. Talboy. *Northwest Med* 67(1):21-26, 1968.

A number of adjunctive techniques, varying in their rationale and their indications, are proving effective in the operative management of sliding hiatal hernia associated with reflux esophagitis. Based on 31 operative procedures on 30 patients, it is concluded that the most important of these supplementary procedures are concerned with the infradiaphragmatic fixation of the gastroesophageal junction, the reinforcement of the cardioesophageal sphincter mechanism, and the reduction of gastric secretory activity. When there is little shortening of the involved esophagus, the corrective surgical requirements are minimal. When chronic cases show cicatricial narrowing of the esophageal lumen, a radical operation may be necessary. Gastric fundoplication specifically increases the competence of the cardioesophageal sphincter. This ancillary procedure must be applied precisely, however, if complications from its use are to be avoided.

7984 **EXPERIENCE WITH TANNER'S SUBCARDIAL GASTRIC RESECTION FOR THE TREATMENT OF BLEEDING ESOPHAGEAL VARICES.** *(Ger.)* Gruenagel, H. H. (Surg. Clin., U. Freiburg/Breisgau, Germany) and M. Hanke *Bruns Beitr Klin Chir* 216(4):306-309, 1968.

A Tanner gastrectomy with an abdominal approach was used on 11 patients with bleeding esophageal varices. Splenectomies were performed on all 8 patients who had not undergone this procedure before. Of the 5 patients who underwent emergency surgery for acute bleeding, 1 died immediately after surgery of recurrent bleeding and 2 died of liver failure; all 3 of these patients were cirrhotics. The other 2 patients, both of whom had prehepatic blocks, have had no recurrences. Five of the 6 remaining patients had intrahepatic blocks and one had a prehepatic block. Three of the 5 died of recurrent bleeding 9 months to 2 yr after surgery and 1 died of cirrhosis 1.5 yr after surgery. Only 1 patient with an intrahepatic block and 3 with a prehepatic block were alive at the time of report.

7985 **FASCIA LATA GRAFT REPAIR OF ESOPHAGEAL HIATAL HERNIA.** *(E.)* Brain, R. H. F. (Guy's Hosp., London, England) and J. Maynard. *Amer J Surg* 115(4):488-501, 1968.

Following a review of the pathologic anatomy of hiatus hernia and of the complications and methods of its surgical treatmen a repair procedure is described in which a fascia lata graft is sutured to the under surface of the diaphragm. The approach is through a transthoracic incision on the left side and circumferential incision in the diaphragm. During the last 10 yr, 28 patients had hiatal hernia repair by this procedure, the develo ment of which was prompted by poor results with the Allison technique. The 100 patients (29 males and 71 females, rangin in age from childhood to 85 yr) operated on in the period 1957-63 were followed up 2-6 yr after surgery; complete evalulation with radiologic studies was possible in 89. Although there were 5 recurrent hernias, 4 failed repairs due to esophageal shortening, and 14 patients with abnormal radiographic signs on detailed screening of reflux, 29 of 89 (32%) fully eva uated patients were free of all signs and symptoms. Postopera tive dysphagia of varying degrees of severity accounted for m of the poor over-all results (35 cases). Analysis of the causes of the dysphagia (too narrow a hiatus, mechanical fixation of the cardia, oversecure cardiac mechanism, alterations in esoph geal tonus) has led to a significant improvement in more recen surgical results. Overtight suturing of the graft around the es agus is avoided, the esophageal wall is no longer sutured to th new fascial edges of the hiatus, and resuture of the edges of t phrenoesophageal cuff 1.5 cm anterior and lateral to the new hiatus allows mobility of the cardia.

7986 **THE PREVENTION OF EXPERIMENTALLY I DUCED REFLUX BY ELECTRICAL STIMULATION OF THE DISTAL ESOPHAGUS.** *(E.)* Ellis, F. (Guy's Hosp., London, England), T. V. Berne and K. Settevig. *Amer J Surg* 115(4):482-487, 1968.

Direct observations in normal dogs subjected to thoracotomy vealed that application of electric current (4 volts, 20 cycles/s 3 msec duration) via single pairs of electrodes attached to the esophageal wall produced a well-defined esophageal contractio ring over a distance of 3 cm. With these parameters of stimul tion and intraluminal pressure of 23 cm H_2O immediately developed, dropping to 5 cm H_2O in 2.5 hr; a further increase in strength of the stimulus to 6 volts had little effect. With two pairs of electrodes stimulated alternately, the mean intralumin pressure fell from 23 cm H_2O only to 13 cm H_2O in 2.5 hr. Radiologic studies demonstrated that continuous stimulation with alternating pairs of electrodes produced a sustained contraction in the esophagus for 5 hr, without the mechanical con striction observed using 1 pair of electrodes. In dogs with gastroesophageal incompetence produced either by a combination of cardioplasty and upward displacement of the hiatus or by resection of the cardia, similar low-energy electrical stimulatio applied to the inferior esophageal muscle via 2 pairs of electro produced vigorous and sustained contraction of the esophagus for 15 min. Pressure studies 2-4 weeks after recovery confirm that a zone of increased pressure could be maintained for several hours. The prevention of reflux by this method also was demonstrated by radiographic studies. With the application of of current competence was maintained for 3 hr, even when the animal was in a position known to produce reflux. No change in cardiac activity, movement of the diaphragm or peristaltic a tivity of the stomach occurred during stimulation. No macro- or microscopic signs of esophageal damage were found after electrodes had been present for several weeks. This pro- re may find widest clinical application in the prevention eflux after cardiomyotomy for achalasia.

7 **THERAPEUTIC EFFECT OF A HYDROPHILIC GEL CONTAINING SILICONES IN DISORDERS THE ESOPHAGUS.** *(Sp.)* Llorens Salvans, V. (Clin. Hosp., elona, Spain). *Rev Esp Enferm Apar Dig* 27(7):1053-1056, 8.

ty patients (17 men and 13 women, aged 17-76 yr) with a ety of esophageal disorders were given 3-5 teaspoons/day n emulsion of methylpolysiloxane in a hydrophilic gel. patients had pyrosis and all but 2 had radiological signs of ial insufficiency. Treatment was continued for 4-45 days. ptomatic improvement was noted in 23 cases, very slight rovement in 13, and no improvement in 3. The best results e obtained when esophageal reflux was caused by a recent hanical disorder and when cardial insufficiency was not as- ated with other underlying disorders. Patients with diaphragic hernias did not respond to therapy as well as others. side effects were observed.

38 **REPAIR OF THE ESOPHAGEAL MUSCULATURE WITH SYNTHETIC MATERIALS AND FREE N GRAFTS.** *(Rus.)* Nechiporuk, V. M. (Ternopol Med. ., USSR). *Eksp Khir Anest* 13(1):3-5, 1968.

xperiments on 54 dogs, defects in the musculature of the phagus were repaired either by means of free full-thickness grafts or with two brands of dacron, Terylene and Lavsan. 47 animals surviving the operation were observed for up to days. Dogs given free skin grafts recovered quickly. Adher- to the surrounding tissues, the autotransplants filled in the ect in the muscle tissue and prevented the mucosa from pro- ling or perforating. The esophageal wall around the trans- t was slightly thicker and denser than usual but was suffi- tly elastic. Neither deformation nor constriction occurred. ological studies showed that the skin fragments underwent phological changes, with the edges manifesting necrobiosis gradual decay. Tissues around the synthetic materials, on other hand, showed granulation and eventual fibrosis, pre- ting the desired adherence to natural tissue. Such synthetic splants were gradually surrounded by connective tissue and e eventually rejected.

39 **RETROSTERNAL REPLACEMENT OF THE ESOPHAGUS BY THE LEFT COLON.** *(Sp.)* a Azurdia, R. (Roosevelt Hosp., Guatemala City). *Rev Col Guatemala* 19(3):188-197, 1968.

method of Luna and Ernst was used for complete retrosternal replacement of the esophagus by a segment of left colon in cases and partial intrathoracic replacement in 2 cases. These patients included 10 with esophageal carcinomas, 6 with ex- sive stenosis caused by swallowing caustic, 6 with peptic osis, and 1 with a benign tumor. Complications developed cases: 2 fistuals, 1 of which was followed by complete ration of the anastomosis; rupture of the abdominal incis- in a patient with severe malnutrition; 2 cases of intermittent operative gastric distension, one of which necessitated a roplasty for stenosis in the area of the gastrocolostomy and other a pyloroplasty; and an intestinal obstruction produced dhesions 21 days after surgery. There were no operative hs.

7990 **REPAIR OF EXPERIMENTAL HIATAL HERNIA IN DOGS.** *(E.)* Earlam, R. J. (Mayo Clin. Found., Rochester, Minn.) and F. H. Ellis Jr. *Arch Surg* 95(4):585-594, 1967.

Hiatal hernias with a longitudinal myotomy through the gastroesophageal sphincter were produced in 25 mongrel dogs. Five different corrective surgical procedures were performed 4-6 weeks later: crural approximation and phrenoesophageal ligation fixation (Allison); crural approximation, phrenoesophageal ligament fixation, and reinforcement with Teflon mesh; fundoplication (Nissen); fundic pouch (Thal); and plication of cardia with fixation to diaphragm (Belsey). The results were assessed by esophageal manometric and mucosal potential difference studies, cinefluoroscopy, and autopsy after 6 months. The best results were obtained by crural approximation and phrenoesophageal ligament fixation. Reflux occurred most frequently after the fundic patch repair. The addition of a Teflon mesh or plication of the cardia did not improve the results, and fundoplication was ineffective in this experimental animal.

7991 **OESOPHAGEAL ATRESIA.** *(E.)* Cozzi, F. (Hosp. Sick Child., London, England) and A. W. Wilkinson. *Lancet* 2(7528):1222-1225, 1967.

Of 93 patients with esophageal atresia, 57 (65%) survived the complete treatment of the lesion; whereas 30 patients (28%) who were judged on admission to be good risks all survived, 8 of 28 who were moderate risks and 26 of 35 who were poor risks died. In an attempt to reduce the mortality, the operative treatment of 14 poor-risk patients was carried out in stages, but only 1 survived. Of 7 other poor-risk patients in whom operative treatment was completed in 1 stage, all survived. In babies with the poorest chances of survival, the results of extrapleural ligation of the fistula and gastrostomy with delayed primary anastomosis of the 2 portions of esophagus may prove better than any of the methods used in the present series.

7992 **THE PATHOGENESIS OF MEGAESOPHAGUS.** *(Ger.)* Köberle, F. (Fac. Med., U. São Paulo, Ribeirão Preto, Brazil). *Z Gastroent* 5(5):287-290, 1967.

Histological examinations were made of sections from the lower 1/3 of the esophagus in 50 Brazilian patients (35 males and 15 females, aged 15-69 yr) with megaesophagus and chronic Chagas' disease and 14 Europeans (8 males and 6 females, aged 33-83 yr) with megaesophagus. In both forms of megaesophagus there was more than a 90% reduction in the number of ganglionic cells at all 7 levels examined. These findings indicate that both forms of megaesophagus are caused by destruction of the ganglionic cells.

7993 **ESOPHAGEAL INJURIES.** *(Fr.)* Maillard, J. N. (Beaujon Hosp., Clichy, France). *Acta Chir Belg* 66(8):725-746, 1967.

The etiology and treatment of esophageal injuries are reported on the basis of experience with 28 patients. Lesions were located in the cervical region in 11 cases, in the thoracic region in 15, and in the abdominal region in 1. Firearms accidents were responsible for 2 injuries and a knife wound for 1. The remaining 25 were produced iatrogenically: 10 during esophagoscopy, 7 during esophageal dilatation, 4 during removal of a foreign body, 2 during intubation for cancer, and 1 each during gastroscopy and gastric aspiration. Although most injuries were diagnosed shortly after they were produced, 3 were found only after infection developed in the cervical mediastinum. There were 4 operative deaths: 1 in a patient with pernicious anemia who developed pulmonary and pleural infection; 2 in patients whose sutures opened up despite bipolar exclusion of the esophagus, and 1 in a patient with esophageal cancer. One patient had a nonfatal recurrence of an esophagopleural fistula soon after surgery for repair of the esophagus.

7994 **POSTEMETIC GASTROESOPHAGEAL LACERATION WITH HEMORRHAGE.** *(E.)* Hinchey, E. J. (Montreal Gen. Hosp., P.Q., Canada) and A. Hreno. *Surg Gynec Obstet* 126(2):324-330, 1968.

The case histories of 7 patients (3 men and 4 women, 29-70 yr old) with gastrointestinal hemorrhage from postemetic lacerations of the gastroesophageal junction are presented. A history of vomiting normal gastric contents before the onset of hematemesis was present in 6. Three patients had hepatic cirrhosis and 1 of the 3 had a carcinoma of the hepatic flexure. Other associated lesions were acute cholecystitis, hiatal hernia, and gastritis. Five patients required surgery to control the hemorrhage. The correct diagnosis was made preoperatively in only 2 patients, but a standard operative approach employing gastrotomy with inspection and differential packing of the stomach resulted in the correct diagnosis. The frequent association of postemetic gastroesophageal tears with other types of intraabdominal disease and the difficulty in diagnosis demand a constant awareness of their possible presence in a patient with upper gastrointestinal hemorrhage.

7995 **GASTRIC DIVISION IN THE CRITICALLY ILL INFANT WITH ESOPHAGEAL ATRESIA AND TRACHEOESOPHAGEAL FISTULA.** *(E.)* Randolph, J. G. (George Washington U. Sch. Med., Washington, D C.), W. P. Tunell and J. R. Lilly. *Surgery* 63(3):496-502, 1968.

Case reports are presented for 3 critically ill infants (2 females and 1 male) with esophageal atresia and tracheoesophageal fistula in whom proximal gastric division was performed prior to esophageal repair. The operative technique is described in detail, as well as the postoperative management of the divided stomach and the advantages of this staged procedure over the single-stage operation in premature or other poor-risk infants.

7996 **THE TREATMENT OF BLEEDING ESOPHAGEAL VARICES BY COMPLETE LIGATURE OF THE ESOPHAGUS.** *(Fr.)* Boerema, I. (Wilhelmina Hosp., Amsterdam, Netherlands), P. J. Klopper and A. A. Holscher. *Presse Med* 75(37):1849-1850, 1967.

A modification was developed of Vosschulte's procedure for total ligation of the esophagus in the esophageal hiatus of the diaphragm. The new procedure involves complete mobilization of the esophagus in the hiatus so that all veins on the external surface of the esophagus and all other veins in the hiatus and crura of the diaphragm are included. This is done in such a way that the 2 pneumogastric nerves are separated from the of the esophagus and are not ligated. Thus, it is unnecessary to perform a pyloroplasty. This procedure was performed successfully on 6 patients, aged 9-81 yr, 3 of whom had apparently fatal bleeding.

7997 **THOUGHTS ON THE SUBJECT OF STRANGULATED HIATUS HERNIA: REPORT OF CASE WITH TOTAL GANGRENE OF THE STOMACH, CURED BY SURGERY.** *(Fr.)* Tingaud, R. (Pellegrin Hosp., Bordeaux, France), P. Boissieras and M. R. Tingaud. *Ann C* 21(21/22):1263-1270, 1967.

7998 **MANAGEMENT OF HEMORRHAGES FROM ESOPHAGEAL AND GASTRIC VARICES.** *(Rus.)* Patsiora, M. D. (Sci.-Res. Inst. Clin. Exp. Surg., Min. Hlth. USSR, Moscow), K. N. Tsatsanidi and M. I. Nikolaieva. *Sovet Med* 31(3):96-103, 1968.

7999 **THE MOPHOLOGY OF EXPERIMENTALLY DUCED ESOPHAGEAL CARCINOMA IN THE RAT.** *(Ger.)* Gibel, W. (Inst. Cancer Res., German Acad. Sc Berlin). *Arch Geschwulstforsch* 31(1):5-17, 1968.

8000 **DIVERTICULA OF THE ESOPHAGUS.** *(It.)* Savino, L. (Inst. Gen. Surg. Clin., U. Pavia, Ital G. Pedroni and L. Scarabelli. *Chir Ital* 19(Suppl. 1):106-153 1967.

8001 **DIAPHRAGMATIC HERNIA OF THE STOMA COMPLICATED BY VOLVULUS: REPORT O 5 CASES.** *(It.)* Molteni, F. (Inst. Gen. Surg. Clin., U. Pavia, Italy), L. A. De Santis, G. Pedroni, C. Scotti-Foglieni and A. Caresano. *Chir Ital* 19(Suppl. 1):154-181, 1967.

8002 **CHRONIC TRAUMATIC DIAPHRAGMATIC HERNIA.** *(Rus.)* Makhov, N. I. (Moscow Med. Stomatol. Inst., USSR) and K. K. Merdenov. *Khirurgiia (Moskva)* 44(5):125-128, 1968.

8003 **WAYS OF IMPROVING THE FUNCTION OF AN ARTIFICIAL ESOPHAGUS.** *(Rus.)* Popov V. I. (S. M. Kirov Acad. Milit. Med., Leningrad, USSR), V. I. Filin, A. I. Reshetov, V. F. Zhupan and L. I. Zykov. *Khirur (Moskva)* 43(10):115-120, 1967.

8004 **COMBINATION OF DIVERTICULA AND CAN CER OF THE ESOPHAGUS.** *(Rus.)* Kolomiichenko, M. I. and L. G. Rozenfel'd. *Khirurgiia (Mos* 43(10):136-141, 1967.

8005 **ESOPHAGEAL PERFORATION.** *(E.)* Gerhard F. P. (Clara Maass Hosp., Belleville, N. J.), A. M Sabety, R. A. Trillo and M. B. Fernando. *Arch Surg* 96(3):4 419, 1968.

8006 **THE PREPARATION AND POSTOPERATIVE CARE OF AN ESOPHAGEAL FISTULA IN TH DOG.** *(E.)* Gantt, W. H. (VA Hosp., Perry Point, Md.), E. Brown and W. P. Pare. *Condit Reflex* 3(4):263-268, 1968.

8007 **PERFORATION OF THE ESOPHAGUS: A CHALLENGE TO EARLY DIAGNOSIS.** *(E.)* Stephenson, H. E. Jr. (U. Missouri Sch. Med., Columbia), R. McLeod, J. B. McCraw, J. W. MacKenzie, W. MacDonald and M. T. English. *Amer J Surg* 115(5):648-650, 1968.

3 **SUDDEN DEATH DUE TO REGURGITATION OF A PEDUNCULATED ESOPHAGEAL LIPOMA.**
SE REPORT] *(E.)* Allen, M. S. Jr. (U. Virginia Sch. Med.,
lottesville) and W. H. Talbot. *J Thorac Cardiov Surg*
):756-758, 1967.

9 **EXPERIMENTAL STUDY OF A NEW OPERATION FOR THE TREATMENT OF REFLUX**
PHAGITIS. *(E.)* Demos, N. J. (New Jersey Coll. Med.,
y City), J. J. Timmes and J. Di Bianco. *J Thorac Cardiov*
54(6):832-838, 1967.

0 **HIATAL HERNIA: THE CRUX OF THE MATTER.** *(E.)* Ehrlich, L. (San Juan VA Hosp.,
to Rico). *Bol Asoc Med P Rico* 60(7):310-321, 1968.

1 **POISONING DUE TO CAUSTICS.** *(Fr.)* Genot, A. (St. Justine Hosp., P. Q., Canada), C. A.
ier and L. Chicoine. *Un Med Canada* 97(3):279-282, 1968.

2 **PAPILLOMATOSIS OF THE ESOPHAGUS: A CLINICAL CASE.** *(Fr.)* Dubarry, J. J.
deaux, France), C. Richir, J. P. Bernard, G. Lacoste, J.
e and J. Bretelle. *Arch Franc Mal Appar Dig* 56(7/8):713-
1967.

3 **COURSE, COMPLICATIONS AND TREATMENT OF MEGAESOPHAGUS.** *(Fr.)* Hillemand, P.
tal (Paris) 55(783-bis):683-685, 1967.

4 **SELECTION OF SURGICAL CANDIDATES FOR REPAIR OF HIATUS HERNIA.** *(E.)* Moen,
. (Seattle, Wash.). *Northwest Med* 67(1):27-30, 1968.

5 **CARCINOSARCOMA OF THE ESOPHAGUS. [CASE REPORT]** *(E.)* Stener, B. (Orthopedic
., U. Göteborg, Sweden), N. G. Kock, S. Pettersson and
etterlund. *J Thorac Cardiov Surg* 54(5):746-750, 1967.

6 **PALLIATION FOR MALIGNANT ESOPHAGOTRACHEAL FISTULA.** *(E.)* Judd, D. R. (Dept.
., St. Louis U., Mo.) and J. Codd. *J Thorac Cardiov Surg*
):751-755, 1967.

7 **SURGICAL TREATMENT OF CARCINOMA OF THE CERVICAL ESOPHAGUS. [CASE RE-**
T] *(E.)* Moore, W. T. (U. Mississippi Med. Sch., Jackson),
. Arnold and L. H. Day. *Southern Med J* 60(11):1159-
3, 1967.

8 **A COMMON CAUSE OF UPPER GASTROINTESTINAL BLEEDING - THE MALLORY-WEISS**
IDROME. *(E.)* Wells, R. F. (Brooke Gen. Hosp., San
onio, Texas). *Southern Med J* 60(11):1197-1201, 1967.

9 **HIATAL HERNIA.** *(E.)* Blattner, R. J. (Houston, Texas). *J Pediat* 72(3):424-426, 1968.

0 **THE ESOPHAGUS.** *(E.)* Oettlé, A. G. Pp. 90-98 in *The Prevention of Cancer.* Raven, R. W.
F. J. Roe, Eds. London, Butterworths, 1967.

8021 **PARAESOPHAGEAL HERNIA.** *(E.)* Hill, L. D. (Virginia Mason Hosp., Seattle, Wash.) and J. A. Tobias. *Arch Surg* 96(5):735-744, 1968.

8022 **KYPHOSCOLIOSIS--A DETERMINING FACTOR IN THE DEVELOPMENT OF HIATUS HERNIA.** *(Ser.)* Matejčić, M. (Dr. Zdravko Kučić Gen. Hosp., Rieka, Yugoslavia). *Med Glas* 21(1-2):13-17, 1967.

8023 **THE HELLER OPERATION FOR ACHALASIA OF THE ESOPHAGUS IN CHILDREN.** *(It.)* Bifani, I. (Inst. Pediat. Surg., U. Naples, Italy). *G Ital Chir* 23(1):81-101, 1967.

8024 **COMPLICATED DIAPHRAGMATIC HERNIA.** *(It.)* Cevese, P. G. (Inst. Gen. Clin. Surg., U. Padua, Italy) and R. Vecchioni. *Acta Chir Ital* 23(6):631-657, 1967.

8025 **INTERMITTENT DYSPHAGIA OF THE LOWER ESOPHAGEAL RING.** *(It.)* Zotti, E. F. (Munic. Hosp. Padua, Italy), G. Galassi and G. Fasolo. *Acta Chir Ital* 23(3):355-362, 1967.

8026 **EVENTRATIONS AND DIAPHRAGM HERNIAS.** *(Sp.)* Flórez T., F. J. *Medicina (Madrid)* 35(12): 1201-1215, 1967.

8027 **TREATMENT OF SEVERE HEMATEMESIS BY GASTRO-ESOPHAGEAL COOLING.** *(Fr.)* Levrat, M. (Lyon, France), J. Pasquier, R. Lambert, A. Tissot, B. Moulinier and C. Champagnat. *Arch Mal Appar Dig* 56(12): 1257-1264, 1967.

8028 **PRIMARY MALIGNANT MELANOMA OF THE ESOPHAGUS.** *(Ger.)* Witonsky, O. (Inst. Anat. Path., Provincial Hosp., Salzburg, Germany). *Krebsarzt* 23(2): 94-96, 1968.

8029 **HIATUS HERNIA IN ADULTS.** *(Por.)* Figueroa, E. O. (Fac. Med., Natl. U. Córdoba, Argentina), J. A. Castillo, C. Amiano, R. Bonadero, A. H. S. Niasi and O. Javurek. *Bol Sanat S Lucas* 29(8):151-155, 1968.

8030 **ESOPHAGOPLASTY WITH THE LARGE INTESTINE.** *(Sp.)* Androsoff, P. I. (Sclifosovsky Inst., Moscow, USSR). *Rev Cir Urug* 37(1/2):46-52, 1967.

8031 **CONGENITAL PARAESOPHAGEAL HERNIA OF THE ANTRUM IN INFANTS.** *(It.)* Emmola, C. (Inst. Radiol., U. Palermo, Italy) and U. Guajana. *Radiol Prat* 17(2):187-201, 1967.

8032 **THE POSSIBILITIES OF DEVELOPING POSTOPERATIVE HIATUS HERNIAS.** *(Pol.)* Dobrowolski, J. (Acad. Med., Zabrze, Poland) and M. Gliński. *Pol Przegl Chir* 39(12):1309-1312, 1967.

See also: 7600,7858,7875,7889,8079,8203,8655

STOMACH

(Gastric ulcer is placed with peptic ulcer)

8033 **CLINICAL AND EXPERIMENTAL STUDIES ON GASTRIC HEMORRHAGE DURING HYPOTHERMIA WITH SURFACE COOLING.** *(Jap.)* Nagano, K. (Fac. Med., Kagoshima U., Japan). *Acta Med (Fukuoka)* 38(5): 90-94, 1968.

The mechanism of gastric hemorrhage during surface cooling was studied by gastric analysis and histological techniques. It was noted that during hypothermia, marked hemorrhagic tendencies and pooling of blood in the abdominal organs occurred. At 30C (rectal temperature), gastric analysis revealed an increase in gastric secretion, higher levels of total acid, slight to moderate decreases in free acid, extremely high pH levels, and gross bleeding. A decrease in gastric vascular resistance was also demonstrated. The intragastric temperature was always greater than the rectal temperature. On histologic examination, the Berlin blue test was highly positive in the gastric cavity, gastric pits, and lumina of the gastric glands. Turnbull's blue test was positive in the gastric gland lumina and dilatation and hyperemia of the capillary vessels were demonstrated in the gastric pits. It is concluded that the severe gastric hemorrhage occurring during hypothermia with surface cooling is caused by rupture of capillaries in the gastric pits.

8034 **EXPERIMENTAL STUDIES ON STAPLERS FOR SUTURING THE STOMACH AND INTESTINE.** *(Ger.)* Draznin, N. (Inst. Radiol. Radiat. Ther., U. Bonn, Germany), A. Blömer and E. Bücheler. *Zbl Chir* 92(52):3069-3073, 1967.

B-shaped tantalum staples were used on 10 dogs who underwent Billroth I and II gastrectomies. Three different Russian staplers were used: one (the UKZ-8) for closing the gastric stump, another (the NZKA-60) for stapling the side-to-side gastrointestinal anastomosis and Braun's anastomosis in the Billroth II procedure, and a third (the UKL-60) for closing the duodenal stump. One of the dogs developed a ruptured stomach 8 days after surgery and another contracted distemper. No other complications occurred. Dilatation of the gastric stump and a slight delay in gastric emptying were found 1 week after Billroth I gastrectomies but subsequently disappeared. Dilatation of the gastric stump was not as pronounced after Billroth II gastrectomies. Autopsies, performed 2 days to 8 months after surgery, showed no evidence of foreign-body reactions.

8035 **RESULTS FOLLOWING REMEDIAL OPERATION FOR SEVERE DUMPING SYNDROME.** *(E.)* Gerwig, W. H. Jr. (West Virginia U. Med. Ctr., Morgantown), G. W. Easley and C. B. Mendoza Jr. *Arch Surg* 95(4):631-635, 1967.

Results are presented for 6 patients with the dumping syndrome who were treated by a remedial operation. One of these 6 had originally undergone a vagotomy and gastrojejunostomy, while the other 5 had undergone subtotal gastrectomy, all for ulcers. The gastrojejunal anastomosis was converted by transection of the jejunum at 2 sites in order to permit the creation of an isoperistaltic jejunal segment between the stomach and the duodenum, varying from 6-12 inches in length. A bilateral vagotomy was also performed in all 6 cases. Each of the patients promptly gained weight and obtained marked benefit from the remedial operations. One patient died of unrelated causes 2 yr later; the remaining 5 have been able to return to their regular occupations relieved of dumping symptoms. Neither jejunal ulceration nor intestinal obstruction have been noted in the limited follow-up.

8036 **THE PATHOGENESIS OF IRON DEFICIENCY ANEMIA FOLLOWING PARTIAL GASTRECTOMY: A STUDY OF IRON BALANCE.** *(E.)* Kimber, C. (New England Med. Ctr. Hosp., Boston, Mass.), J. F. Patterso and L. R. Weintraub. *JAMA* 202(10):935-938, 1967.

Eight patients (5 females and 3 males, 36-63 yr old) who developed iron-deficiency anemia after partial gastrectomy were tested to determine the role of blood loss and impaired iron absorption. Significant intermittent blood loss was document in 7 patients, while decreased iron absorption was found in 5 In 4 patients, both factors were additive in causing the iron d ficiency. In the single patient with decreased absorption of a test dose of iron without evidence of bleeding, the anemia responded rapidly to therapeutic doses of orally administered iron. These results stress the relative importance of blood los as a causal factor in iron deficiency after gastric surgery.

8037 **IMPROVEMENT IN THE END-RESULTS OF SURGICAL TREATMENT OF GASTRIC CANC** *(E.)* Muto, M. (Tohoku U. Sch. Med., Sendai, Japan), T. Mal S. Majima and I. Yamaguchi. *Surgery* 63(2):229-235, 1968.

From 1941 to 1960, gastric resection was performed on 157 of 2014 patients with gastric cancer admitted to the surgery department at Tohoku University. A total of 1441 patients (91.3%) survived the operative procedure, consisting of either subtotal gastrectomy (1259 cases), proximal gastrectomy (59 cases), or total gastrectomy (261 cases). No distinction betw curative and palliative resection was made. A total of 1347 cases were available for follow-up 5 yr or more postoperative 376 were alive after 5 yr (27.9% 5-yr survival rate for the traced cases, or 18.7% 5-yr survival rate for the total admissi The survival rate increased from 22.6% in the period 1941-45 to 34.9% in the period 1956-60. The survival rate for total a missions increased from 10.4% during 1941-45 to 26.5% duri 1956-60. These trends in the end-results of the surgical treatment of gastric cancer correlated well with the relative freque of the mucosal or submucosal carcinomas in the resected spe mens. It is concluded that the recent improvement in end-results is, for the most part, attributable to an increase in the incidence of early cases among surgically treated patients, and to recent advances in the early diagnosis of this disease.

8038 **DIETARY AND PURGATION FACTORS IN T EPIDEMIOLOGY OF GASTRIC CANCER.** *(E.)* Graham, S. (Dept. Prev. Med., State U. New York, Buffalo), A. M. Lilienfeld and J. E. Tidings. *Cancer* 20(7):2224-2234, 1967.

A questionnaire study was made of 188 men and 88 women with gastric cancer plus 800 men and 1400 women with non-neoplastic, nondigestive disease seen at Roswell Park Memori Institute between 1957 and 1965. Sex- and age-specific comparisons of cases and controls showed few positive results for men and almost none for women with regard to the ingestion

large number of specific foods and beverages, including
hol. Higher proportions of men with gastric cancer ate
bage and potatoes than controls, though the latter result
not significant. Eating infrequently was also more char-
ristic of patients with cancer; this result may parallel those
egi and Wynder regarding 'irregular' eating. The current
ing of a higher risk for use of purgatives was similar to the
reported earlier by Boyd and Doll. It is acknowledged
many sources of possible error in these results may be in-
nt in the interview method used.

9 **STROMAL REACTION AROUND TUMOR AND METASTASIS AND PROGNOSIS AFTER CURA-**
E GASTRECTOMY FOR CARCINOMA OF THE STOM-
I. *(E.)* Inokuchi, K. (Fac. Med., Kyushu U., Fukuoka,
n), S. Inutsuka, M. Furusawa, K. Soejima and T. Ikeda.
cer 20(7):1924-1929, 1967.

prognosis after curative gastrectomy for carcinoma of the
nach was studied by histologic examination of the resected
imen in 270 cases in which the over-all 5-yr survival was
%. In cases without metastases, when the main tumor
wed both fibrous and cellular stromal reactions, the 5-yr
ival rate was 100%; when the reaction was negative, how-
, the prognosis was only 30%. In cases with metastases
ch showed no stromal reaction, the prognosis was as low
%, regardless of the reaction in the main tumor. It is
cluded that the stromal reaction around the carcinoma de-
nines the prognosis and that the prognosis can be estimated
increased accuracy after evaluation of the reaction in the
astases.

0 **CLINICAL AND BIOLOGICAL STUDY OF TWO FORMS OF ASPIRIN: DETERMINATION OF**
STRIC TOLERANCE. *(Fr.)* Vignalou, J. (d'Ivry Hosp.,
nce) and H. Beck. *Therapie* 22(5):967-975, 1967.

omparison was made of the therapeutic effectiveness and
ric tolerance of ordinary aspirin and tablets of enteric-coated
rograins. Trials were run on 92 elderly patients with chronic
ases; 59 of these patients were over 75 yr old. No gastro-
stinal disturbances and no evidence of melena were seen
n patients took 2 tablets q.i.d. Benzidine and guaiac tests
occult blood were positive in 16 of 23 patients taking or-
ary aspirin and in 18 of these same patients after taking
eric-coated aspirin. Two patients who developed gastroin-
inal disturbances after taking ordinary aspirin on an empty
nach had no difficulties with enteric-coated aspirin.

41 **SUBCLINICAL VITAMIN-D DEFICIENCY FOLLOWING GASTRIC SURGERY: HISTOLOGICAL**
DENCE IN BONE. *(E.)* Bordier, P. (Lariboisiere Hosp.,
s, France), H. Matrajt, D. Hioco, G. W. Hepner, G. R.
mpson and C. C. Booth. *Lancet* 1(7540):437-440, 1968.

e biopsies were taken before and after an i.v. dose of vita-
D_3 (1 mg or 4000 IU in 11 patients who had undergone
ric surgery. The undecalcified bone was then stained with
idine-blue and solochrome-cyanine and quantitative measure-
ts were made with Zeiss integrating eyepieces. The osteoid
me was found to be normal in all cases. However, in 3 pa-
ts with elevated serum alkaline phosphatase levels there was
icrease in the osteoid surface area and a decrease in the pro-
ion of osteoid with a calcification front. In addition, the administration of vitamin D resulted, within 8 days, in a marked increase in the proportion of osteoid with a calcification front, without causing any change in the osteoid surface area. Similar but lesser changes occurred in some of the patients with a normal serum alkaline phosphatase. The data suggest that vitamin D is necessary for the proper formation of the calcification front, and that a decrease in the proportion of osteoid with a calcification front is an early indication of vitamin D deficiency.

8042 **POSTOPERATIVE BLEEDING IN GASTRIC SURGERY.** *(Ger.)* Kolig, G. (Surg. Clin., U. Heidelberg, Germany) and A. Encke. *Langenbeck Arch Klin Chir* 318(4):281-302, 1967.

There were 25 cases of postoperative bleeding among 1519 patients subjected to gastric surgery. Of these 25 patients (22 men and 3 women, aged 27-85 yr), 19 had undergone their first operation for peptic ulcers and 6 had stomach cancer. Preoperative complications included 14 patients with bleeding, 5 with perforated ulcers, 9 with penetrating ulcers and pancreatitis, and 3 with pyloric stenosis. The causes of postoperative bleeding were diagnosed in 18 cases and included overlooked ulcers (6 cases); sutured ulcers (3 cases), inoperable tumors (3 cases), erosion (2 cases), and 1 case each of blood-clotting disorder, enterocolitis, liver injury, and aneurysm of the pancreaticoduodenal artery. Conservative treatment was used in 20 cases and surgical treatment in 5. Deaths occurred in 3 of the operated patients and in 9 conservatively treated patients for a total mortality of 48%. Two of the patients who died were women; 11 were over 50 yr old; and 10 of the 12 who died had peptic ulcers.

8043 **DIGESTIVE AND ABSORPTIVE FUNCTION IN THE GASTROINTESTINAL TRACT OF GASTRECTOMIZED RATS.** *(Ger.)* Maurer, W. (Waldkrankenhaus St. Marien, Erlangen, Germany). *Z Gastroent* 5(5):282-286, 1967.

By determining blood sugar levels before and after oral loading with 2 ml of 25% glucose or 0.15 g of starch, glucose absorption and starch digestion were studied in rats subjected to Billroth I and II gastrectomies and modified gastrointestinal anastomoses with reversal of the duodenum. After oral glucose loading rats with modified anastomoses had significantly smaller increases in blood sugar levels than rats with Billroth I and II gastrectomies, but not significantly different from control values. There were no significant differences in starch digestion between gastrectomized and control animals, but the mean time required for blood sugar levels to reach a maximum was significantly longer for operated rats after starch loading. Radiological examinations with $BaSO_4$ showed that stomach emptying was rapid in 12 of 14 animals with Billroth II gastrectomies, in 11 of 16 of those with Billroth I gastrectomies, and in 11 of 27 of those with modified anastomoses. Adaptation to gastrectomy was seen in 13 of 16 with Billroth I gastrectomies, in 14 of 14 with Billroth II gastrectomies, and in 12 of 27 with modified anastomoses. Of the latter group, 13 had signs of small intestine dysfunction, 4 had reflux into the stomach, and 10 had extreme dilatation. These findings suggest that increased glucose absorption in rats with Billroth I and II gastrectomies is due to the presence of an intact fore-stomach and aberrant function of the anastomosis and not to increased absorption from the jejunum.

8044 **CHANGES IN CHLORIDE AND PROTEINS IN THE GASTRIC MUCOSA. II. CARCINOMA OF THE STOMACH.** *(It.)* Breglia, A. (Santa Maria di Loreto Hosp., Naples, Italy) and F. De Luca. *Riv Gastroent* 19(6, Suppl.):875-882, 1967.

Protein and chloride determinations were run on gastric mucosa removed from 6 patients with stomach cancer. All patients had hypochlorhydria. Samples were taken from the margin of the tumor, 2 and 4 cm away from it, and from the greater and lesser curvatures and gastric fundus. Protein and chloride contents were identical at the edge of the tumor and at sites near it, but increased slightly at greater distances from the tumor. This increase was more pronounced for proteins than for chlorides. Chloride and protein contents were higher in mucosa from the greater curvature and gastric fundus than in that from the lesser curvature. Differences between the chloride content in the lesser and greater curvatures were slight while differences between the protein contents were greater. While chloride contents were much higher in the fundus than in the greater and lesser curvatures, the protein contents of the fundus were definitely higher than those of the lesser curvature and slightly lower than those of the greater curvature. Protein and chloride contents of the mucosa from the edge of the tumor and from sites near it were considerably lower than those found in a previous study made in ulcer patients. Hypochlorhydria which is normally found in patients with stomach cancer is probably due to a decrease in chloride and protein levels in the gastric mucosa.

8045 **CHANGES IN SERUM AND URINARY CHOLECYSTOKININ AND ANTICHOLECYSTOKININ AFTER GASTRIC RESECTIONS.** *(It.)* Fontana, G. (Inst. Spec. Med. Path., U. Bologna, Italy), P. Azzaroli, P. Vezzadini and G. A. Lanfranchi. *G Clin Med* 48(12):1413-1431, 1967.

Serum and urinary cholecystokinin and anticholecystokinin activities were determined on 20 normal subjects, on 14 patients (10 men and 4 women, aged 32-71 yr) after Billroth II gastrectomies for peptic ulcers, and on 1 patient who had undergone pancreatectomy, duodenectomy, cholecystoenterostomy, gastrojejunostomy, and end-to-end pancreaticojejunostomy for a carcinoma of the head of the pancreas. Determinations were performed under basal conditions and after administration of corn oil or sorbitol, either through a catheter into the duodenum in the controls or orally in gastrectomized patients. Under basal conditions there were no appreciable differences between controls and gastrectomized patients in urinary or serum cholecystokinin or anticholecystokinin activities. After stimulation serum cholecystokinin activities took longer to reach a maximum in gastrectomized patients than in controls, probably because of the use of different routes of administration. In 2 gastrectomized patients low values, particularly of urinary cholecystokinin, were associated with gallbladder hypotonia. In the duodenectomized patient urinary cholecystokinin was present, but its activity was very low, suggesting that this hormone is also produced outside of the duodenum.

8046 **A CASE OF EOSINOPHILIC GRANULOMA OF THE STOMACH.** *(Jap.).* Ohsumi, K. (Shinkori Hosp., Osaka, Japan), M. Kurosawa and A. Ikeuchi. *Med J Mutual Aid Ass* 17(1):77-80, 1968.

8047 **GASTRIC CANCER WITH DISSEMINATED CUTANEOUS METASTASIS: REPORT OF 2 CASES AND REVIEW OF THE LITERATURE.** *(Jap.)* Os
K. (Sasebo Kyosai Hosp., Japan), K. Kaetsu, Y. Ikeda, S. Nakajima and M. Hirakida. *Med J Mutual Aid Ass* 16(3):81
87, 1967.

8048 **GASTRIC X-RAY FINDINGS IN THE LONG-TERM FOLLOW-UP OF PYLOROMYOTOMY IN CHILDREN.** *(Ger.)* Herrmann, K. (Radiol. Clin. Polycli
Friedrich Schiller U., Jena, Germany) and H. Schickedanz.
Kinderchir 6(1):34-38, 1968.

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8111 **THE EFFECT OF PERISTALSIS ON THE DIRECTION OF METASTASIS OF STOMACH CANCER.** *(Rus.)* Galandina, T. I. (N. N. Burdenko Main Milit. Hosp., Moscow, USSR). *Eksp Khir Anest* 12(6):30-34, 1967.

See also: 7677,7689,7839,7845,7846,7849,7850,7855,7856,7857,7869,7872,7875,7885,7888,7896,7900,7912,7916, 7929,7935,7995,7997,7998,8001,8027,8166,8176,8182,8184,8187,8213,8276,8309,8389,8694,8712

8112 **SOME EFFECTS OF EXTENSIVE INTESTINAL RESECTION IN CHILDHOOD.** *(E.)* Wilkinson, A. W. (Inst. Child Health, U. London, England). *Bibl Paediat* (87):191-208, 1968.

Clinical evaluation of 11 infants (3 hr-6 months of age) who underwent extensive resection of the small and large bowel showed that postoperative survival is determined chiefly by the length of intestine remaining after surgery. Infants with 60 cm or more of small intestine should survive surgery, while those with less than 60 cm but more than 30 cm and those with less than 30 cm have an "even" or "poor" chance, resp. Of the 11 original patients 5 died, succumbing 26 days to 30 months after surgery. The diagnoses leading to resection were gastroschisis; small bowel volvulus; small bowel, cecal, and colonic gangrene; jejunal and ileal atresia; and gangrenous intussusception. The ileocecal valve was resected in 5 patients, 2 of whom died, and was preserved in 6, 3 of whom died. One patient with only 25 cm of small intestine remaining has survived 4 yr. It is suggested that milk feeds be resumed as soon as possible after surgery. Due to the rapid transit time, shortened exposure of bowel to nutrient and decreased absorption, full-strength feeds should be used from the beginning to insure proper nutrition and growth. Observations on children have led to the conclusion that there is no compensatory hypertrophy or dilatation in the absence of obstruction following extensive small bowel resection and that predigested milk feeds and those fortified with amino acids, carbohydrates, and medium-chain triglycerides instead of natural milk fat may overcome digestive and absorptive deficiencies. Incomplete obstruction due to adhesions may interfere with food consumption; this nutritional impairment affects fat and muscle formation more than bone growth and length of body. Survival in these children depends on careful attention to diet and immediate hospitalization when necessary.

8113 **CHRONIC ULCERATIVE (NONGRANULOMATOUS) JEJUNITIS.** *(E.)* Jeffries, G. H. (New York Hosp., N. Y.), H. Steinberg and M. H. Sleisenger. *Amer J Med* 44(1):47-59, 1968.

Case reports are presented for 4 patients (1 man and 3 women, 43-69 yr old) suffering from chronic ulcerative (nongranulomatous) jejunitis. Severe diarrhea with abdominal pain and fever, steatorrhea and hypoproteinemia due to enteric loss of plasma protein were the predominant clinical features. Peroral biopsies of the jejunal mucosa revealed villous atrophy in 3 patients but the characteristic pathologic abnormality in surgical biopsy specimens of the jejunum and in the small intestine examined at postmortem was the presence of multiple mucosal ulcers with adjacent areas of both atrophic and normal mucosa. Treatment with a gluten-free diet was ineffective, whereas corticosteroid therapy, although unpredictable in its effect, appeared to be of benefit to several patients. The differential diagnosis of this condition is discussed.

8114 **EFFECTS OF DECOMPRESSION DURING REMOVAL OF INTESTINAL OBSTRUCTION.** *(E.)* Singleton, A. O. Jr. (U. Texas Med. Branch, Galveston) and P. Montalbo. *Ann Surg* 167(6):909-911, 1968.

Mechanical obstruction of the small bowel 20 cm proximal to the ileocecal valve was created in 30 pairs of dogs and then removed 5 days later. In 1 of each pair, the bowel was decompressed by enterotomy and removing the gas and liquid content; the other dog in each pair was not decompressed. At sacrifice 24 hr later, the nondecompressed bowel was 1.5-3 times as distended and 2-3 times as heavy as the decompress bowel. No shock or other ill-effects resulted from the decompression procedure. The decrease in serum potassium, sodium and chloride and the slight increase in serum carbon dioxide, blood urea nitrogen, and hematocrit, which had been present in the animals at the time of the release of the obstruction, were restored to normal in the decompressed animals 24 hr later; in the nondecompressed animals, however, abnormal val persisted. It is concluded that decompression of the distende bowel is followed by a more rapid return to a more physiolog cal state. Trauma to the bowel wall can and should be kept to a minimum.

8115 **INTUSSUSCEPTION AS A POSTOPERATIVE COMPLICATION.** *(E.)* McGovern, J. B. (Harva Med. Sch., Boston, Mass.) and R. E. Gross. *Surgery* 63(3):50 513, 1968.

Data are presented for 15 patients (10 boys and 5 girls, 5 months-6 yr old) who developed intestinal obstruction as a re sult of intussusception occurring during the 1st 30 days after intestinal surgery. The average age was 17 months and the median age was 22 months. The length of time between the primary surgical procedure and the subsequent reduction of the intussusception was 5-26 days (average 11 days). Patients were symptomatic for 2-21 days (average 6 days) before unde going reoperation. Abdominal distension and vomiting was present in 13 (87%) and abdominal pain was present in 11 (73%). Only 2 infants passed blood per rectum, and none of the patients had a palpable abdominal mass. Seven patients (47%) had temperatures over 101F and leukocytosis greater than 10,000/mm^3. The flat and upright abdominal X-ray examinations were considered to be most helpful in arriving at a diagnosis of small bowel obstruction. Only 3 had preoperative diagnoses of intussusception. All patients except 1 we treated for a period of time with intestinal suction, and all pa tients subsequently came to operation. In 14, the intussuscep tion was reduced manually, while in 1, a gangrenous bowel prompted a Mikulicz exteriorization resection. The intussusceptions were located in the ileum (9), jejunum (5), and ascending colon (1). One patient died of pneumonia and peritonitis following operation for a gangrenous perforated intussu ception, 2 died of unrelated causes 3 and 21 months later, an 12 made uneventful recoveries (follow-up of 6 months-14 yr). It is concluded that postoperative intussusception occurs with sufficient frequency as a complication of pediatric surgery so that consideration of this entity is warranted when obstructive bowel symptoms appear.

8116 **SMALL-INTESTINAL STRUCTURE AND FUNCTION AND HAEMATOLOGICAL CHANGES IN DERMATITIS HERPETIFORMIS.** *(E.)* Fry, L. (London Hosp., England), P. Keir, R. M. H. McMinn, J. D. Cowan and A. V. Hoffbrand. *Lancet* 2(7519):729-734, 1967.

ur out of 12 patients with dermatitis herpetiformis and 1 th subcorneal pustular dermatosis had both macroscopic d microscopic abnormalities of the jejunal mucosa resembling ose seen in untreated idiopathic steatorrhea; a further 5 owed microscopic changes only. Fecal fat excretion was vated in 7 of the patients, but the absorption of xylose, ic acid, and vitamin B_{12} were almost invariably normal. In of the patients there was hematological and/or biochemical dence of folate deficiency, and in 5, evidence of iron de- iency. The patients with the severest changes in the jejunal icosa also had the severest steatorrhea and folate deficiency. ere were also signs of IgM-globulin deficiency, raised levels agglutinating-factor to *Lactobacillus casei* in the serum, and enic atrophy; these abnormalities are also present in untreated opathic steatorrhea. The significance of these structural ab- rmalities of the small intestinal mucosa in dermatitis herpeti- rmis is unknown.

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STUDY OF HEALING OF INVERTING AND EVERTING INTESTINAL ANASTOMOSES.

) Canalis, F. (Dept. Surg., U. Chicago, Ill.) and M. M. Ravitch. *rg Gynec Obstet* 126(1):109-114, 1968.

o types of everting and 3 types of inverting anastomosis re performed in dogs; in each instance the ileum was trans- ted and resutured. In group 1, open everting anastomosis was rformed with a 1-layer closure using interrupted through-and- rough silk mattress sutures. In group 2, open everting anasto- osis was performed with a stapling machine that inserted a gle row of stainless steel staples. In group 3, open inverting astomosis was performed with a 1-layer closure using inter- pted Halsted silk sutures. In group 4, open inverting anasto- osis was performed with a 2-layer closure; continuous catgut tures for the inner layer and interrupted silk sutures for the ter layer. In group 5, closed inverting anastomosis was per- rmed with a 1-layer closure using interrupted mattress Halsted k sutures. In each of these groups of 24 dogs, half received eoperative antibiotics; also, in each group, half had the anas- mosis wrapped with a layer of silastic gauze. Animals were crificed 15-16 days later. In group 1, all 12 dogs with anas- moses which were not wrapped survived. Of the 12 dogs in iom the anastomoses were wrapped, there was only 1 surviror; e 11 others all had severe inflammatory reactions with large scesses around the anastomoses and 7 had general peritonitis death. In group 2, 9 of the 12 dogs with no wrapping showed od healing; in the 12 with wrapping, all were dead from neralized peritonitis in 6-11 days. In group 3, those without apping showed healing without complications, whereas of the with wrapping, 10 died in 7-11 days from abscess or peri- nitis or both. In group 4, those without wrapping healed thout complications, while 10 of 12 with wrapping died. In oup 5, 11 of the 12 without wrapping survived and 8 of 12 th wrapping died. In all groups, there was good correlation tween antibiotic preparation and prevention of adhesion for- ation.

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LOW MOLECULAR WEIGHT DEXTRAN (RHEOMACRODEX) AND VASODILATORS THE PREVENTION OF POSTOPERATIVE ILEUS. *(E.)*

hobinger, R. A. (St. Anna Clin., Lucerne, Switzerland). *ll Soc Int Chir* 27(3):153-161, 1968.

lministration of low-molecular-weight dextran (LMWD; 500 i.v. starting before the onset of anesthesia and given over a 6-hr period; another 1 or 2 500-ml units administered as necessary until flatus was passed) and vasodilators (1 ampule Ronicol compositum every 8 hr; usually continued for 24 hr after the 1st passage of flatus) to 43 patients subjected to surgery for hiatus hernia (6 cases), lumbar sympathectomy (10 cases), cholecystectomy (9 cases), gastrointestinal resections with anastomoses (3), and various intraabdominal operations (15 cases) was effective in preventing postoperative paralytic ileus. Compared with a group of 31 patients operated on for similar indications, but not given the combined therapy, treated patients showed earlier resumption of intestinal peristalsis (12-24 hr vs. 3-4 days postoperatively) and spontaneous passage of flatus (on 2nd day vs. 3-6 days postoperatively). Postoperative abdominal distension was minimal, facilitating breathing and coughing and thus reducing the incidence of postoperative pulmonary complications. This combined therapy was also associated with earlier removal of nasogastric tubes, lower blood transfusion requirements, stabilization of blood pressure, improved postoperative conditions and significantly reduced postoperative urinary retention. The beneficial effects of LMWD and vasodilators can be jeopardized or completely blocked by an intervening complication (pancreatitis, hematoma). Special precautions should also be taken in older patients or in those with heart disease, since abnormal elevation of blood pressure can result from LMWD. Increased bleeding tendency of transected tissues occurs because LMWD improves the microcirculation. LMWD combined with vasodilators, and possibly with anticoagulants, seems to be able to restore normal viability in preinfarcted intestine.

8119

PROTEIN STARVATION AND THE SMALL INTESTINE. I. THE GROWTH AND MORPHOLOGY OF THE SMALL INTESTINE IN WEANLING RATS.

(E.) Hill, R. G. Jr. (U. Colorado Med. Ctr., Denver), J. Prosper, J. S. Hirschfield and F. Kern Jr. *Exp Molec Path* 8(1):66-74, 1968.

The effect of 5-45 days of protein deprivation on the growth and development of the small intestine was investigated in 22-day-old weanling male albino rats. In animals given a synthetic protein-free diet for 45 days the overall length (61.5 cm) and weight (2.05 g) of the small intestine did not increase significantly in comparison with starting values (58.5 cm and 1.65 g, resp.), while animals fed a protein-containing diet showed marked increases in both parameters (to 89.0 cm and 6.37 g, resp.). The protein-deficient rats also lost an average of 80% of their starting weight after 45 days of protein deprivation, while the control rats steadily gained weight. There were minimal increments in the muscle thickness of jejunal and ileal segments during 45 days of protein starvation, while muscle thickness increased from 0.6 to 1.0 ± 0.12 cm and from 0.6 to 1.3 ± 0.45 cm, resp., in the corresponding segments of control animals. Crypt size and morphology were not appreciably affected by protein depletion, and villus length was only minimally affected in the jejunum (2.2 cm growth in 45 days compared to 2.4 cm in controls. The villi tended to be thinner, especially at their bases, but not shorter, than in fed controls. In contrast, there was a total lack of longitudinal growth of villi in the ileum (0.2 cm in protein-deficient rats compared to 1.4 cm in controls). Light- and electron-microscopic observations of the epithelium revealed only a slight excess of membrane-bound structures containing portions of cytoplasm in various states of disintegration (predominantly in the ileum).

Mucous globules appeared smaller and more opaque in protein-deprived animals than in controls.

8120 **DEFECTS IN THE MESENTERY AND MESOCOLON IN CHILDREN.** *(Ger.)* Steinert, V. (Clin. Pediat. Surg., Karl Marx U., Leipzig, Germany). *Bruns Beitr Klin Chir* 215(3):368-375, 1967.

Clinical findings are presented for 12 infants (11 boys and 1 girl, aged 12 hr to 2 2/3 yr), 10 of whom had defects in the mesentery of the terminal ileum, 1 in the mesosigma, and 1 in the transverse mesocolon. Volvulus was present in 5 cases and a wide variety of associated congenital anomalies in 8. Only 4 of these patients survived surgical treatment. The causes of death were peritonitis in 3 cases, ileus in 2 cases, pyopneumothorax and suppurative pneumonia, toxic jaundice and thrombocytopenic bleeding, and respiratory insufficiency due to severe aspiration pneumonia in 1 case each.

8121 **CLINICAL AND RADIOLOGICAL CONTRIBUTIONS TO THE PATHOLOGICAL PICTURE OF DUODENAL DIVERTICULOSIS.** *(Fr.)* Bercu, G. (Radiol. Svc., Tirgoviste Hosp., Romania) and T. Rodica. *J Belg Radiol* 50(5):370-372, 1967.

8122 **A CASE OF EXTRAMEDULLARY PLASMOCYTOMA WITH PRIMARY SITE IN THE ILEUM.** *(Jap.)* Sugawara, I. (Otemae Hosp., Osaka, Japan), K. Nakajima, T. Izukura, T. Shimoji and Y. Morimura. *Med J Mutual Aid Ass* 17(1):81-84, 1968.

8123 **REPORT OF A CASE OF PEUTZ-JEGHERS SYNDROME.** *(It.)* Battistini, C. (Inst. Clin. Gen. Surg., U. Parma, Italy) and L. Chiampo. *Aten Parm [Biomed]* 38(6):605-617, 1967.

8124 **THE PATHOLOGY OF MECKEL'S DIVERTICULUM: REPORT OF 15 CASES.** *(It.)* Moncalvi, S. ("Ca' Granda" Maggiore Hosp., Milan, Italy), M. Porati and G. Candela. *Chir Ital* 19(Suppl. 1):182-194, 1967.

8125 **CONTRIBUTION TO THE STUDY OF PRIMARY MALIGNANT NEOPLASMS OF THE DUODENUM: REPORT OF 14 CASES.** *(It.)* Molteni, F. (Inst. Gen. Surg. Clin., U. Pavia, Italy), L. A. De Santis, C. Scotti-Foglieni and G. Pedroni. *Chir Ital* 19(Suppl. 1):45-93, 1967.

8126 **DUODENO-JEJUNAL INJURIES FOLLOWING BLUNT TRAUMA TO THE ABDOMEN.** *(E.)* Keplinger, J. B. (Henry Ford Hosp., Detroit, Mich.), A. S. Tootla, T. A. Fox and M. A. Block. *Henry Ford Hosp Med Bull* 16(2):111-118, 1968.

8127 **RADICAL ENTERECTOMY FOR HEREDITARY MEGADUODENUM.** *(E.)* Newton, W. T. (Washington U. Sch. Med., St. Louis, Mo.) *Arch Surg* 96(4): 549-553, 1968.

8128 **PERFORATION OF SMALL BOWEL BY FOREIGN BODY DURING PREGNANCY. [CASE REPORT]** *(E.)* Adducci, J. E. (Williston, N. D.) *J Abdom Surg* 10(7):187-188, 1968.

8129 **CHRONIC NONSPECIFIC ENTEROPATHY.** *(Rum.)* Fodor, O. (Inst. Med. Pharmacol, Cluj, Romania) and D. Dumitrascu. *Med Intern (Bucur)* 19(9): 1027-1036, 1967.

8130 **SELECTIVE ARTERIOGRAPHY IN A CASE O RECURRENT MELENA WITH DUODENAL POLYADENOMA.** *(Fr.)* Geffroy, Y. (U. Hosp. Ctr., Rouen, France), J. Fablet, C. Hernandez, P. Jouanneau and R. Laumonier. *Arch Franc Mal Appar Dig* 56(10):965-969, 1967

8131 **HEMORRHAGES OF THE SMALL INTESTINE.** *(Fr.)* Hivet, M. *Bull Soc Chir Paris* 58(1):13-18, 1968.

8132 **THE MANAGEMENT OF SMALL BOWEL FIST LAS: AN IMPROVED TECHNIQUE.** *(E.)* Belc W. W. (Peterborough, Ont., Canada) and R. K. Magee. *Canad Surg* 11(1):101-103, 1968.

8133 **THE EFFECT OF ILEAL BYPASS ON DIETAR INDUCED ATHEROSCLEROSIS IN THE RABBIT.** *(E.)* Okuboye, J. A. (Dept. Surg., U. Manitoba, Winnipe Canada), C. C. Ferguson and J. P. Wyatt. *Canad J Surg* 11(1): 69-77, 1968.

8134 **ULTRASTRUCTURAL STUDIES OF ABSORPTION OF METHOXYCHLOR IN THE JEJUNAL MUCOSA OF THE RAT.** *(E.)* Imai, H. (Albany Med. Coll., Union U., Albany, N. Y.) and F. Coulston. *Exp Molec Path* 8(2):135-158, 1968.

8135 **PROGRESSIVE ULTRASTRUCTURAL CHANG IN THE MUCOSAL EPITHELIUM OF THE SMALL INTESTINE OF BEAGLE DOGS FED METHOXYCHLOR.** *(E.)* Tegeris, A. S. (SPAL, Div. Pharmacol., FDA, Washington, D. C.), G. Vander Weide and J. M. Curtis. *Exp Molec Path* 8(2):243-257, 1968.

8136 **INDUCTION OF HEMORRHAGIC LESION OF INTESTINAL MUCOSA BY HYPERCALCEMIA IN ADRENALECTOMIZED RATS.** *(E.)* Fukuda, T. (Chiba U. Sch. Med., Japan) and N. Akagawa. *J Physiol Soc Japan* 30(5):355-356, 1968.

8137 **CARCINOID OF THE DUODENUM. [CASE REPORT]** *(E.)* Shah, N. C. (Sassoon Gen. Hosp India) and V. R. Bhave. *J Indian Med Ass* 51(9):463-464, 196

8138 **INTERNAL DIVERTICULUM OF THE DUODENUM (INTRALUMINAL DIVERTICULUM OF THE DUODENUM. [CASE REPORT]** *(Fr.)* Debray, C., J. Leymarios, R. Jouve and A. Grall. *Arch Franc Mal Appar Dig* 57(6/7):593-611, 1968.

8139 **TRANSFORMATION OF THE GRANULES OF INTESTINAL EOSINOPHIL POLYMORPHONUCLEAR LEUCOCYTES BY HELMINTHIC INFESTATION.** *(E.)* Casley-Smith, J. R. (Dept. Electr. Microsc., U. Adelaide, South Australia). *J Path Bact* 95(1):299-301, 1968.

0 ONE CASE OF THE PEUTZ-JEGHERS SYNDROME. *(E.)* Setka, J. (Charles U. Hosp., gue, Czechoslovakia). *Amer J Proct* 18(5):388-391, 1967.

41 POST-VAGOTOMY JEJUNAL INTERPOSITIONS. *(E.)* Smith, R. S. (Boise, Idaho). *Northwest Med* 4):364-368, 1968.

42 INTESTINAL VILLI IN THE DOG AND THE EFFECT OF *ANKYLOSTOMA CANINUM* IN-STATION. *(E.)* Verma, S. (Postgrad. Inst. Med. Ed. Res., andigarh, India), R. N. Chakravarti and P. N. Chhuttani. *ath Bact* 95(2):568-571, 1968.

43 FISTULA OF THE SMALL INTESTINE. *(E.)* Large, A. M. (Wayne State U. Coll. Med., Detroit, ch.). *Amer J Surg* 115(5):740-741, 1968.

44 HYPERPLASTIC LYMPHOID POLYPS OF THE TERMINAL ILEUM. [CASE REPORT] *(E.)* thony, J. E. Jr. (DeKalb Hosp., Decatur, Ga.). *Southern Med* 1(6):571-572, 1968.

45 URINARY AND FAECAL HISTAMINE IN RATS WITH A BLIND JEJUNAL POUCH. *(E.)* astad, O. (Veterinary Coll. Norway, Oslo) and K. Nygaard. *d Pharmacol Exp* 17(3):273-280, 1967.

46 GANGLIONEUROMA OF THE DUODENUM: RELATIONSHIP TO NONCHROMAFFIN PARA-NGLIOMA OF THE DUODENUM. [CASE REPORT] *(E.)* ldman, R. L. (Cedars-Sinai Med. Ctr., Los Angeles, Calif.). *ner J Surg* 115(5):716-719, 1968.

47 THE SMALL INTESTINE. *(E.)* Raven, R. W. Pp. 131-136 in *Tne Prevention of Cancer.* Raven, W. and F. J. Roe, Eds. London, Butterworths, 1967.

48 LEIOMYOMA OF THE DUODENUM. A CASE REPORT. *(E.)* Hansen, D. A. (Cape Town, ion S. Africa). *S Afr Med J* 41(44):1151-1152, 1967.

49 LEIOMYOSARCOMA OF THE SMALL INTESTINE IN YOUNG CHILDREN. *(Ger.)* Welcker, (Garl Gustav Carus Med. Acad., Dresden, Germany) and G. llmann. *Beitr Klin Chir* 216(2):177-183, 1968.

50 PRIMARY HODGKIN'S DISEASE OF THE SMALL INTESTINE. (ONE CASE WITH MUL-PLE PERFORATIONS). *(Gr.)* Papadimitriou, Th. *Galenos* (7-8):365-377, 1968.

51 LEIOMYOMA OF THE DUODENUM WITH SECONDARY DIVERTICULOSIS. *(It.)* Taccari, (Santa Lucia Civil Hosp., Recanati, Italy). *Ann Radiol gn (Bologna)* 40(5-6):510-516, 1967.

52 BILIARY ILEUS. *(It.)* Grapulin, G. (Inst. Gen. Surg. Clin., U. Padua, Italy) and O. Fontanin. *ta Chir Ital* 23(2):87-119, 1967.

8153 MECKEL'S DIVERTICULUM: RADIOLOGICAL DIAGNOSIS AND PRESENTATION OF A CASE. *(It.)* Caresano, A. (Inst. Radiol., Varese, Italy) and A. Borghi. *Acta Chir Ital* 24(1):89-99, 1968.

8154 SELECTIVE ARTERIOGRAPHY IN A CASE OF MELENA WITH RECURRENT DUODENAL POLYADENOMA. *(Fr.)* Geffroy, Y. (Hosp. Cent., U. Rouen, France), J. Fablet, C. Hernandez, P. Jouanneau and R. Laumonier. *Arch Franc Mal Appar Dig* 56(10):965-969, 1967.

8155 A CONTRIBUTION TO THE STUDY OF MALIGNANT MESENCHYMAL TUMORS OF THE FIRST JEJUNAL LOOP. *(It.)* Boscaino, N. (Civil Hosp., Maddaloni, Italy), P. Pennino and I. Piscicelli. *Arch Ital Pat Clin Tumori* 10(3-4):235-258, 1967.

8156 BILIARY ILEUS SURGERY. *(Rum.)* Raileanu, R. (Inst. Med. Pharm., Iasi, Romania), V. Vasile, C. Teodorescu and R. Balan. *Rev Medicochir Iasi* 71(4):1037-1040, 1967.

8157 INTESTINAL PERFORATION OF TUBERCULAR ETIOLOGY. [CASE REPORT] *(Rum.)* Ciurdea, A. (Moroieni Sanitor., Ploiesti, Romania), G. Dinca and D. Marian. *Rev Medicochir Iasi* 71(4):1029-1032, 1967.

8158 INTESTINAL ULCERS AND FEVER IN ENDOGENOUS SPRUE. *(Ger.)* Wagner, A. (Med. Clin., U. Heidelberg, Germany) and J. Meiser. *Schweiz Med Wschr* 98(24):892-898, 1968.

8159 PHYSIOPATHOLOGICAL PROBLEMS DUE TO EXTENSIVE RESECTIONS OF THE SMALL INTESTINE. *(Fr.)* Flamand, J. P. *Acta Chir Belg* 66(8):787-805, 1968.

8160 PNEUMATOSIS CYSTOIDES INTESTINALIS. *(Ger.)* Dohrmann, R. (Behring Munic. Hosp., Berlin, Germany). *Bull Soc Int Chir* 27(5):355-361, 1968.

8161 BLEEDING OF THE SMALL INTESTINE AND OF MECKEL'S DIVERTICULUM. *(Sp.)* Vilar B., J. (Fac. Med., U. Barcelona, Spain). *Rev Esp Enferm Apar Dig* 27(2):314-338, 1968.

8162 STENOSIS OF THE DUODENUM BY THE SUPERIOR MESENTERIC ARTERY. *(Pol.)* Jacyna-Onyszkiewicz, T. (Acad. Med., Lublin, Poland) and A. Sawa. *Pol Przegl Chir* 40(2):142-145, 1968.

8163 INTESTINAL NECROSIS IN THE COURSE OF PERIARTERITIS NODOSA. *(Pol.)* Napierala, M. (Acad. Med., Poznań, Poland), A. Kompf and K. Wojciechowski. *Pol Przegl Chir* 40(2):154-156, 1968.

8164 RADIOLOGY OF MECONIUM ILEUS. *(Fr.)* Du Bois, R. (Reg. Hosp. Clin., Lille, France), P. Debeugny, J. P. Maillard and S. Moyon. *Pediatrie* 23(3):283-294, 1968.

See also: 7609,7812,7814,7819,7871,7881,7966,8061,8063,8074,8077,8101,8195,8255,8263,8420,8678

8165 **ANTRAL FUNCTION AND RECURRENT ULCERATION.** *(E.)* Giles, G. R. (Dept. Surg., U. Leeds, England), M. C. Mason and C. G. Clark. *Amer J Surg* 115(4): 472-476, 1968.

Stomach acid secretion following i.v. histamine infusion (0.15 mg/kg) and antral stimulation (meat extract technique of Giles and Clark, *Scand. J. Gastroent.* 1:159, 1966) was studied in 22 patients (17 males and 5 females, 21-55 yr old) with recurrent ulceration after vagotomy and gastric drainage (pyloroplasty in 19, gastroenterostomy in 3 cases) and in 36 patients (31 males and 5 females, 18-68 yr old) treated successfully for chronic duodenal ulcers by vagotomy and pyloroplasty. In 19 patients with recurrent ulceration, vagotomy was judged to be incomplete by the insulin test. All control subjects had complete vagotomy. Fourteen patients with a postive response to insulin in the first hr (early) had basal (1.5 ± 0.9 mEq/15 min) and histamine-stimulated (7.6 ± 3.2 mEq/15 min) acid secretions which were similar to those observed in the duodenal ulcer patients before surgery (1.2 ± 1.0 and 9.2 ± 3.2 mEq/15 min, resp.). In 5 patients with recurrent ulcer and a positive response to insulin in the 2nd hr (late), both basal (0.2 mEq/15 min) and histamine-stimulated (3.6 ± 2.3 mEq/15 min) acid secretions were comparable to those found after complete vagotomy for duodenal ulcer (0.3 ± 0.3 and 3.8 ± 2.1 mEq/15 min, resp.). While maximal acid secretion in response to antral stimulation in the duodenal ulcer group was high (4.0 ± 2.4 mEq/15 min) before surgery and was abolished in 23 of 36 patients after surgery, antral function was retained in the majority of patients with recurrent ulcer (3.6 ± 2.4 mEq/15 min). Among patients with an early positive response to insulin, 4 showed responses to antral stimulation similar to those observed with histamine stimulation; in the patients with a late positive response, the residual antral function was not particularly marked. Two of 3 patients with recurrent ulceration despite complete vagotomy had marked antral activity, and the response to antral stimulation paralleled the maximal acid output of the stomach to histamine infusion.

8166 **THE ASSOCIATION BETWEEN PEPTIC ULCERS OR GASTRITIS AND CHOLELITHIASIS.** *(Ger.)* Kuss, B. (St. Johannes Hosp., Duisburg-Hamborn, Germany) and V. Mehrabi. *Zbl Chir* 92(50):2979-2985, 1967.

Radiologically or surgically confirmed cholelithiasis was found in 18 of 325 patients with peptic ulcers (5.5%) and in 4 of 80 patients with gastritis (5.0%). If presumptive diagnoses are included, 9.9% of all patients with peptic ulcers and gastritis had cholelithiasis. There were no clinical symptoms of cholelithiasis in about 67% of these cases. Cholelithiasis occurred about twice as often in patients with atypical stomach disease as in those with typical symptoms of peptic ulcers or gastritis. No significant correlation could be established between the incidence of cholelithiasis and gastric acidity or the course of the gastric disease, although the incidence of cholelithiasis increased from 8.1% of 186 patients with hyperacidity to 15.4% of 26 patients with hypoacidity, and cholelithiasis was found in 3 of 8 patients with hemorrhagic gastritis. Among another group of 281 patients with cholelithiasis and 68 with cholecystitis, 10 (2.9%) were found to have probable or confirmed peptic ulcers and 32 (9.2%) had probable or confirmed gastritis. The incidence of gastric disease in patients treated for gallbladder disease was thus about the same as the incidence of gallbladder disease in patients treated for gastric conditions. These results provide no support for the hypothesis of a causative relation between peptic ulcers, or gastrectomy, and cholelithiasis.

8167 **THE ASSOCIATION OF PEPTIC ULCER AND BRONCHIAL CARCINOMA.** *(Ger.)* Salzer, G. (Surg. Clin., U. Innsbruck, Austria), H. Kutschera and A. Decristoforo. *Bruns Beitr Klin Chir* 216(4):316-321, 1968.

An analysis of autopsy findings in 7130 men and 5737 women all over 30 yr old, showed that the incidence of bronchial carcinoma was significantly higher among men with chronic or healed peptic ulcers (10.2%) than among those with no stomac diseases (5.9%). There was also a significantly higher morbidit for bronchial carcinoma among men who had undergone gastri resection for ulcers (11.5%). The difference in the incidence c bronchial carcinoma among men with chronic or healed peptic ulcers and those who had undergone gastric resection for pepti ulcers was not significant. None of these correlations could be established for women. This association between peptic ulcers and bronchial carcinoma is attributed to cigarette smoking, cor sidered to be an important etiological factor in both diseases.

8168 **THE EFFECT OF ULCERATION ON THE ABIL ITY OF THE GASTRIC MUCOSA TO CONTAIN AN ACID SOLUTION.** *(E.)* Wlodek, G. K. (U. Surg. Clin., McGill U., Montreal, Quebec, Canada) and R. K. Leach. *Canad J Surg* 11(1):5-13, 1968.

Using 10 g/liter polyethylene glycol as a volume marker, the *in* *situ* ionic fluxes and volume changes within resting and histamine-stimulated Heidenhain and Pavlov gastric pouches were studied and the effects of induced and spontaneous ulceration on ionic changes in denervated and innervated gastric pouches, resp., were determined. After ulcer induction in Heidenhain pouch dogs by the suction-thermocautery technique, the total loss of hydrogen ions (285 μEq) in exchange for sodium ions (268 μEq) was significantly increased compared with the hydro gen (234 μEq) and sodium (196 μEq) ion fluxes before ulceration. The mean half-hourly volume changes in gastric acid secr tion before (1.24 ml) and after (1.41 ml) ulceration were not statistically different. It is assumed that the increased H^+ loss occurred through the ulcer crater where the granulation tissue allows free exchange of sodium for hydrogen ions. Prior to ulceration in these Heidenhain pouches, s.c. injection of histamine (3-5 mg) increased the mean half-hourly acid secretion fro a basal level of 1.24 ml to 8.45 ml; the mean half-hourly net Na^+ gain decreased from 196 to 139 μEq. In the same animals after ulcer induction, comparable volume changes were observe after histamine stimulation (from 1.41 to 8.00 ml) and the half hourly net gain of sodium decreased from 268 μEq before to 184 μEq after administration of histamine. These findings indicate that the ability of the rest of the gastric mucosa to contain an acid solution is unaffected by the presence of an ulcer in the pouch. Studies in 2 dogs with spontaneous ulceration o Pavlov pouches also suggested that the increased loss of hydrog ions could be due to an ion-for-ion exchange for sodium secondary to the ulcer. Since ulceration in either the innervated or denervated pouches did not produce a general impairment of

e mucosal barrier, it is concluded that the increased loss of
drogen in exchange for sodium ions occurs through the ulcer
ter itself and not through a mucosal barrier defect in the
nulcerated mucosa. This abnormal acid flux could result in
hemical peritonitis, causing the pain of peptic ulceration.

69 **SELECTIVE GASTRIC VAGOTOMY: A CRITICAL REAPPRAISAL.** *(E.)* Kraft, R. O.
. Michigan Med. Ctr., Ann Arbor), W. J. Fry, K. G. Wilhelm
d H. K. Ransom. *Arch Surg* 95(4):625-630, 1967.

lective gastric and standard total abdominal vagotomies,
mbined with a drainage procedure, were performed in an
ernating manner in 100 consecutive patients with gastro-
odenal ulcers or hiatus hernia. Four-yr follow-up informa-
n was obtained from 76 surviving patients. Both procedures
ere 90% effective in controlling the ulcer diathesis. A 50%
cidence of 1 or more side effects was noted, but these were
rely of even moderate severity. Total patient satisfaction
as good or excellent in 82% of cases. Specific advantages
tributable to the selective form of vagotomy could not be
nfirmed.

70 **THE PHYSIOPATHOLOGY, SURGICAL ANATOMY, AND TECHNIQUE OF VALDONI'S
ASTRODUODENAL RESECTION FOR ULCERS.** *(It.)*
tzu, E. (Munic. Hosp., Taranto, Italy). *G Ital Chir* 23(1):
3-235, 1967.

follow-up study of 387 patients who had undergone Valdoni's
ocedure for gastric resection 8 months to 10 yr before showed
at 4 had died of unrelated causes and 383 survived. Examin-
ions and histories showed that the results of gastrectomy were
cellent or good in 310 cases, poor in 67, and unsatisfactory
6. The reasons for poor or unsatisfactory results were an-
tomotic inflammation in 35 cases, late post-prandial syndrome
15, dumping syndrome in 10, other early post-prandial syn-
omes in 6, dehiscence of the duodenal stump in 5, a gastrec-
my-induced peptic ulcer in 1, and diarrhea in 1. Valdoni's
ocedure is described in detail.

171 **THE INCIDENCE OF PEPTIC ULCER IN BILIARY TRACT DISEASES.** *(Ger.)*
ettenhäuser, K. (Radiol. Clin., Acad. Med., Erfurt, Germany)
d H. Mühlan. *Radiol Diagn (Berlin)* 8(5):539-544, 1967.

adiological contrast studies of the stomach and duodenum
ere made on 686 of 1500 patients who had been examined
r biliary tract disease but whose symptoms were atypical.
hanges in the stomach and duodenum were found in 261
ses (38%). These included post-ulcerative scarring of the
ulb in 70, duodenal diverticula in 36, periduodenal adhesions
24, duodenal ulcers in 23, stomach ulcers in 20, stomach
mors in 13, hiatus hernias in 7, and post-ulcerative scarring
the stomach in 5. Stomach ulcers were found in 7 (4.3%)
d duodenal ulcers in 19 (11.7%) of the 161 patients with
diologically normal gallbladders and biliary tracts and in 13
3%) and 4 (4%), resp., of the 100 patients with gallbladder
d biliary tract abnormalities. Because of the relatively large
mber of patients with duodenal diverticula and peptic ulcers
d their sequelae in both groups, it is recommended that
diological examinations of the stomach and duodenum be
mbined with cholangiography and cholecystography in pa-
ents with atypical upper abdominal symptoms.

8172 **PREVENTION OF STEROID ULCEROGENESIS BY VAGOTOMY IN DOGS.** *(E.)* Göksen, Y. (U. Mississippi Med. Ctr., Jackson) and J. D. Hardy. *Surg Gynec Obstet* 126(3):547-551, 1968.

The protective role of vagotomy and pyloroplasty in the prevention and healing of steroid-induced ulcers (cortisone acetate, 200 mg/day) was studied in 95 adult mongrel dogs. The experimental protocol was divided into 4 parts: (I) the ulcerogenic effect of 200 mg cortisone acetate daily for 2 months was determined in 12 dogs with an interposed segment of ileum; 14 similarly prepared dogs served as controls; (II) the effect of unilateral vagotomy on steroid ulcerogenesis was assessed in 20 dogs with interposed loops and anterior vagotomy, half treated with cortisone, and in 20 dogs with posterior vagotomy, half treated with cortisone; (III) the incidence of ulcers in the interposed segment of the ileum in dogs with bilateral vagotomy was determined in 12 controls and 17 cortisone-treated animals; and (IV) the effects of cortisone treatment on the healing of ulcers were studied in dogs in which ulcers had already developed; the degree of ulceration present in the ileal segment was noted at bilateral vagotomy in 10 dogs, and after a 4-day recovery period, cortisone was given for 2 months. In the 1st experiment, ulceration developed in the interposed ileal segment in 9 of 14 untreated dogs and in all 12 cortisone-treated dogs. Unilateral vagotomy did not significantly alter the incidence of ulceration in the ileal loop in the cortisone-treated group, but bilateral vagotomy did significantly reduce the incidence of ulceration in both cortisone-treated and untreated dogs. Bilateral vagotomy permitted the healing of established ulcers, even though cortisone therapy was continued. Vagotomy plus a drainage procedure may be considered an effective operation for the surgical management of uncomplicated steroid ulceration.

8173 **SURGICAL TREATMENT OF CURLING'S ULCER.** *(E.)* O'Neill, J. A. Jr. (Brooke Army Med. Ctr., Fort Sam Houston, Texas), B. A. Pruitt Jr. and J. A. Moncrief. *Surg Gynec Obstet* 126(1):40-44, 1968.

Curling's ulcers developing after thermal injury in 24 patients (21 males and 3 females, 8 months to 49 yr old) were treated surgically. The burned surface area in these patients ranged from 13% to 72%. Ten of the 24 (42%) survived the operation. Two patients had perforated duodenal ulcers and the remaining 22 were operated on for massive hemorrhage from duodenal or gastric ulcers. Practically every patient was asymptomatic prior to the onset of bleeding or perforation. Although several types of operative procedures were utilized, vagotomy and antrectomy with possible removal of the ulcer was considered to be the preferred mode of treatment. Even if a duodenal ulcer is found to be the source of either bleeding or perforation, a careful search must be made for associated gastric ulcers.

8174 **ANALYSIS OF USE OF VAGOTOMY WITH DRAINAGE PROCEDURE IN SURGICAL MANAGEMENT OF DUODENAL ULCER.** *(E.)* Whittaker, L. D. Jr. (Mayo Clin. Found., Rochester, Minn.), E. S. Judd and M. H. Stauffer. *Surg Gynec Obstet* 125(4):1018-1026, 1967.

The results of vagotomy combined with a gastric drainage procedure for peptic ulcer are discussed on the basis of experience with 436 patients during a 15-yr period. The postoperative follow-up period averaged 8.2 yr in the vagotomy and gastroenterostomy group and 3.7 yr in the vagotomy and pyloroplasty group. The hospital mortality rate for the series was 2.2%. Postoperative complications included gastric stasis without organic obstruction, which required reoperation in the immediate postoperative period in 1.1%, and dysphagia requiring treatment in 0.9%. Unsatisfactory results were obtained in 9.1%. This figure includes suspected, but unproved, recurrences in 1.2%, proved recurrences in 5.6%, subsequent development of gastric ulcer in 1.4%, patient dissatisfaction in 0.7%, and pyloric obstruction after pyloroplasty in 0.2%. The overall result was unsatisfactory in 9.9% of the vagotomy and gastroenterostomy group and 6% of the vagotomy and pyloroplasty group. The dumping syndrome occurred in 8 patients, although it was mild in 5 of these. Troublesome diarrhea and nutritional problems were virtually absent.

8175 PATHOGENESIS OF GASTRIC ULCERATION PRODUCED UNDER STRESS.

(E.) Singh, G. B. (Indust. Toxicol. Res. Ctr., Lucknow, India), J. N. Sharma and K. Kar. *J Path Bact* 94(2):375-380, 1967.

The frequency of gastric ulcers produced by stress and the variations in the histamine content of the blood and stomach wall in relation to the degree of gastric ulceration produced were studied in 35 male guinea pigs (average weight 500 g). Stress was induced by placing the animals on a linear rocking machine at 200 to-and-fro movements/min. The gastric mucosa in control animals showed contracted, irregular, longitudinal folds while 8 of the 22 stressed animals developed various grades of mucosal hemorrhage, erosion and ulceration. No ulcerative lesions could be demonstrated microscopically in control animals. Hemorrhagic erosions with depletion of the superficial mucosal cells were evident in stressed animals and the submucosa below the ulcerated area showed edema, congestion of blood vessels and infiltration with large numbers of inflammatory cells. The total volume of gastric juice was significantly raised during stress but there was no direct correlation between the increase in the total volume of gastric juice and the degree of ulceration in this group. No significant differences were seen in the free acid and the stomach histamine values, although there was a slight increase in free acidity during stress. Differences with regard to blood histamine levels were highly significant between controls and the ulcerated animals of the stressed group, as well as between ulcerated and nonulcerated animals in this group, but not between controls and the nonulcerated animals of the stressed group.

8176 GASTRIC SECRETION IN DUODENAL DISEASES.

(It.) Allegra, G. C. (Maggiore Hosp., Novara, Italy) and S. Maduli. *G Clin Med* 48(8):855-883, 1967.

Lambling's method was used to study gastric acid secretion before and after administration of 0.5 mg of histamine dihydrochloride in 91 patients with idiopathic duodenal ulcers, 10 with duodenal ulcers and chronic liver disease, 2 with prolapse through the pylorus, 5 with duodenal diverticulosis, and 9 with duodenitis. In most cases, disorders had been diagnosed from biopsies of the fundal mucosa. In patients with idiopathic duodenal ulcers hyperchlorhydria was found in 38% before histamine stimulation and in 56% after. Gastric acid secretion was normal in all those with duodenal ulcers and chronic liver disease. No correlation was found between gastric acid secretion and the length of the disease, age of the patient, or development of complications. The only significant correlation was a tendency for the acid output to decrease in patients over 60 y old. A tendency toward hyperchlorhydria was found in patients with duodenal diverticulosis and in those with gastric prolapse. This is attributed to gastrin sensitization of parietal cells to histamine stimulation. The duodenal bulb is believed to be an important factor in the development of the tendency toward hyperchlorhydria seen in patients with pseudoulcerativ duodenitis.

8177 POST-VAGOTOMY SIDE-EFFECTS AND BLOO GROUP SUBSTANCES.

(E.) Pringle, R. (Dept. Surg., Queen's Coll., Dundee, Scotland). *Brit J Surg* 55(1):41 43, 1968.

The incidence of side-effects occurring 10 yr or more followin bilateral vagotomy and gastrojejunostomy for chronic duodena ulcers was determined in 175 patients and correlated with the distribution of ABO blood groups and secretor status. Diarrhe occurred in 35 (30.2%) secretors and in only 8 (13.6%) nonsecretors. Iron-deficiency anemia occurred more often than e pected in individuals in blood-group A, being found in 29.3% of A group secretors and 14.3% of A group nonsecretors. Thi suggests that there may be an interaction between the presenc or absence of the various blood group factors and the liability of patients to develop diarrhea and iron-deficiency anemia following vagotomy and gastroenterostomy for chronic duodenal ulcer. The overall incidence of side effects (32%) showed no correlation with blood group secretor status, however.

8178 CONTROLLED CLINICAL EVALUATION OF THE THERAPEUTIC VALUE OF AN ANTACI COMBINED WITH AN ANTIULCER AGENT.

(It.) Lattanz A. (Santa Chiara Hosp., Pisa, Italy) and E. De Liperi. *Minerv Med* 58(79):3736-3743, 1967.

Preliminary clinical trials of F.I. 6228/N were run. Each capsule contains 1.25 mg of 2,2-diphenyl-4-hexamethyleneiminobutyramide methiodide, 1.00 mg of cyamemazine, 5.00 mg of papaverine hydrochloride, 150 mg of activated attapulgite, 25 mg of magnesium oxide, 50 mg of anhydrous alumin gel, 10 mg of sodium stearate, and 250 mg of lactose. Symp matic improvement was noted in 13 of 15 patients with pepti ulcers and a variety of other gastrointestinal diseases. F.I. 6628/N was compared with F.I. 6323/B, which contains the antacids alone, in a double-blind study. The subjects included 2 groups of 20 patients each matched for age, disease, and fre quency and type of symptoms. Patients with hyperchlorhydr gastritis were given 2 capsules after each meal. In addition, patients with peptic ulcers received 2 capsules 1 hr before pai usually developed. Statistical treatment of results showed tha the combined antacid and antiulcer preparation was more effective than the antacid alone with a margin of error of 5%.

8179 CURRENT CONCEPTS OF THE ETIOLOGY AND PATHOPHYSIOLOGY OF PEPTIC ULCER DISEASE.

(Ger.) Szarvas, F. (1st Med. Clin., U. Szeged, Hungary) and V. Varró. *Zbl Chir* 92(26a):1211-1217, 1967.

8180 ETIOLOGY AND TREATMENT OF POSTOPERA TIVE GASTRODUODENAL ULCER.

(Ger.) Konrad, R. M. (Surg. Clin., U. Dusseldorf, Germany). *Zbl Chir* 92(26a):1225-1227, 1967.

81 **THE SURGICAL TREATMENT OF DIFFICULTY RESECTABLE DUODENAL ULCERS.** *(Ger.)* umann, H. D. (Distr. Hosp., Dresden-Friedrichstadt, Germany). *l Chir* 92(26a):1260-1264, 1967.

82 **INDICATIONS AND RESULTS OF CORRECTIVE SURGERY FOLLOWING GASTRIC RESECTION R GASTRODUODENAL ULCERS.** *(Ger.)* Hart, W. (Surg. n., U. Munich, Germany). *Zbl Chir* 92(26a):1246-1260, 67.

83 **POSSIBILITIES AND LIMITATIONS OF A FUNCTIONAL TECHNIQUE FOR PEPTIC UL- R SURGERY.** *(Ger.)* Hollender, L. F. (Surg. Clin., U. asbourg, France) and A. G. Weiss. *Zbl Chir* 92(26a):1236-44, 1967.

84 **COMPLICATIONS FOLLOWING GASTRIC RESECTION FOR ULCER AND THEIR TREAT- NT.** *(Ger.)* Rueff, E. (Surg. Clin., U. Munich, Germany). *l Chir* 92(26a):1266-1271, 1967.

85 **LONG-TERM RESULTS OF PEPTIC ULCER SURGERY IN THE LIGHT OF THE CRITICISM MING FROM INTERNISTS AND THE RESULTS OF LLOW-UP STUDIES ON UNIFORM SURGICAL GROUPS.** *er.)* Schmauss, A. K. (Friedrichshain Hosp., Berlin, Germany), Bahrmann, W. F. Poley, C. Lorisch, A. Jungbluth, N. ascheck, J. Karrenberg and R. Meyer. *Zbl Chir* 92(26a):1293-99, 1967.

86 **LONG-TERM RESULTS OF GASTRIC RESECTION IN PEPTIC ULCER DISEASE.** *(Ger.)* the, W. (Surg. Clin., U. Greifswald, Germany). *Zbl Chir* (26a):1287-1293, 1967.

87 **GASTROPLEGIA FOLLOWING VAGOTOMY: SUCCESSFUL TREATMENT BY GASTRECTOMY.** *r.)* Perrotin, J. (Lariboisiere Hosp., Paris, France), C. Boulakia d P. de Lagausie. *Ann Chir* 21(23/24):1421-1425, 1967.

88 **SELECTIVE GASTRIC VAGOTOMY.** *(Heb.)* Reiss, R. (Surg. Clin. B, Meir Hosp., Kfar Saba, ael). *Harefuah* 74(12):457-459, 1968.

89 **GASTRODUODENAL PERFORATION CAUSED BY DRUGS.** *(Sp.)* Juliá Daurella, R. (Clin. Hosp., rcelona, Spain). *An Med [Cir]* 53(4):346-352, 1967.

90 **IATROGENIC ULCER DUE TO STEROIDS.** *(Sp.)* Albornoz Plata, A. (No. 7-18, Calle 66, gota, Colombia). *Medicina (Mex)* 49(1040):356-361, 1968.

191 **EVALUATION OF THE SELECTION OF PATIENTS WITH PEPTIC ULCERS FOR SANA- RIUM CARE.** *(Pol.)* Lazowski, J. (Patria-Kasztelanka nat., State Health Resort Enterpr., Krynica-Zegiestow, Poland). *iad Lek* 21(11):943-946, 1968.

192 **SOME CONSIDERATIONS ON VAGOTOMY AND PYLOROPLASTY: PART I. EXPERIMEN- L STUDIES ON ITS EFFECT ON WEIGHT GAIN.** *(It.)* Vassanelli, P. (Inst. Surg. Path., U. Padua, Italy), G. P. Spina and A. Maffei Faccioli. *Chir Ital* 19(Suppl. 1):195-208, 1967.

8193 **VAGOTOMY IN THE TREATMENT OF BLEEDING PEPTIC ULCERS: REVIEW OF THE IMMEDIATE AND LONG-TERM RESULTS IN 42 CASES.** *(It.)* De Vecchi Pellati, M. (Fatebenefratelli-Fatebenesorelle Ciceri Agnesi Hosp., Milan, Italy), W. Rossi, F. Falcone and N. Fiorenzano. *Chir Ital* 19(Suppl. 1):94-105, 1967.

8194 **IMMEDIATE DEFINITIVE SURGERY FOR PERFORATED PEPTIC ULCERS.** *(E.)* Balasegaram, M. (Gen. Hosp., Seremban, Malaysia). *Amer J Surg* 115(5):642-647, 1968.

8195 **AFFERENT LOOP OBSTRUCTION.** *(E.)* Frey, C. F. (U. Michigan Med. Ctr., Ann Arbor) and W. W. Coon. *Amer J Surg* 115(5):730-733, 1968.

8196 **A STUDY OF THE FUNCTIONAL STATE OF THE LIVER IN ULCER PATIENTS WITH THE AID OF ROSE BENGAL.** *(Rus.)* Rusanov, A. A. (Leningrad Inst. Pediat. Med., USSR), V. P. Zinevich, O. N. Nikonova, L. M. Goreva and I. Ia. Chupin. *Vestn Khir Grekov* 100(6): 23-26, 1968.

8197 **TREATMENT OF DUODENAL AND GASTRIC ULCERS BY SELECTIVE VAGOTOMY WITH PYLOROPLASTY.** *(Rus.)* Seniutovich, R. V. (Ivano-Frankovsk Med. Inst., USSR) and G. S. Izbenko. *Vestn Khir Grekov* 100(6):20-23, 1968.

8198 **THE EFFECT OF PADRIN ON PEPTIC ULCERS AND PAIN.** *(E.)* Miyoshi, A. (Fac. Med., Kyoto U. Japan). *Jap Arch Intern Med* 14(7):211-217, 1967.

8199 **PHYSIOPATHOLOGICAL ASPECTS OF THE ADRENAL CORTEX IN COMPLICATED DUODENAL AND GASTRIC ULCERS.** *(Rum.)* Săvulescu, V. (Tudor Vladimirescu Distr. Hosp., Romania), I. Andronache, S. Ţincu, M. Corpade-Mateica and P. Teodorescu. *Med Intern (Bucur)* 19(11):1311-1320, 1967.

8200 **THE INFLUENCE OF DUODENAL FACTORS UPON THE GENESIS OF GASTRIC ULCER OF RATS.** *(E.)* Kim, H. Y. (Yonsei U. Coll. Med., Seoul, Korea). *Korean J Intern Med* 10(12):775-786, 1967.

8201 **ANTIPERISTALTIC SEGMENT OF JEJUNUM FOR PERSISTENT DIARRHEA FOLLOWING VAGOTOMY. [CASE REPORT]** *(E.)* Craft, I. L. (Westminister Hosp., London, England) and C. W. Venables. *Ann Surg* 167(1):282-286, 1968.

8202 **CHOLEDOCHODUODENAL FISTULA: THE NATURAL HISTORY AND MANAGEMENT OF AN UNUSUAL COMPLICATION OF PEPTIC ULCER DISEASE.** *(E.)* Constant, E. (U. Michigan Med. Ctr., Ann Arbor) and J. G. Turcotte. *Ann Surg* 167(2):220-228, 1967.

8203 **VAGOTOMY AND PYLOROPLASTY IN INFANCY AND CHILDHOOD.** *(E.)* Johnston,

P. W. (Child. Hosp., Los Angeles, Calif.) and W. H. Snyder Jr. *J Pediat Surg* 3(2):238-245, 1968.

8204 CONSTITUTION, GENETICS, AND BODY FORM IN PEPTIC ULCER: A REVIEW. *(E.)* Damon, A. (Dept. Anthropol., Harvard U., Cambridge, Mass.) and A. P. Polednak. *J Chron Dis* 20(10):787-802, 1967.

8205 ULCEROGENIC TUMOR, A CASE REPORT. *(E.)* Smalley, R. S. (St.-Ann's Hosp., Juneau, Alaska) and H. Wilde. *Alaska Med* 10(1):28-31, 1968.

8206 HISTAMINE METABOLISM IN PATIENTS WITH GASTRODUODENAL ULCERS. *(Rus.)* Mozhaiskaya, N. M. (Acad. Med. Sci., Leningrad, USSR). *Ter Arkh* 39(11):83-86, 1967.

8207 STRUCTURE OF THE GASTRIC MUCOSA IN PATIENTS WITH GASTRODUODENAL ULCER. *(Rus.)* Pleshkaneva, A. F. (Acad. Med. Sci., Leningrad, USSR). *Ter Arkh* 39(11):89-91, 1967.

8208 DYNAMICS OF THE PROTEIN FORMULA OF BLOOD SERUM AND SECRETION OF ACID COMPONENT OF THE GASTRIC JUICE IN PATIENTS WITH UNCOMPLICATED GASTRODUODENAL ULCER. *(Rus.)* Geller, L. I. (Khabarovsk Med. Inst., USSR), A. P. Smyshlyaeva and E. A. Svetacheva. *Ter Arkh* 39(11):86-89, 1967.

8209 TREATMENT OF PERFORATING STOMACH AND DUODENAL ULCERS. *(Pol.)* Soltysiak, A. (3rd Surg. Clin., Acad. Med., Lodz, Poland). *Pol Przegl Chir* 40(4):352-357, 1968.

8210 PERFORMANCE OF VAGOTOMY FOR TREATMENT OF PERFORATION OF DUODENAL ULCER. *(Pol.)* Chrościcki, S. (Acad. Med., Warsaw, Poland) and H. Umińska. *Pol Przegl Chir* 40(1):30-34, 1968.

8211 APPLICATION OF ACIDIMETRY IN THE MEDICAL AND SURGICAL TREATMENT OF PEPTIC ULCERS. *(Sp.)* Meeroff, M. (Prof. Dr. Gregorio Araoz Alfaro Polyclin., Buenos Aires, Argentina), J. E. Segal, J. C. Meeroff, J. Mandelman, C. Casadavant and R. Quiros. *Medicina (Mex)* 47(1020):444-450, 1967.

8212 AN INFREQUENT POSTOPERATIVE DEVELOPMENT OBSERVED IN A YOUNG MALE PATIENT WITH PERFORATED DUODENAL ULCER. *(Rum.)* Arnautu, I. (Inst. Med. Pharm., Iasi, Romania), G. Kreisler, C. Dragomir and M. Diaconescu. *Rev Medicochir Iasi* 72(1): 215-218, 1968.

8213 ANTROPYLORIC RESIDUE IN POSTOPERATIV PATHOGENESIS OF GASTRODUODENAL ULCERS. *(It.)* Fesani, F. (Inst. Gen. Surg. Clin., U. Parma, Italy) and P. Bobbio. *Ann Ital Chir* 43(4):673-681, 1967.

8214 PYLORIC STENOSIS OF ULCEROUS ORIGIN IN THE INFANT. *(Fr.)* Ingelrans, P. (Region. Hosp. Ctr., Lille, France), M. Lacheretz and P. Debeugny. *Pediatrie* 22(7):817-824, 1967.

8215 DIARRHEA FOLLOWING VAGOTOMY. *(Pol.)* Uszyński, H. (Acad. Med., Warsaw, Poland) and A. Kotarba. *Pol Przegl Chir* 40(1):98-101, 1968.

8216 EXPERIMENTS IN THE TREATMENT OF DIARRHEA FOLLOWING VAGOTOMY. *(Pol.)* Kotarba, A. (Acad. Med., Warsaw, Poland), H. Uszyński and J. Potocki. *Pol Przegl Chir* 40(2):116-119, 1968.

8217 RADIOLOGICAL AND CLINICAL DIAGNOSIS OF GASTRODUODENAL ULCERS IN CHILDREN. *(Rum.)* Pîrvulescu, M. (United Hosp., Bucau, Romania). *Rev Medicochir Iasi* 71(3):587-592, 1967.

See also: 7675,7676,7680,7686,7849,7882,7910,8035,8042,8049,8082,8102,8108,8141,8443,8694

18 TUBERCULIN SENSITIVITY IN CROHN'S DISEASE: A CONTROLLED STUDY. *(E.)* etcher, J. (Central Middlesex Hosp., London, England) and M. Hinton. *Lancet* 2(7519):753-754, 1967.

uberculin sensitivity was studied in 71 patients with Crohn's sease (40 with small intestinal, 21 with colonic, and 10 with orectal involvement) and in 71 control patients matched ith the Crohn's patients for age, sex, and race. Patients were sted in either an active or quiescent phase of the disease, but ot when severely ill. No patient was tested while receiving eatment or within 2 months of treatment with corticosteroids. nly 4 control subjects and 2 patients with Crohn's disease had ceived BCG (Bacillus Calmette-Guerin) vaccine. Each subject ceived i.c. injections in a forearm of both 1 and 10 T.U. and e reactions were read at 72 hr. If both were negative, the st was repeated with 100 T.U. Induration of 5 mm or more as considered a positive reaction. Comparison of patients with ohn's disease and their matched controls showed no difference tuberculin sensitivity: of the patients with Crohn's disease, were positive to 1 T.U.; 38 were positive to 10 T.U.; and were positive to 100 T.U. Respective figures for the control oup were 24, 39 and 50. When the patients were grouped cording to the anatomical site of the disease, there was again significant difference between the patients with Crohn's sease and the controls. These data supply further evidence at Crohn's disease is distinct from both sarcoidosis and tuberlosis.

19 PSYCHIATRIC STUDY OF A CONSECUTIVE SERIES OF 19 PATIENTS WITH REGIONAL EITIS. *(E.)* Feldman, F. (Cedars-Sinai Med. Ctr., Los geles, Calif.), D. Cantor, S. Soll and W. Bachrach. *Brit Med* (5581):711-714, 1967.

comparative psychiatric study of 19 consecutive, unselected ses of regional ileitis (10 males and 9 females, 10-70 yr old time of onset of disease) and 74 controls rerealed no evidence at an emotional factor was a significant etiological agent. venteen of the 19 patients were regarded as "normal". The sults of this study are qualified by questions concerning the ncept of normality; nevertheless, the significant emotional ctors relating to psychosomatic illness noted in the literature were investigated and found not to apply to any important degree. It is questionable, therefore, whether the disease should be regarded as psychosomatic in any way.

8220 ADENOCARCINOMA ASSOCIATED WITH CROHN'S DISEASE. *(E.)* Sheil, F. O'M. (Gen. Infirm., Leeds, England), C. G. Clark and J. C. Goligher. *Brit J Surg* 55(1):53-58, 1968.

Two cases (both females, 40 and 50 yr old) of carcinoma found in association with Crohn's disease of the intestine are described. One tumor developed in the ileum in a patient with ileitis and the other developed in the rectum in a patient with mainly large-bowel involvement. As indicated by the finding of only 13 such cases in the literature, this association is uncommon; the incidence of cancer complicating Crohn's disease of the small intestine may be no greater than the incidence arising spontaneously. The recorded incidence of cancer complicating Crohn's disease of the colon is still lower, and contrasts particularly with the high incidence in patients with ulcerative colitis.

8221 SURGICAL TREATMENT OF CROHN'S DISEASE. *(Fr.)* Edelmann, G. (La Pitié Hosp., Paris, France). *Actualites Hepatogastroent Hotel Dieu* 4(1): A95-A103, 1968.

8222 MEDICAL TREATMENT OF CROHN'S DISEASE. *(Fr.)* Crismer, R. (Beauregard Med. Ctr., Liege, Belgium). *Actualities Hepatogastroent Hotel Dieu* 4(1): A85-A94, 1968.

8223 CROHN'S DISEASE OF THE SMALL INTESTINE: MACROSCOPIC AND MICROSCOPIC ASPECTS. *(Fr.)* Morson, B. C. (St. Mark's Hosp., London, England). *Actualities Hepatogastroent Hotel Dieu* 4(1):A59-A65, 1968.

8224 CLINICAL STUDY OF CROHN'S REGIONAL ILEITIS. *(Fr.)* Paris, J. (Dig. Dis. Clin., Lille, France). *Actualites Hepatogastroent Hotel Dieu* 4(1):A2-A14, 1968.

See also: 7903,7933,8230,8232,8254,8313,8327,8370

Malabsorption

8225 **THE MOLECULAR LESION OF SACCHAROSE INTOLERANCE.** *(E.)* Eggermont, E. (Lab. Physiol. Chem., U. Louvain, Belgium). *Bibl Paediat* (87):121-126, 1968.

Data from 8 patients, 5.5 months-14 yr of age, with hereditary sucrose intolerance revealed significantly lower maltase, isomaltase, sucrase, and γ-amylase activities in the small intestinal mucosa than those found in 35 control subjects in the same age group or younger. Trehalase and alkaline phosphatase activities and protein content were similar in the 2 groups. Sucrose intolerance is concluded to be the result of a single genetic mutation affecting the enzymes with maltase-sucrase activity, the loss of which results in the abnormal association of maltase-isomaltase and γ-amylase, causing a reduction in their activities as well. The multiple enzymic lesion of sucrose intolerance is therefore not in contradiction to the basic law of genetics.

8226 **NORMAL AND DEFECTIVE PRODUCTION OF IMMUNOGLOBULINS IN THE INTESTINAL TRACT.** *(E.)* Crabbé, P. A. (Dept. Int. Path., U. Louvain, Belgium) and J. F. Heremans. *Bibl Paediat* (87):161-181, 1968.

Immunofluorescent studies of normal gastrointestinal mucosal biopsies showed that 80% of the subepithelial plasma cells contained the exocrine immunoglobulin IgA; these findings were not only consistent in all normal mucosal biopsies tested, but also at almost every level of the gastrointestinal tract except the appendix. The number of IgA-containing plasma cells was greater in the gastrointestinal tract than in any other lymphoid tissues tested. Patients with primary hypogammaglobulinemia and other intestinal disorders were then investigated in order to determine the relation between gastrointestinal disease and the immunoglobulins. In 4 patients, 3 female adults (aged 45-65 yr) and 1 male child (4 yr old), with nonselective hypogammaglobulinemia, steatorrhea and diarrhea, there was a simultaneous decrease in circulating and gastrointestinal mucosal IgA, IgM, and IgG, and a severe reduction in the number of plasma cells in the villi and between the crypts. An increase in polymorphonuclear eosinophils and lymphoid follicle hyperplasia were also observed. Rectal biopsies on 2 patients revealed a scarcity of plasma cells; rectal biopsy on a 3rd patient showed rich infiltration of plasma cells while the jejunal biopsy of the same subject was almost devoid of such cells. There was a good correlation between plasma cell numbers and immunoglobulin-containing cells. Gluten-free diets and liver therapy were not lastingly beneficial. In 1 male and 2 females (59-65 yr of age) with selective IgA deficiency and chronic, moderately severe steatorrhea (IgA-deficient sprue), biopsies showed abundant plasma-cell infiltration in the gastrointestinal mucosa; however, most of these cells contained IgM instead of IgA. One patient experienced a complete remission with a gluten-free diet but had a relapse when gluten was reintroduced into the regimen; a 2nd patient had a partial remission on the same regimen. Intestinal biopsies of 5 patients with multiple myeloma, another major derangement of the immunoglobulin system, revealed no immunohistochemical abnormalities; the patients exhibited no gastrointestinal symptoms, although mucosal lesions were present as localized tumors. Diffuse involvement of intestinal plasma cells is apparently quite rare in multiple myeloma, but might be associated with absorptive malfunctions. Further investigation is necessary to elucidate the frequency and significance of the association of gastrointestinal functional disorders, intestinal subepithelial lymphoid tissue abnormalities, the presence of a monoclonal immunoglobulin component, and the clinical picture of multiple myeloma.

8227 **FAMILIAL CHLORIDE DIARRHEA-CHLORID MALABSORPTION.** *(E.)* Launiala, K. (Childre Hosp., U. Helsinki, Finland), J. Perheentupa, A. Pasternack and N. Hallman. *Bibl Paediat* (87):137-149, 1968.

Familial occurrence, hydramnios, a gestation period of less than 38 weeks, excessive weight loss, hyperbilirubinemia, persistent diarrhea from the first few days of life, low serum electrolyte and urinary Cl^- levels, and signs of postnatal ileus were the major neonatal characteristics in 3 male and 7 femal children (1 day-14 months of age) treated for familial chlorid diarrhea during the period 1961-67; neonatal stool Cl^- concentrations varied from 30 to 100 mEq/liter. In the early stages of the disease the stool K plus Na concentration was greater than the Cl^- concentration. As the infants became older, ther was a marked retardation of growth and motor development. These were associated with constant watery, slightly acidic diarrhea, and a stool Cl^- of about 150 mEq/liter, which exceeded the combined K and Na concentration. Hypokalemia and hypochloremia persisted, and metabolic alkalosis develope The urinary Cl^- was extremely low and urinary K high. Susceptibility to urinary infections was evident in these patients, and vascular and degenerative kidney changes were observed; renal biopsy revealed juxtaglomerular cell hyperplasia. Secondary hyperaldosteronism with an elevation in plasma renin activity was also noted. Absorption studies in 2 patients showe a high Cl^- concentration, about 140 mEq/liter, throughout the intestinal tract, except the duodenum. The sum of the K and Na concentrations was generally less than the Cl^- concentratio in the intestinal contents. The data suggest that Cl^- absorptio is defective in familial chloride diarrhea and that the defect, most prominent in the ileum and colon, also interferes with th movement of bicarbonate ions; the defect may also lie in the exchange between the 2 anions. Failure of Cl^- absorption lead to electrolyte and water depletion, which leads to juxtaglomer lar hyperplasia, reflected in an increase of plasma renin and subsequently of angiotensin; this causes angiopathy and leads to secondary hyperaldosteronism, which leads to a rise in urinary K and eventual hypokalemic alkalosis.

8228 **METABOLISM OF HYDROXY FATTY ACIDS IN DOGS WITH STEATORRHEA SECONDARY TO EXPERIMENTALLY PRODUCED INTESTINAL BLIND LOOPS.** *(E.)* Kim, Y. S. (Bellevue Hosp., New York, N. Y.) and N. Spritz. *J Lipid Res* 9(4):487-491, 1968.

The metabolism of hydroxy fatty acids was studied in dogs with steatorrhea resulting from experimentally produced blind loops. Hydroxy acids were found to be present in the stools in amounts far above normal (1.1 g/day versus 20 mg/day in controls). During tetracycline administration or after exclusio of the blind loop, these acids disappeared from the feces and the steatorrhea was corrected. However, after administration

taurocholic acid, although the steatorrhea was corrected,
hydroxy acids persisted in higher than normal amounts.
th in normal dogs and in those with blind loops, hydroxy
d constituted a higher percentage of total fatty acids in the
unum than lower down in the gastrointestinal tract. When
droxy acids were fed to normal dogs, steatorrhea was not
oduced and absorption in amounts similar to that of unsub-
tuted stearic acid was noted. A possible conclusion is that
droxy fatty acids have an enterohepatic circulation via the
rtal system. Isotopic oleic and linoleic acids were converted
hydroxy acids both *in vivo* and during *in vitro* incubation
h feces; stearic acid was not. These findings support the
a that hydroxy acids arise by addition of water across double
nds, the reaction being catalyzed by the enzymes of intestinal
cteria.

29 **NORMAL ABSORPTION IN THAI SUBJECTS WITH NON-SPECIFIC JEJUNAL ABNORMALI-**
ES. *(E.)* Troncale, F. J. (SEATO Med. Project, Bangkok,
ailand), G. T. Keusch, L. H. Miller, R. A. Olsson and R. D.
chanan. *Brit Med J* 4(5579):578-580, 1967.

rty asymptomatic Thai subjects (22 females and 18 males,
72 yr old) were studied for evidence of tropical sprue by
ans of the following tests: xylose excretion, ^{57}Co-vitamin
2 and vitamin A absorption, fecal fat excretion, small in-
tine radiographs, and jejunal biopsy. Xylose excretion was
ver than in North American subjects but above the range
pected in tropical sprue. Lactose malabsorption and mild
nspecific histological abnormalities of the jejunal mucosa
re present in all subjects. The other subjects revealed no
normalities. These findings were considered insufficient evi-
nce to support a diagnosis of subclinical tropical sprue, and
refore, no evidence exists for the hypothesis that subclinical
ue is a widespread disease in Thailand.

30 **BILE SALT MALABSORPTION IN REGIONAL ILEITIS, ILEAL RESECTION, AND MANNITOL-**
DUCED DIARRHEA. *(E.)* Meihoff, W. E. (U. Colorado
d. Ctr., Denver) and F. Kern Jr. *J Clin Invest* 47(2):261-267,
58.

al bile-salt loss was investigated in 7 healthy volunteers, 3
ients with regional enteritis, and 6 patients with ileal resec-
n for 5-10 days after oral administration of 10 μC of car-
xyl-^{14}C-cholic acid. Laboratory studies were normal in the
olunteers. In all 3 patients with regional enteritis the jejunal
cosal biopsy and d-xylose excretion were normal. Two pa-
nts had a decreased absorption of ^{57}Co-labeled vitamin B_{12}
h intrinsic factor, and a significant increase in the rate of
salt excretion compared to normal control subjects, even
ugh the intestinal transit time was normal. The decay con-
nts were 0.505 and 0.599 days^{-1}, but only 1 of these pa-
nts had steatorrhea. The third patient had a normal Schilling
t and a normal rate of bile salt excretion (0.1585 days^{-1}).
of the patients with ileal resection demonstrated normal
unal mucosal biopsy. Absorption of ^{57}Co-labeled vitamin
2 with intrinsic factor was abnormally low in 5 patients,
l serum carotene was below 75 μg/100 ml in these 5 pa-
nts. Watery diarrhea was characteristic of this group. The
gest intestinal transit time was 8 hr, compared to an average
26 hr in controls. All 6 patients excreted the labeled bile
rapidly, and 5 excreted 50% of the radioactivity in 24 hr.
The mean decay constant, 1.43 days^{-1}, was much greater in this group, but there was poor correlation of the decay constant with length of resection, stearrhea, and transit time. Four of the control subjects were then given 1200 ml of 10% mannitol orally for 7 days and the labeled cholic acid excretion rate was studied again to determine the effect of rapid intestinal transit on bile salt excretion. The fecal bile salt excretion rate increased slightly, mild steatorrhea developed, and the mean intestinal transit time was markedly shortened. The results indicate that ileal resection and ileal disease are major factors and rapid intestinal transit is a minor factor in causing excessive fecal bile-salt loss.

8231 **THE MECHANISM OF DIARRHOEA IN CONGENITAL DISACCHARIDE MALABSORPTION.** *(E.)* Launiala, K. (Child. Hosp., U. Helsinki, Finland). *Acta Paediat Scand* 57(5):425-432, 1968.

Intestinal malabsorption of lactose and sucrose, and the effect of these disaccharides on the movements of water and electrolytes, were studied in two siblings with congenital lactose malabsorption (CLM) and one infant with congenital sucrose-isomaltose malabsorption (CSIM), using the intestinal intubation technique. Sucrose in the CLM-patients and lactose in the CSIM-patient were absorbed in the proximal part of the small intestine, whereas hardly any lactose (in CLM) or sucrose (in CSIM) disappeared from the small intestinal lumen. The unabsorbed disaccharides caused considerable movement of water and electrolytes into the intestinal lumen, with a 1.5-2.5-fold dilution taking place in the duodenum and proximal jejunum. The contents of the small intestine were isoosmotic with extracellular fluid throughout and the unabsorbed disaccharides represented 25-30% of the osmotic activity. In the colon, the unabsorbed disaccharides disappeared in varying degree, being split into monosaccharides and converted to lactate. Water, Na^+ and Cl^- were absorbed in the colon even in relative excess to the disappearance of the disaccharide and its cleavage products, and in all cases the fecal volume was smaller than the volume of the test fluid. In one CLM-patient a 3-week period on a lactose-containing diet had no effect on the phenomena demonstrated in the small intestine, but disappearance of lactose and formation of lactate was greater in the colon, and the amount of water in the stools was less than before. It is suggested that the retention of water in the intestinal lumen due to the osmotic activity of the unabsorbed disaccharide, together with the organism's tendency to Na^+-equilibrium between the luminal and extracellular fluid, is the most important mechanism of diarrhea in disaccharide malabsorption.

8232 **MALABSORPTION IN GRANULOMATOUS ILEITIS AND ILEOCOLITIS.** *(E.)* Tytgat, G. (Acad. Hosp., Louvain, Belgium), G. Vantrappen, L. Rutgeerts and J. Vandenbroucke. *Acta Chir Belg* (Suppl. II):66-79, 1967.

Various parameters of malabsorption were studied in 55 patients (13 with ileitis and 42 with ileocolitis) over a 3-yr period. The diagnosis was based on the pathologic examination of resected specimens or rectal biopsies in 28 patients and on typical clinical and radiological data in 27 patients. Internal fistulization was present in 15 patients. The severity of the malabsorption syndrome was proportional to the degree of activity of the disease. Decreased serum lipid, cholesterol, and carotenoid levels were present in 70-80% of patients with active disease. Decreased serum iron and low iron-binding capacity were noted in 8 of 10 patients with active disease. Vitamin B$_{12}$ levels were always in the normal range, whereas serum folic acid levels were almost always decreased. Hypoalbuminemia, mainly due to excessive intestinal protein catabolism, was noted to 70% of the ileocolitis patients. Although no specific pathogenetic mechanisms of malabsorption were demonstrated in this study, the following contributing factors were suggested: reduction and dysfunction of the absorptive surface, lymphedema, reduced food intake, acceleration of intestinal transit, alterations in the intestinal flora, increased metabolic requirements, disturbance in bile salt metabolism, and disaccharidase deficiency.

8233 **BACILLI-LIPID ASSOCIATIONS IN WHIPPLE'S DISEASE.** *(E.)* Phillips, M. J. (Dept. Path., U. Toronto, Canada) and J. M. Finlay. *J Path Bact* 94(1):131-137, 1967.

Intestinal mucosa taken by peroral biopsies from a patient with untreated Whipple's disease and 3 control patients was studied under the electron microscope in order to identify the finely dispersed extracellular material seen in the intestinal wall and determine its possible lipid compostion. Macrophages were numerous in the lamina propria of the patient with Whipple's disease and these macrophages were found to contain phagocytic vacuoles of varying appearance, many of which contained bacilli. The bacilli, in the extracellular space, were intimately associated with an abundant deposit of a flocculent or foamy material. At higher magnification, this material is seen to consist largely of droplets of low electron density. Previous histochemical and chemical studies and the present findings support the view that the extracellular material is lipid and possibly a mixture of triglycerides and other lipids.

8234 **INTESTINAL ABSORPTION IN PROTEIN-CALORIE MALNUTRITION.** *(E.)* James, W. R. T. (Med. Res. Council, Kingston, Jamaica, West Indies). *Lancet* 1(7538):333-335, 1968.

Sugar absorption was studied by jejunal perfusion in 5 malnourished children, both on admission and after at least 2 months on a high-protein diet. All children initially had poor glucose absorption, 4 had defective lactose hydrolysis and 2 had a defect in sucrose hydrolysis. The hydrolytic defects were related to low disaccharidase activities in the jejunal mucosa. Mean transit times of the fluid in the jejunal test segment were correlated with the amount of unabsorbed sugar in the lumen. Abnormal intestinal motility was not a primary feature of the diarrhea of these children. After treatment, glucose absorption and lactose and sucrose hydrolysis all improved and disaccharidase activities increased, showing that these intestinal defects in malnutrition can be reversed.

8235 **WHIPPLE'S DISEASE (A CLINICAL AND MORPHOLOGICAL ANALYSIS OF A CASE).** *(Rus.)* Zabelinskii, E. K. (Leningrad Sanit. Hyg. Med. Inst., Acad. Med. Sci. USSR) and B. G. Lisochkin. *Ter Arkh* 40(9):112-114, 1968.

8236 **UPPER INTESTINAL TRACT INFECTION PRODUCED BY *E. ACERVULINA* AND ABSORPTION OF ^{65}Zn AND ^{131}I-LABELED OLEIC ACID.** *(E.)* Turk, D. E. (Dept. Poultry Sci., Clemson U., S. C.) and J. F. Stephe *J Nutr* 93(2):161-165, 1967.

8237 **INTESTINAL DISACCHARIDASES AND DISACCHARIDE INTOLERANCE.** *(Fr.)* Dahlqvist, A. (Dept. Physiol. Chem., U. Lund, Sweden). *Ann Biol Clin* 26(1/2):3-15, 1968.

8238 **BACTERIA, ABSORPTION AND MALABSORPTION.** *(E.)* Floch, M. H. *Amer J Clin Nutr* 20(11):1244-1248, 1967.

8239 **ABSORPTION OF DIETARY FAT: USE OF MEDIUM-CHAIN TRIGLYCERIDES IN MALABSORPTION.** *(Rev.) (E.)* Scheig, R. (Yale U. Sch. Med., New Haven, Conn.). *Amer J Clin Nutr* 21(4):300-304, 1968.

8240 **METHIONINE MALABSORPTION SYNDROME.** *(E.)* Hooft, C. (Dept. Pediat., State U., Ghent, Belgium) and I. Antener. *Bibl Paediat* 87:127-136, 1968.

8241 **GLUCOSE-GALACTOSE MALABSORPTION.** *(E.)* Lindquist, B. (Dept. Pediat., U. Lund, Sweden) and G. Meeuwisse. *Bibl Paediat* 87:92-100, 1968.

8242 **SELECTIVE MALABSORPTION OF VITAMIN B$_{12}$.** *(E.)* Visakorpi, J. K. (Child. Hosp., U. Helsinki, Finland) and U. Furuhjelm. *Bibl Paediat* 87:150-160 1968.

8243 **MALIGNANCY AND STEATORRHEA.** *(E.)* Read, A. E. (Dept. Med., U. Bristol, England). *Bibl Paediat* 87:182-190, 1968.

8244 **DISACCHARIDE MALABSORPTION.** *(E.)* Frezal, J. F. (Hosp. Dis. Child., Paris, France), J. Rey and M. Lamy. *Bibl Paediat* (87):113-120, 1968.

8245 **FAMILIAL MONOSACCHARIDE MALABSORPTION. [CASE REPORT]** *(E.)* Zetterström, R. (Dept. Pediat., Karolinska Inst., Stockholm, Sweden) and J. Waldenström. *Bibl Paediat* (87):101-112, 1968.

8246 **DISEASES ASSOCIATED WITH PROTEIN LOSING ENTEROPATHY: ELECTRON MICROSCOPIC STUDIES OF THE INTESTINAL MUCOSA.** *(E.)* Dobbins, W. O. III. (VA Hosp., Durham, N. C.). *Southern Me J* 60(10):1077-1081, 1967.

8247 **A STUDY OF PATHOLOGICAL INTESTINAL ABSORPTION.** *(Sp.)* Suarez P., M. *Bol Soc Val Ped* 10(38):121-122, 1968.

8248 **CELIAC DISEASE: CLINICAL NOTE AND THERAPY.** *(It.)* Grassi, M. (Inst. System. Med. Ther., U. Rome, Italy) and S. Spada. *Clin Ter* 47(1):85-90, 1968.

See also: 7699,7838,8201,8338,8495

49 AGANGLIONIC MEGACOLON IN INFANCY.

(E.) Márquez, E. (U. Hosp. San Juan, Puerto co) and E. Torres Reyes. *Bol Asoc Med P Rico* 60(3):118-3, 1968.

e diagnosis and management of aganglionic megacolon in ancy are discussed on the basis of clinical experience with patients (6 females and 7 males, 2-60 days old) and reports the literature. Unlike Hirschsprung's disease in older children, ere chronic constipation, enlargement of the abdomen, and lnutrition form a classical picture, aganglionosis of the in- tine in infancy does not present characteristic symptoms ex- ot for the presence of abdominal distension (12 cases) asso- ted with diarrhea (6 cases), constipation (7 cases), or vomit- (8 cases). Onset of symptoms in this series usually occurred thin the 1st week of life (1-4 days of age). Despite barium ema studies (9 cases), rectal biopsy (5 cases), and upper gas- intestinal X-rays (2 cases), 15 different diagnoses were made l in only 3 cases was the disease suspected on admission. e high mortality reported for aganglionic megacolon (70%) s not seen in this series (54%). Aganglionic megacolon ould be suspected in any infant who fails to pass stool in hr, especially if there is abdominal distension and dramatic ease of stool and gas after rectal instrumentation. X-ray dies and rectal biopsy aid in the diagnosis. As soon as the tient is in fairly good condition, a colostomy should be per- med in preparation for a Swenson's pull-through procedure.

50 CONTRIBUTION TO THE STUDY OF MEGACOLON: CASES FROM THE SÃO LUCAS NATORIUM.

(Sp.) Amiano, C. A. (São Lucas Sanatorium, o Paulo, Brazil). *Bol Sanat S Lucas* 29(8):131-144, 1968.

nong 1780 patients with diseases of the large intestine, 52 .8%) had megacolon. Most of these 52 patients (28 males d 24 females, aged 6-89 yr) were between 31 and 50 yr old th an average age of 32 yr. Most (73%) of the patients came om São Paulo State and 15% from Paraná State where Chagas' sease is endemic; 34.6% were domestic workers and 17.3% ere laborers. The most common symptoms were constipation 6 cases), colic (18 cases), fecalomas (16 cases), weight loss 6 cases), abdominal distension (15 cases), and vomiting (12 ses). Radiological examinations revealed megasigmoid alone 24 cases and in combination with dilatation of other parts the colon in 16. Megacolon was associated with megaesoph- us in 3 cases and with ulcerative colitis in 1. Examination material removed from 35 operated patients showed that 5% had hypertrophied muscles in the walls of the colon. This as associated in 3 cases with reticulosis of the lymph glands d in 1 case each with mucosal hypersecretion, hypertrophy the muscularis mucosae, and desquamation of the mucosal oithelium. Nonspecific perisigmoiditis with normal colonic alls, submucosal edema, and chronic inflammation with ir- gular dilatation of the sigmoid cavities were found in 1 case ch. Two patients had ECG changes compatible with Chagas' sease. In 2 other cases, megacolon could have either been condary to ulcerative colitis or the cause of ulcerative colitis.

51 SIGMOID VOLVULUS: CLINICAL AND SURGICAL REFLECTIONS BASED ON A STUDY OF 223 CASES.

(Por.) Nahas, P. (Fac. Med., U. São Paulo, Brazil), J. B. de Mello, A. Garrido Jr. and E. Vasconcelos. *Rev Paul Med* 72(6):273-288, 1968.

During a 15-yr period, sigmoid volvulus accounted for 25% of all intestinal obstructions in adults in São Paulo. Of 223 patients seen at the Emergency Service of the São Paulo University Hospital in 1944-60, 171 were men and 52 women; 80.8% of the women had children and 55.8% had 3 or more. Sigmoid volvulus was significantly more common among blacks and mulattoes than among Caucasians. The Machado-Guerreiro test for Chagas' disease was positive in 80% of the 75 patients on whom it was performed, and ECG tracings were abnormal in about 2/3 of the 57 patients examined. Treatment was surgical in 199 cases (89.2%) and conservative in 11 (4.9%). Thirteen patients (5.8%) who did not respond to conservative treatment were treated surgically. Decompression of the colon with an endoscopic method is currently used as conservative therapy. On surgery, 69 patients had dolichocolon; 21 had a dark fluid, a sign of vascular damage, in the peritoneal cavity; 10 had necrosis of the sigmoid, combined in 6 cases with involvement of the descending colon; and 10 had peritonitis. There were 45 operative deaths (20.1%) and 23 patients (10.3%) who survived developed complications. The cause of death was toxemia in 32 cases (13 had necrosis of the sigmoid), evisceration in 4 cases, toxemia due to necrosis of the proximal stump after abdominoperineal prolapse in 1, stercoreous peritonitis following necrotic perforation of the sigmoid in 1, mesenteric thrombosis in 1, and an embolism in the right iliac artery in 1. No autopsies were performed in 5 cases.

8252 HUMAN APPENDIX AND NEOPLASIA.

(E.) Bierman, H. R. (Inst. Cancer Blood Res., Beverly Hills, Calif.). *Cancer* 21(1):109-118, 1968.

The relation between previous appendectomy and cancer was studied by a review of the consecutive autopsy records of 1287 cases; 122 living patients with lymphomas and leukemias were also studied. Among 608 autopsy cases with cancer, 35.2% had had a previous appendectomy. Among a comparable group of 679 cases without cancer, 24% had no appendix. The appendix had been removed in 23% of 443 cases with vascular disease, 47.9% of 94 cases with cancer of the colon and rectum (significant at $P = < 0.0001$), 57.6% of 66 cases with cancer of the breast ($P = < 0.0001$), 83% of 24 cases with ovarian cancer ($P = 0.0001$), 40% of 57 patients with Hodgkin's disease, and 50% of 22 patients with granulocytic leukemia. The data suggest a systemic and local effect which is exhibited only in approximately 1/3 of the patients with cancer, lymphoma or leukemia; they also suggest that the function of the appendix may influence the induction of leukemia, lymphoma, or other neoplastic disease in a restricted susceptible population. Further investigations, including animal experimentation, will be necessary to define the nature of this association.

8253 CARCINOMA OF THE COLON SUPERIMPOSED ON CHRONIC POST-AMEBIC ULCERATION: PROBABLE MECHANISM AND INCREASED SEVERITY. REPORT ON FIVE CASES.

(Fr.) Carayon, A., R. Colomar and B. Piquard. *Med Trop (Marseille)* 27(5):511-518, 1967.

Case reports are presented for 5 patients (4 men and 1 woman, aged 47-69 yr), 3 of whom had confirmed amebiasis and had been given emetine. All of these patients were Europeans. Examination of pieces of the colon removed during surgery showed progressive chronic ulceration varying in extent and depth along with diffuse parietal hyperplasia or atrophy with the features of extensive cicatricial sclerosis. All of these lesions had developed before the tumors. One of the tumors was located in the ascending colon, another in the right 1/3 of the transverse colon, and 3 were found in the sigmoid or rectosigmoid angle. All of the tumors were partially differentiated, cylindrical, glandular carcinomas. Anterior destruction of the wall of the large intestine by amebae apparently facilitated local spreading of the tumor by extension. The tumor had metastasized into lymph nodes in 3 cases and into the ovary and fallopian tubes in 1 case.

8254 **RESPONSE TO BYPASS ILEOSTOMY IN ULCERATIVE COLITIS AND CROHN'S DISEASE OF THE COLON.** *(E.)* Kivel, R. M. (Stanford U. Sch. Med., Palo Alto, Calif.), K. B. Taylor and H. Oberhelman Jr. *Lancet* 2(7517):632-636, 1967.

Ileostomy, excluding the intestinal contents from the colon, was performed in 10 patients (5 males and 5 females, 12-58 yr old) with Crohn's disease, 5 patients (3 males and 2 females, 9-32 yr old) with ulcerative colitis, and 1 patient (female, 18 yr old) with unclassified colitis. The diagnoses were based on histological findings. The mean duration of symptoms was 28 months in both groups. Clinical recovery followed operation in the Crohn's disease group; the only relapse was in 1 of 2 patients in whom intestinal continuity was restored. In the ulcerative colitis group, relapse occurred in all 5 shortly after surgery; 4 required subsequent total colectomy and the 5th patient responded well to corticosteroids during several relapses. In the patient with unclassified colitis, abdominal pain and profuse blood and purulent discharge from the colon after ileostomy, uncontrollable by intensive medical treatment, prompted colectomy and ileorectal anastomosis 2 months later. This differential response to bypass ileostomy suggests that there is a fundamental difference between ulcerative colitis and Crohn's disease.

8255 **CHRONIC DEHYDRATION AND SODIUM DEPLETION IN PATIENTS WITH ESTABLISHED ILEOSTOMIES.** *(E.)* Clarke, A. M. (U. Otago Med. Sch., Dunedin, New Zealand), A. Chirnside, G. L. Hill, G. Pope and M. K. Stewart. *Lancet* 2(7519):740-743, 1967.

In 21 patients with established ileostomies and 21 normal comparable subjects used as controls, the total body water (based on a 3-hr equilibration period after an oral dose of deuterium oxide) and the total exchangeable sodium (measured by the method of Veall and Vetter using ^{24}Na) were determined. All the patients had undergone total colectomy more than 3 months previously for chronic ulcerative colitis (19 patients) or polyposis coli (2 patients) and were otherwise well. Compared with the control group, the ileostomy patients showed an 11% average reduction in total body water and a 7% deficit in total exchangeable sodium. The urinary excretion of water and sodium in the ileostomy patients was also significantly reduced. It is concluded that ileostomy is associated with chronic sodium depletion and dehydration.

8256 **ACUTE IRRADIATION PROCTITIS IN MAN: DEVELOPMENT OF EOSINOPHILIC CRYPT ABSCESSES.** *(E.)* Gelfand, M. D. (Yale U. Sch. Med., New Haven, Conn.), M. Tepper, L. A. Katz, H. J. Binder, R. Yesner and M. H. Floch. *Gastroenterology* 54(3):401-411, 1968.

A prospective study was carried out on the response of the rectum in 11 randomly selected patients (8 men and 3 women, 50-76 yr old) undergoing pelvic irradiation for malignant disease. Nine of these patients developed symptoms such as diarrhea, mucoid rectal discharge, and tenesmus during therapy. In half the cases, an abnormal mucosa was seen sigmoidoscopica ly, characterized by edema, duskiness, and loss of vascularity. The rectal mucosa in biopsy specimens taken during treatment was abnormal in every case; leukocytic infiltration of the lamina propria and dysplasia of the superficial and crypt epithelium with prominent tissue eosinophilia were noted. The striking feature in 9 patients was the presence of eosinophilic crypt abscesses. Histochemical studies revealed a luminal pattern of nicotinamide adenine dinucleotide phosphate dehydrogenase in 3 patients. The histological abnormalities were more severe early in the course of radiation therapy, suggesting that the bowel is capable of adapting to an injurious stimulus. One mon following therapy the mucosa had returned toward normal. It is felt that the marked tissue eosinophilia seen during irradiation reflects the nonspecificity of this response. The finding of eosinophilic crypt abscesses had not been previously described.

8257 **SQUAMOUS-CELL CARCINOMA OF THE ANAL CANAL: REVIEW OF 21 CASES.** *(E.)* Rosato, F. E. (Sch. Med., U. Pennsylvania, Philadelphia), W. Buck and E. F. Rosato. *Dis Colon Rectum* 2(3):209-212, 1968.

Among 21 patients (16 women and 5 men, 45-82 yr old) with squamous-cell carcinoma of the anal canal seen during the period 1943-66, 15 were treated by abdominoperineal resection, 3 by local excision, 2 by radiotherapy, and 1 received no treatment. Venous invasion and hepatic metastasis occurred in only 1 patient. Palpable inguinal nodes were present in 4 patients at the time of diagnosis; in 3 there was carcinoma and all died within 2 yr. In only 1 patient did inguinal metastases develop subsequently, after local excision of the primary lesion. Invasion of the rectovaginal septum was present in 6 patients; 3 are alive without evidence of recurrence. Mesenteric nodes were positive in 4 cases; 3 of these patients died less than 5 yr after surgery. Mesenteric nodes were negative in 9 patients; 6 of thes are living and well. It is felt that abdominoperineal resection with wide perineal dissection, including the posterior vaginal wall, is the indicated form of treatment. Inguinal node dissection should be performed for clinically positive nodes but not prophylactically. The similarity of symptoms and frequent concomitance of malignant and benign perianal lesions demand close supervision and prompt biopsy of suspicious areas if early detection of carcinoma in this readily accessible region is to be accomplished.

8258 **ASPARTATE TRANSCARBAMYLASE IN NORMAL AND NEOPLASTIC HUMAN COLON.** *(E.)* Smith, E. E. (Beth Israel Hosp., Boston, Mass.) and A. M. Rutenburg. *Cancer Res* 27(8):1470-1473, 1967.

Qualitative and quantitative studies were performed on crude preparations of aspartate transcarbamylase from normal and neoplastic human colon. Although the activity of the enzyme s markedly higher in neoplastic tissue, no qualitative differ- ces were observed between the enzymes from the 2 types of sue. Negative feedback inhibition of human colon aspartate anscarbamylase could not be demonstrated.

259 ASYMPTOMATIC POLYPS OF THE RECTUM AND COLON. II. FREQUENCY, SMOKING, ND ARTERIOSCLEROTIC HEART DISEASE.

(E.) Drexler, (Southern California Permanente Med. Group, Los Angeles). *rch Intern Med* 121(1):62-66, 1968.

outine proctosigmoidoscopic examination of 390 patients 09 men and 181 women, all over 45 yr old) revealed polyps 46%, including 38% of those examined before July 1, 1965 d 59% of the patients investigated after this date when greater tention was paid to minute lesions. This frequency parallels at of polyps discovered in the rectosigmoid colon by careful topsy studies, but exceeds that of polyps reported in associa- on with rectocolonic cancer. Polypoid lesions were found al- ost twice as often in cigarette smokers as in nonsmokers. tients with arteriosclerotic heart disease also had more polyps an patients without coronary artery disease, but at less than e 5% level of confidence. The incidence of polyp formation men was not significantly greater than in women. A higher eld on routine proctosigmoidoscopy may be achieved by se- cting cigarette smokers at a younger age than nonsmokers and electing all patients with arteriosclerotic heart disease for this rocedure.

8260 TRANSITIONAL CLOACOGENIC CARCINOMA OF THE ANAL CANAL: CLINICOPATHOLOGIC TUDY OF THREE HUNDRED SEVENTY-THREE CASES.

E.) Klotz, R. G. Jr. (Abington Mem. Hosp., Pa.), T. Pamukcoglu nd D. H. Souilliard. *Cancer* 20(7):1727-1745, 1967.

he clinical and pathological features of 373 transitional cloa- ogenic carcinomas are presented. This tumor accounted for .7% of 1171 anorectal neoplasms seen during this period, oc- urred twice as often in women as in men, and was most pre- alent between the ages of 60 and 70. The majority of the esions showed an identical gross appearance to that of the ommon rectal neoplasms, although a small group presented no ntraluminal lesion, infiltrated the bowel wall, and created dif- iculty with early diagnosis. A histologic classification was de- ised that appears to correlate well with prognosis. Rare pat- erns such as 'oat cell', mucoepidermoid and adenoid cystic arcinomas were recognized as variants. Lymph node metasta- is was present at surgery in 1/3 of the cases. Recurrences vere common. Metastases occurred in 19% of cases. Wide ab- lominal perineal resection produced a cure rate of 50%. Addi- ional posterior vaginectomy is recommended for anterior le- ions. The over-all cure rate was 46.7%. The average survival ime was 19.7 months without and 27.8 months with some orm of treatment.

8261 PREVALENCE AND PROGNOSIS OF INDIVIDUAL CLINICAL AND PATHOLOGIC VARIABLES SSOCIATED WITH COLORECTAL CARCINOMA.

(E.) pratt, J. S. Jr. (Ellis Fischel State Cancer Hosp., Columbia, Io.) and H. J. Spjut. *Cancer* 20(7):1976-1985, 1967.

he frequency of various clinical and pathologic variables asso- ated with colorectal carcinoma was studied in 1137 consecutive patients treated between 1940 and 1961. All patients were followed to death or to the end of 1963. All clinical, pathologic and survival data were coded for transfer to IBM cards. The frequency with which each variable occurred was tabulated and the accumulative survival rate and standard deviation of the rate were calculated through the 10th posttreatment year by the life-table method for each variable. The basic adverse determinants of host survival with colorectal carcinoma were found to be the patterns of cellular appearance or grade, including signet-ring-cell cancers, undifferentiated carcinomas (grade III), cancers complicating ulcerative colitis and unclassified malignant cancers. These accounted for 150 of the 1137 cancers (13.2%) and less than 10% of those with cancers having these characteristics survived 5 yr. When there were more than 6 lymph nodes containing metastatic cancer, less than 10% survived 5 yr, and when more than 16 nodes were involved, all died within 5 yr. The only chance for the surgical cure of high-grade colorectal cancers having a tendency to develop extensive lymph node metastasis would require wide resection of the mesenteric lymph nodes while the primary cancer is less than 3 cm (and preferably less than 2 cm) in diameter. Nonresection for any cause resulted in the death of 90-100% of hosts within 5 yr. There is a biological variant of colorectal carcinoma comprising over 54% of all resectable carcinomas that does not metastize to lymph nodes regardless of the size of the primary cancer. Thus, any large colorectal carcinoma that can be mobilized with adequate surgical margins should be resected unless there is unequivocal evidence of metastases outside of the surgical field.

8262 LOW COLORECTAL ANASTOMOSIS AFTER RESECTION FOR CANCER.

(E.) Adam, Y. G. (Albert Einstein Coll. Med., Bronx, N. Y.), H. Volk and D. State. *Surg Gynec Obstet* 125(4):1259-1264, 1967.

During the 10-yr period 1955-1965, 117 low anterior resections were performed for cancer of the rectum and rectosigmoid (57 men and 60 women, all but 1 over 50 yr of age). The over-all operative mortality rate was 11.1%. The majority of postoperative complications were related to operative technical errors. The absolute 5-yr survival rate for curative resections was 50%. Earlier diagnosis through routine proctosigmoidoscopic examination, meticulous surgical technique, and more frequent use of the diverting colostomy should improve the morbidity and mortality in this disease.

8263 SOME HISTOCHEMICAL STUDIES ON INTESTINAL EPITHELIUM WITH PARTICULAR REFERENCE TO ULCERATIVE COLITIS AND OTHER INFLAMMATORY DISEASES.

(E.) Van Noorden, S. (Yale U. Sch. Med., New Haven, Conn.), W. R. Thayer Jr., R. Yesner and H. M. Spiro. *J Path Bact* 94(1):13-18, 1967.

A total of 110 biopsies of intestinal mucosa from 25 patients with active ulcerative colitis, 10 with this disease in remission, 20 with granulomatous colitis, 4 with localized proctitis, 10 with polyps or adenomas, and 24 with various other diseases were stained by histochemical techniques to demonstrate reduced NAD- and NADP-dehydrogenases, lactate, succinate and glucose-6-phosphate dehydrogenases, monoamine oxidase and nonspecific esterase. NADP-linked enzyme activity was distributed throughout the cytoplasm of the epithelial cells in biopsies of normal mucosa. In some histologically abnormal biopsies the reaction was more intense than normal and localized to the luminal border of the epithelium. This abnormality was always found in active ulcerative colitis and was frequent in other inflammatory diseases; its severity varied with the intensity of the inflammation. This is considered to be a nonspecific response of the intestinal epithelium to inflammation, which is apparently not related to any particular disease. Inflammation is not associated with redistribution of any of the other enzymes studied in intestinal epithelial cells.

8264 **VOLVULUS OF THE SIGMOID, WITH SPECIAL REFERENCE TO THE ROLE OF GANGLION DEFICIENCY AS AN ETIOLOGICAL FACTOR.** *(E.)* Hildebrand, H. D. (Crease Clin., Essondale, British Columbia, Canada) and J. W. Wilson. *Canad J Surg* 10(4):449-454, 1967.

A total of 34 patients with sigmoid volvulus (21 men and 13 women, average age 67 yr with 68% greater than 60 yr old), including 11 with chronic brain syndrome and psychotic reaction, 12 with schizophrenia, and 1 with mental deficiency, were reviewed. Of the 34, 23 complained of pain, 24 had a history of constipation, 16 vomited during the acute episode, and 3 patients presented with intermittent diarrhea. Only 1 patient had rectal bleeding; 31 had a grossly distended abdomen, 10 with tenderness. The radiological diagnosis was correct in 31. Twelve patients had more than 1 episode, and 2 had 5 or 6, for a total of 54 episodes. Treatment with the sigmoidoscope and tube was successful in 88% of all episodes; 12 patients underwent laporatomy for detorsion. In 3 of these 12, the torsion was reduced and they were immediately prepared for elective resection. Three patients died following simple laparotomy and detorsion. The remaining 6 all had recurrence and 5 came to resection; the 6th was managed by sigmoidoscope and tube, and later died following a 3rd episode. Nineteen patients ultimately came to elective sigmoid resection or left hemicolectomy; in all, the volvulus had previously been reduced by surgical or medical means; 12 of these (63%) have had an uneventful course since. It is concluded that sigmoid volvulus is relatively common among mental patients and the aged. It is postulated that severe constipation starts a chain of events that ends in megacolon and volvulus; the diagnosis is easy if the condition is suspected.

8265 **COLOSTOMIES AND INTERNAL BYPASS OPERATIONS AS PALLIATIVE TREATMENT FOR INOPERABLE CARCINOMA OF THE LARGE INTESTINE AND RECTUM. (RESULTS AND DISCUSSION).** *(It.)* Grapulin, G. (Inst. Gen. Surg. Clin., U. Padua, Italy) and G. Rosa. *Acta Chir Ital* 23(4):435-447, 1967.

Of 193 patients with cancer of the colon and rectum, 126 (65.3%) underwent radical surgery, 60 (31%) had palliative surgery, and 7 (3.7%) had exploratory laparotomies. Palliative surgery consisted of emergency colostomies for obstruction in 10 cases, elective colostomies in 50, and intestinal bypasses in 10. None of these patients received radiation therapy or cancer chemotherapy. None of the patients subjected to emergency colostomies lived for more than 1 yr, and 5 died in the postoperative period. Follow-ups were made on 49 of the 60 patients who underwent elective surgery. Of these, 6 died immediately after surgery, 36 died during the 1st yr, 9 during the 2nd yr, and 2 in the 3rd yr. Only 1 patient survived after 4 yr. Of the 7 patients subjected to laparotomy, 1 died in the postope
tive period of cardiovascular collapse. The mean survival time
was 10.5 months, but 1 patient with hepatic metastases surviv
for 20 months and 2 patients (one with hepatic metastases, th
other with diffuse metastases into the lymph glands) survived
for 18 months. It is concluded that palliative surgery for can
of the colon and rectum does not increase the survival time.

8266 **THE ACCELERATION OF ACUTE APPENDICITIS.** *(Ger.)* Schostok, P. (Munic. Hosp., Friedrichshafen, Germany) and H. Bottcher. *Med Klin* 62(45
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An analysis of 926 patients (478 males and 448 females) witl
acute appendicitis, treated between 1960 and 1966, showed
that 67 (7%) had free perforations and 9.2% had peri- or para
appendicitis or a para-typhlitic abscess. In contrast to previous findings, most patients were between birth and 19 yr ol
(60.1%). The mean time from the onset of symptoms to hos
pitalization was 2.25 days for the entire group and 3.28 days
for patients with perforations. However, 1/3 of patients with
perforations had a history of less than 24 hr, 1/3 had an aver
age history of 2.25 days, and 1/3 had histories of 3-14 days.
Preoperative complications included 67 with free perforations
and diffuse peritonitis, 11 with paratyphlitic abscesses, 7 with
diffused peritonitis, 4 with tuberculosis or carcinoids, and 10
with Meckel's diverticulum. Relaparotomies had to be performed within 12 days after surgery in 9 cases because of surgical ileus (3 cases) or peritonitis and insufficiency of the
stump (6 cases). One patient with insufficiency of the stump
died. Although antibiotic therapy was used in 248 cases, intr
abdominal drainage had to be used in 85 cases.

8267 **THE TREATMENT OF SOME DISEASES OF THE LARGE INTESTINE WITH A DRUG HAVING A SELECTIVE ANTICHOLINERGIC ACTION ON THE COLON.** *(It.)* Abbona, C. (Inst. Gen. Med. Clin., U. Genoa, Italy), G. C. Bezante and P. L. Gatto. *Minerva Med* 58(79):3620-3623, 1967.

Clinical trials were run on 15 patients (3 men and 12 women,
aged 20-65 yr) with various spastic, dyskinetic diseases of the
large intestine, including irritable colon. Patients were given
50-75 mg/day of N-methyl-3-piperidyldiphenylglycolate methy
bromide (mepenzolate bromide) or its combination with phen
barbital for 10-40 days. Symptoms were reduced in intensity
or disappeared in all cases. The only side effect observed was
dryness of the throat which developed after 1 week of therap
and lasted until the drug was discontinued.

8268 **IS THERE AN INTESTINAL PHASE OF ACQUIRED IMMUNITY TO *ASCARIS SUUM?*** *(Ger.)* Matoff, K. (Cent. Lab. Helminthol., Sofia, Bulgaria) and A. Tersijski. *Z Tropenmed Parasit* 18(3):343-353, 1967.

In experiments on 119 white mice (80 experimental animals
and 39 controls), group 1) was given 1 oral dose of viable
Ascaris suum eggs, group 2) was given 2 oral doses of *A. suun*
eggs 12-16 days apart, and group 3) was immunized orally wi
4 doses of 15 mg of antigen from adult ascarids at 6-8 day in
tervals. All mice and a group of untreated controls were chal
lenged orally with 2000-5000 viable *A. suum* eggs 11-30 days
after their last immunization. After 17 and 48 hr animals we

ed, their small intestines and livers were examined for larvae,
l the ratio of larvae in the cecum of immunized animals to
se of the controls and the ratio of larvae in the livers of
nunized animals to those of the controls were determined.
ese values showed that a single immunization did not always
rease the resistance of the intestine to penetration by the
vae. Immunity was greater in animals which had been given
oses of eggs. Since some of the larvae penetrating into the
estinal wall die and cell infiltrates are seen around them,
s suggested that the intestinal wall takes part in the immune
cess, acting as a barrier to the larvae.

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e patients (4 males and 1 female, aged 7-33 yr) with con-
ital megacolon were treated by ablation of the distended
on and most of the aganglionic lower bowel followed by end-
end colorectal anastomosis. An abdominal approach was used
prevent trauma to the anal sphincter and urogenital tract.
o of these patients had undergone unsuccessful surgery 13
before, and 1 of these had also been treated conservatively
2 episodes of intestinal obstruction. The postoperative
rse was uncomplicated in all cases. Follow-ups, made 3-11
after surgery, showed all 5 patients had 1-2 bowel movements
ay without the use of laxatives. One patient still has residual
itis with frequent and malodorous stools, but he does not
low a proper diet. All 3 of the postpubertal males have
mal erections and ejaculations. The only female has given
th to 3 children, 1 of whom has had symptoms of congenital
gacolon since birth.

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ERISTICS CONTRIBUTING TO APPENDICITIS IN WO-
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amarca and P. Gallinaro. *Minerva Chir* 22(19):1065-1072,
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See also: 7820,7863,7917,7944,7961,8115,8218,8220,8417

59 ULCERATIVE COLITIS IN THE TROPICS. *(E.)* Chuttani, H. K. (Maulana Azad Med. Coll., w Delhi, India), S. P. Nigam, S. K. Sama, P. C. Dhanda and S. Gupta. *Brit Med J* 4(5573):204-207, 1967.

urvey of medical records from leading hospitals in different ts of India for the period 1955-60 revealed that ulcerative itis is not rare in India; the admission rate in different pitals varied from 8.3 to 89.1 cases/100,000 hospital ad-sions. A detailed study was then made of 46 cases. By nparison with Western series, the disease as seen in India nilder with fewer complications and less severe changes on noidoscopy and rectal biopsy. The disease remains limited the rectum, sigmoid colon, and descending colon in the jority of cases. Difficulties are encountered in making the gnosis of ulcerative colitis due to the high prevalence in ia of specific infections of the colon.

50 A QUANTITATIVE COMPARISON OF THE FAECAL FLORA OF PATIENTS WITH UL-RATIVE COLITIS AND THAT OF NORMAL PERSONS.) Cooke, E. M. (Westminster Med. Sch., London, England). *ath Bact* 94(2):439-444, 1967.

uantitative study of the fecal bacterial flora was undertaken 20 normal persons and 20 patients with ulcerative colitis. *e E. Coli,* lactobacilli and bacteroides counts were similar ll 40 subjects while the viable counts of *Streptococcus calis* were slightly higher in the patients with ulcerative itis. Other organisms were present in smaller numbers. No nificant differences were demonstrated in the number and es of bacteria present in the 2 groups of subjects.

1 CONTRIBUTION TO THE STUDY OF ULCERATIVE COLITIS: OBSERVATIONS AND CON-ERATIONS ON SEVERAL CASES OF THE DISEASE SUB-TED TO PROCTOCOLECTOMY. *(It.)* Ocelladore, D. t. Spec. Surg. Path., U. Milan, Italy) and A. Peracchia. *· Ital* 19(Suppl. 1):1-44, 1967.

2 ILEORECTAL ANASTOMOSIS IN COLECTOMY FOR ULCERATIVE COLITIS. *(Rus.)* Knoblokh, (Inst. Postgrad. Med., Prague, Czechoslovakia) and I. Novak. *rurgiia (Moskva)* 43(12):67-72, 1967.

3 STUDIES OF THE RELATIONS BETWEEN THE ENDOSCOPIC, HISTOLOGICAL AND RADIO-GICAL ASPECTS IN HEMORRHAGIC RECTOCOLITIS. *(Rum.)* Ban, A. (3rd Med. Clin., Cluj, Romania), L. Stanescu and G. Badea. *Med Intern (Bucur)* 19(10):1261-1266, 1967.

8364 MEISSNER'S PLEXUS IN A CASE OF HEMORRHAGIC RECTOCOLITIS. *(Fr.)* Lagarde, R. (U. Hosp. Ctr., Clermont-Ferrand, France), H. Dalle and J. Delage. *Arch Franc Mal Appar Dig* 56(10):971-976, 1967.

8365 ALTERATIONS OF THE HISTOLOGICAL AND HISTOCHEMICAL PATTERN IN ULCERATIVE COLITIS BIOPSY SPECIMENS. *(E.)* Hradsky, M. (Charles U. Med. Sch., Hradec Králové, Czechoslovakia), F. Langr, V. Nerad and V. Skaunic. *Amer J Proct* 18(5):392-398, 1967.

8366 SERUM LACTIC ACID DEHYDROGENASE ISOENZYME PATTERNS IN ULCERATIVE COLITIS. *(E.)* Hendrich, F. (J. E. Purkyne U., Brno, Czechoslovakia) and V. Hule. *Amer J Proct* 18(5):399-401, 1967.

8367 PSYCHOLOGICAL CONSIDERATIONS IN ULCERATIVE COLITIS. *(E.)* O'Connor, J. F. (Columbia U. Coll. Phys. Surg., New York, N. Y.). *New York J Med* 68(8):1055-1065, 1968.

8368 THE COLON - ULCERATIVE COLITIS. *(E.)* MacDougall, I. P. M. Pp. 146-151 in *The Prevention of Cancer.* Raven, R. W. and F. J. Roe, Eds. London, Butterworths, 1967.

8369 TOXIC DILATATION OF THE COLON IN CHRONIC ULCERATIVE COLITIS. [CASE REPORT] *(E.)* McEwan-Alvardo, G. and J. A. Bargen. *Scott & White Med* 5(1):36-40, 1968.

8370 CLINICAL AND RADIOLOGICAL DIAGNOSIS OF BLEEDING RECTOCOLITIS AND CROHN'S DISEASE OF THE COLON. *(Fr.)* Le Quintrec, Y. (Bichat Hosp., Paris, France). *Actualites Hepatogastroent* 4(5):A285-A303, 1968.

8371 ACUTE DILATATION OF THE LARGE INTESTINE IN ULCERATIVE COLITIS. *(Sp.)* Etala, E. and P. A. López. *Bull Soc Int Chir* 27(5):408-414, 1968.

See also: 7895,8254

PANCREAS

8372 **SODIUM TRANSPORT: INHIBITORY FACTOR IN SWEAT OF PATIENTS WITH CYSTIC FIBROSIS.** *(E.)* Mangos, J. A. (U. Wisconsin Med. Sch., Madison) and N. R. McSherry. *Science* 158:135-136, 1967.

When the duct system of the rat parotid gland was perfused with sweat from patients with cystic fibrosis, marked inhibition of sodium reabsorption was noted. Perfusion with sweat from normal controls caused no change in the degree of sodium reabsorption. It is concluded that a factor inhibitory to sodium transport exists in the sweat of patients with cystic fibrosis of the pancreas, which may be responsible for the increased sodium concentrations in the sweat of these patients.

8373 **ANGIOGRAPHIC STUDIES OF BENIGN AND MALIGNANT CYSTADENOMA OF THE PANCREAS.** *(E.)* Abrams, R. M. (New York U.-Bellevue Hosp. Med. Ctr., N. Y.), E. R. Beranbaum, S. L. Beranbaum and N. L. Ngo. *Radiology* 89(4):1028-1032, 1967.

Radiological findings in 4 cases of pancreatic cystadenoma, 3 benign and 1 malignant (all females, 29-83 yr old), are described. These tumors presented as large masses, showed wide displacement of arteries, and were highly vascular; 1 tumor, however, was relatively avascular in an area of cyst formation. Marked irregularity and truncation of the vessels were only seen in the malignant form. Important features in the diagnosis are avascular areas separated from, or mixed in with, areas of hypervascularity indicating the cystic elements of the tumor. Barium studies indicate the presence of a mass due to displacement phenomena, but do not give an indication of the benign or malignant nature of the lesion. Angiography not only plays a decisive role in the demonstration of the anatomic origin of the tumor, its extent and vascular nature, but may at times permit the differentiation of benign from malignant lesions.

8374 **ARTHRITIS AND SKIN LESIONS RESEMBLING ERYTHEMA NODOSUM IN PANCREATIC DISEASE.** *(E.)* Mullin, G. T. (U. Hosps., Minneapolis, Minn.), E. M. Caperton Jr., S. R. Crespin and R. C. Williams Jr. *Ann Intern Med* 68(1):75-87, 1968.

Based on 3 personal cases and a review of the literature, it is concluded that the clinical picture of subcutaneous nodules, subcutaneous fat necrosis, and acute synovitis present in some patients with pancreatitis or carcinoma of the pancreas may mimic erythema nodosum. Predominant severe polyarthritis secondary to periarticular fat necrosis or relatively frequent great-toe involvement may make this clinical symptom complex difficult to distinguish from a generalized connective tissue disorder or gouty arthritis. Eosinophilia has been noted frequently (5%-21%) when subcutaneous fat necrosis accompanies carcinoma of the pancreas. Definitive diagnosis of subcutaneous or periarticular fat necrosis by skin or soft tissue biopsy was frequently possible among the 36 patients previously reported. Unusual subcutaneous nodules often appearing in successive crops and exhibiting a diffuse distribution (buttocks, trunk, back, and even scalp), associated with signs of active synovitis, should alert the clinician to the possibility of occult or unconsidered pancreatic disease.

8375 **EVALUATION OF EXOCRINE PANCREATIC FUNCTION WITH THE SECRETIN TEST.** *(It.)* Bertero, R. (Inst. Gen. Med. Clin., U. Turin, Italy), C. Sarac G. Bruno and M. De La Pierre. *Minerva Med* 58(79):3574-3583, 1967.

The pH, volume, and bicarbonate content of pancreatic juic was determined before and after i.v. administration of secret in 27 subjects with no pancreatic abnormalities and in 69 su jects with various pancreatic diseases. In 11 patients with chronic pancreatitis the total volume of juice was reduced ir only 3 cases and the volume/kg body weight in only 2 cases but the maximum bicarbonate concentration was abnormall low in all 11 and the total quantity of bicarbonate was abnormally low in 7. Of 8 patients with pancreatic neoplasms all but 2 had reductions in the volume of pancreatic juice secreted and the volume/kg body weight, and the maximum bicarbonate concentration. The total quantity of bicarbona secreted was low in 7 cases. The total volume of pancreatic juice secreted was low in 17 and the volume/kg body weigh was low in 15 of 30 patients with diabetes mellitus. Maxim bicarbonate concentrations were low in 12 cases and total b carbonates were low in 20. The total volume of pancreatic juice was reduced in 3 of 11 patients with ulcerative hemorrhagic rectocolitis and the volume/kg body weight was eleva in 9. Maximum bicarbonate concentrations were reduced in 4 cases and elevated in 3, while total bicarbonate was norma in only 1 case. Of 8 patients with cholelithiasis, only 2 secreted normal amounts of pancreatic juice and only 3 had subnormal values for the volume/kg body weight. Maximun bicarbonate concentrations were changed in 7 and total bica bonate in 6. The total volume of pancreatic juice/kg body weight was high in all 3 patients with primary malabsorptior maximum bicarbonate concentrations were changed in 2, bu total bicarbonate was normal in all 3 cases. One patient wit candidiasis had low values for maximum bicarbonate concen trations and total bicarbonate. Reasons for these changes ar discussed.

8376 **PLEUROPULMONARY MAINFESTATIONS O PANCREATIC ORIGIN AND THEIR MEDICO SURGICAL TREATMENT.** *(Fr.)* Witz, J. P. (U. Hosp. Ctr. Strasbourg, France), G. Copin, G. Miech and A. C. Weiss. *A Chir* 21(21/22):1313-1322, 1967.

8377 **THE SUPPLY OF VITAMIN A AFTER PANCREATECTOMY.** *(Ger.)* Kasper, H. (Inst. Nu Sci., Justus Liebig U., Giessen, Germany). *Med Welt* 18(3): 178-180, 1968.

8378 **MUCOVISCIDOSIS AND CHRONIC NONSPEC FIC PNEUMONIA IN CHILDREN.** *(Rus.)* Gorodetskaia, E. G. (Kiev Inst. Postgrad. Med., USSR) and Zh. P. Gudzenko. *Pediatriia (Moskva)* 46(12):24-28, 1967.

8379 **CYSTS AND PSEUDOCYSTS OF THE PANCR AS: CLINICAL, ETIOLOGICAL AND RADIO LOGICAL ASPECTS.** *(Fr.)* Sarles, H. (Fac. Med., Marseille, France), R. Camatte, J. C. Sarles, M. Martin and C. Guien. *Actualities Hepatogastroent Hotel Dieu* 3(3):115-132, 1967.

8380 **TREATMENT OF PANCREAS CYSTS AND PSEUDOCYSTS.** *(Fr.)* Poilleux, F. (Broussais

p., Paris, France) and G. Bloch. *Actualities Hepatogastroent*
el Dieu 3(3):133-145, 1967.

31 **SURGICAL TREATMENT OF PANCREATIC PSEUDOCYSTS: REPORT OF 34 CASES.** *(Fr.)*
emon, P. (Surg. Clin., West Lille, France), C. Gautier-
oit, Y. Salembier and P. Quandalle. *Lille Chir* 12(1/2):43-
1968.

32 **CARCINOMA OF THE HEAD OF THE PANCREAS PRESENTING AS DUODENAL OB-**
UCTION WITHOUT JAUNDICE: AN UNCOMMON MAN-
STATION WITH A NOTE ON VISCERAL ANGIOGRAPHY
AN AID IN DIAGNOSIS. *(E.)* Lindenauer, S. M. (Wayne
nty Gen. Hosp., Eloise, Mich.), S. R. Reuter and R. R.
eph. *Amer J Surg* 115(5):705-708, 1968.

33 **CYSTADENOMAS AND CYSTADENOCARCINOMAS OF THE PANCREAS.** *(Fr.)* Gauchet, A.
Antoine Hosp., Paris, France), R. Girardin and M. Hivet.
Chir 21(15-16/17-18):973-982, 1967.

34 **CYSTADENOMA OF THE PANCREAS. [CASE REPORT]** *(E.)* Van Cangh, P. J. (Winnipeg Gen.
p., Manitoba, Canada) and R. H. Thorlakson. *Canad J*
11(1):63-68, 1968.

5 **AN AUTOPSY CASE OF FIBROCYSTIC DISEASE IN A 17-YEAR-OLD KOREAN GIRL.**
r.) Yu, J. Y. (Chonnam U. Med. Sch., Korea) and H. B.
. *Korean J Intern Med* 10(9):599-602, 1967.

36 **INFERTILITY IN MALE PATIENTS WITH CYSTIC FIBROSIS.** *(E.)* Denning, C. R. (Coll.
s. Surg., Columbia U., New York, N. Y.), S. C. Sommers
H. J. Quigley Jr. *J Pediat* 41(1):7-17, 1968.

37 **PORTAL HYPERTENSION IN CYSTIC FIBROSIS.** *(E.)* Tyson, K. R. T. (Child. Hosp. Med. Ctr., Boston, Mass.), S. R. Schuster and H. Shwachman. *J Pediat Surg* 3(2):271-277, 1968.

8388 **THE PANCREAS.** *(E.)* Raven, R. W. Pp. 137-140 in *The Prevention of Cancer.* Raven, R. W. and F. J. Roe, Eds. London, Butterworths, 1967.

8389 **TWO CASES OF ABERRANT PANCREAS IN THE STOMACH.** *(Jap.)* Ochi, K. (Fac. Med., Kyoto U., Japan), K. Kogawa, M. Moriga, T. Miyake and A. Miyoshi. *Jap Arch Intern Med* 14(11/12):317-326, 1967.

8390 **CYSTIC FIBROSIS IN THE AMERICAN INDIAN.** *(E.)* Harris, R. L. (Child. Mem. Hosp., Oklahoma City, Okla.) and H. D. Riley Jr. *Pediatrics* 41(4):733-738, 1968.

8391 **INVESTIGATION OF PANCREATIC SECRETION.** *(Rus.)* Mkhitarova, G. B. (North-Ossetian Med. Inst., Ordzhonikidze, USSR). *Eksp Khir Anest* 13(4): 42-43, 1968.

8392 **CARCINOMAS OF THE PANCREAS DIAGNOSED RADIOLOGICALLY AS INTRINSIC TUMORS OF THE GASTROINTESTINAL TRACT.** *(It.)* Canossi, G. C. (Inst. Radiol., U. Modena, Italy), R. Romagnoli and G. Ferolla. *Ann Radiol Diagn (Bologna)* 40(5-6):395-414, 1967.

8393 **EXTERNAL PANCREATIC INSUFFICIENCY COMBINED WITH HEMATOPOIETIC MEDULLARY INSUFFICIENCY IN THE INFANT.** *(Fr.)* Jeune, M. (Debrousse Hosp., Lyon, France), M. Hermier, D. Germain, C. Collombel, M. Mathieu, H. Duc and B. Ponson. *Pediatrie* 22(5):551-568, 1967.

8394 **AN ACCESSORY PANCREAS SITUATED WITHIN THE DUODENAL WALL.** *(Pol.)* Rawa, M. (Cent. Mining Hosp., Bytom, Poland). *Pol Przegl Chir* 40(2):139-141, 1968.

See also: 7838,7847,7848,7853,7865,7868,7880,7884,7907,7936,7937,7939,7954,7958,7970,8648,8659

8395 **FAILURE OF A POTENT TRYPSIN INHIBITOR TO MODIFY EXPERIMENTAL PANCREATITIS IN DOGS: p-AMINOBENZAMIDINE.** *(E.)* Geratz, J. D. (U. North Carolina Sch. Med., Chapel Hill). *Arch Surg* 96(3):450-457, 1968.

The effect of p-aminobenzamidine (p-ABA) on experimental pancreatitis produced by intraductal injection of trypsin was investigated in dogs. Group 1a (10 dogs) received intraductal buffered saline only, group 1b (10 dogs) received buffered saline and 2 mg trypsin/ml, group 1c (10 dogs) received buffered saline, 2 mg trypsin/ml and 10 mg p-ABA/ml; group 2a (10 dogs) received buffered saline and 5 mg trypsin/ml, group 2b (10 dogs) received buffered saline, 5 mg trypsin/ml and 10 mg p-ABA/ml in addition to systemic p-PBA; group 3a (3 dogs) received autolyzed trypsin (5 mg/ml), group 3b (3 dogs) received autocatalyzed trypsin-p-ABA, group 4 (3 dogs) received trypsinogen (10 mg/ml), group 5 (3 dogs) received trypsin (5 mg/ml) and 7.5 mg soybean trypsin inhibitor/ml, and group 6 (3 dogs) received α-chymotrypsin (5 mg/ml). Blood was removed at various time intervals before and after operation for enzyme and inhibitor assays. Autopsies were performed on all dogs by the 4th postoperative day. It was noted that moderate amounts of trypsin resulted in a significant decrease in the hematocrit level, but caused only slightly more histologic changes in the pancreas than seen in the saline-injected controls. Large amounts of trypsin led to extensive intrapancreatic hemorrhage, bloody ascites and severe hemoconcentration. In both types of trypsin pancreatitis, p-ABA failed to affect the symptoms. Autocatalytic activation of trypsinogen was found to be of no importance in experimental trypsin pancreatitis. The local and general circulatory effects of trypsin could not be blocked by p-ABA at concentrations sufficient to suppress other known activities of the enzyme.

8396 **PANCREATIC ABSCESSES AND LESSER OMENTAL SAC COLLECTIONS.** *(E.)* Bolooki, H. (Downstate Med. Ctr., New York, N. Y.), B. Jaffe and M. L. Gliedman. *Surg Gynec Obstet* 126(6):1301-1308, 1968.

The 74 patients with pancreatic abscesses and lesser omental sac collections due to acute pancreatitis seen during a 14-yr period represented about 4% of all patients treated for acute pancreatitis during that period. The overall mortality rate of 57% could be related both to the cause of the pancreatitis and to a delay in drainage procedures. Thus, the group with biliary tract disease had the highest mortality rate (85%) while alcoholics had the lowest rate (45%), and survival decreased from 71% to 25% as the period between admission and operation increased from 24-48 hr to 18 days or more. An acute septic process in patients with subsiding pancreatitis or a recurrence of pancreatitis after the onset of acute pancreatitis usually signified pancreatic abscess formation or omental sac collection. Positive bacterial cultures were obtained in 60 of 62 cases studied, and 80% of the isolated strains were sensitive to chloramphenicol.

8397 **PANCREATITIS I. REVIEW OF 733 CASES OF PANCREATITIS FROM THREE SEATTLE HOSPITALS.** *(E.)* White, T. T. (Doctors' Hosp., Seattle, Wash.), J. E. Murat and A. Morgan. *Northwest Med* 67(4):374-378, 1968.

Among the 733 cases of acute (358 cases) and chronic pancreatitis revealed by a survey of the 10-yr records of 3 Seattl hospitals, 192 acute cases had gallstones. Treatment of the biliary disease was effective in arresting the pancreatitis in most cases. Alcoholism was the concomitant factor in 106 of the 179 cases of chronic pancreatitis. Calcification occurr in 54 of these patients. Treatment consisted of pancreaticojejunostomy for the patients with dilated ducts, splanchnicectomy for those with a small, hard pancreas without ductal dilatation, and a resection where all lesser procedures failed. For the 196 patients with pancreatitis related to external or operative trauma, duodenal ulcer and related diseases, the treatment was for the accompanying disease. Long-term results are not indicated.

8398 **CHRONIC PANCREATITIS (ETIOLOGY AND DIAGNOSIS).** *(Fr.)* Dubarry, J. J., A. Quinton and J. Beylot. *J Med Bordeaux* 144(12):1837-1857, 1967.

Chronic pancreatitis was diagnosed radiologically in 54 of 10 patients (86 men and 19 women) by detection of pancreatic calcifications, and was detected at surgery in 30 additional cases. A history of biliary tract disease or one or more episodes of acute pancreatitis was found in 40% of these patient The incidence of biliary disease was 60% among women, but only 36% among men. Of the patients with histories of acut pancreatitis, 3 had recent histories of mumps. Fifteen patien had other gastrointestinal diseases (5 cases of peptic ulcer, 2 of which required gastrectomies, 2 other gastrectomies, 4 case of cirrhosis, 2 of viral hepatitis, and 2 of duodenal diverticula Infectious and parasitic diseases of possible etiological significance included tuberculosis (20 cases), amebiasis (2 cases), lambliasis (2 cases), and malaria (2 cases). Sixteen patients had vascular disorders, and in 7 cases it was difficult to distinguish between chronic pancreatitis and a gastrointestinal arteriopathy. Although none of these patients had histories of alcoholism or excessive use of tobacco, alcohol and tobacc were the only possible etiological factors found in 14 cases (16.6%). One patient had an unusual combination of pancreatic and salivary lithiasis. All but 6 patients had pain which was generally located above the umbilicus but did not necessarily predominate in the left hypochondrium. Other less common clinical symptoms included nausea and vomiting in about 30%, diarrhea in 23%, and steatorrhea in 17%. Symptoms of complications included jaundice in 21 cases (20%), glucosuria in 23 accompanied by clinical symptoms of diabete in 6, and induced hyperglycemia in 18. Complications includ 11 pseudocysts, 5 cases of gastrointestinal bleeding, 5 of pleu effusions, 1 of ascites, 1 of erythema nodosa, and 2 of pulmonary tuberculosis.

8399 **HEREDITARY PANCREATITIS: A KINDRED WITHOUT GROSS AMINOACIDURIA.** *(E.)* Davidson, P. (Ross Gen. Hosp., Calif.), D. Costanza, J. A. Swieconek and J. B. Harris. *Ann Intern Med* 68(1):88-96, 196

Among the 71 members of a 5-generation kindred with hereditary pancreatitis, data were obtained on 55, and 18 of these 5 were examined by the authors. Definite pancreatitis was foun in 6 members, and 5 had suspected pancreatitis. The mode of genetic transmission appeared to be autosomal dominant and

n-sex-linked. The disease is characterized by the onset in
ldhood of recurrent episodes of abdominal pain and is fre-
ently accompanied by pancreatic calcification, diabetes mel-
s, and steatorrhea. The present kindred and the 5 kindreds
viously reported from the Mayo Clinic differ from the kin-
d reported from France in the clinical course, the patho-
ical state of the pancreas, and the results of surgery. The
sent kindred differs from those from the Mayo Clinic in that
asurements of the urinary α-amino nitrogen indicate that
editary pancreatitis can exist in the absence of gross amino-
luria; this was also noted to be the case in the kindred from
nce. It is concluded that while the findings may be similar
he various members of a kindred with hereditary pancrea-
s, there may be differences among the kindreds.

00 **EXPERIMENTAL TRYPSIN-INDUCED PANCREATITIS PRODUCED BY SELECTIVE INTRA-
RTERIAL ADMINISTRATION OF BRADYKININ.** *(Ger.)*
össling, F. K. (Inst. Path., Johannes Gutenberg U., Mainz,
rmany), M. Nagel, J. Schier and B. Metz. *Z Gastroent*
5):306-311, 1967.

ncreatitis was induced in 2 groups of dogs by: 1) ligature
the pancreatic duct followed by i.v. administration of 5 U
olecystokinin and 20 μg bradykinin into the pancreaticoduo-
nal artery or, in 1 case, 10 U secretin i.v. and 20 μg brady-
nin i.v. or 2) ligature of the pancreatic duct, instillation of
ml physiological saline into the pancreatic duct under a
essure of 15 cm H_2O, administration of 10 U cholecysto-
nin i.v. followed by 50 μg bradykinin into the pancreatico-
odenal artery. The only gross changes in the pancreas in
gs belonging to group (1) were bleeding in the body, parti-
larly near the mouth of the pancreatic duct, and mild edema-
us swelling. Edema was more severe and small areas of
crosis were present in fatty tissue in the animal receiving
cretin and bradykinin i.v. Histological changes included signs
circulatory disturbances, but little evidence of secretory ob-
uction or lesions in the duct system. Only a few animals
d very small areas of necrosis in the epithelium of the ex-
etory glands. Gross changes in group (2) included edema
d bleeding localized to the body of the pancreas. Only 2
imals had extensive areas of necrosis in the fatty tissue.
stological changes were typical of those found in hemorrhagic
d necrotic pancreatitis with trypsin- or autodigestion.

01 **POSTOPERATIVE PANCREATITIS.** *(Ger.)* Lorenc, J. (Med. Fac., Charles U., Prague,
echoslovakia). *Zbl Chir* 92(26a):1468-1472, 1967.

02 **SUBACUTE PANCREATITIS WITH WEBER-CHRISTIAN SYNDROME, VASCULAR AND
TEO-ARTICULAR MANIFESTATIONS: REPORT OF A
SE.** *(Fr.)* Levrat, R. (Red Cross Hosp. Lyon, France), F.
rthezène, J. P. Porta, A. Bel and M. Girard. *Arch Franc Mal
par Dig* 57(3):345-358, 1968.

03 **RECENT EXPERIENCE WITH SURGICAL EXCISION FOR CHRONIC PANCREATITIS.** *(Fr.)*
utier-Benoit, C. (Surg. Clin. West, Lille, France), Y. Salembier,
Quandalle, G. Derasse and P. Razemon. *Lille Chir* 23(1/2):
28, 1968.

8404 **THE TREATMENT OF ACUTE PANCREATITIS WITH TRASYLOL.** *(Rus.)* Ryneiskii, S. V. (N. I. Pirogov 2nd Moscow Med. Inst., USSR), P. I. Ostrin, M. I. Filimonov and B. F. Khovanskii. *Khirurgiia (Moskva)* 44(3): 115-119, 1968.

8405 **BILIARY PANCREATITIS: EXPERIENCE AT THE SURGICAL DEPARTMENT OF THE FIRST MUNICIPAL HOSPITAL IN SOFIA.** *(Bul.)* Lozanov, L. (1st Munic. Hosp., Sofia, Bulgaria). *Khirurgiia (Sofiia)* 20(5): 418-422, 1967.

8406 **ANTIENZYMES IN THE TREATMENT OF PANCREATITIS.** *(Rus.)* Vashkialis, V. (Kaunas Med. Inst., USSR). *Sovet Med* 31(6):74-78, 1968.

8407 **CONSERVATIVE THERAPY OF ACUTE PANCREATITIS.** *(Rus.)* Kochnev, O. S. (Kazan Inst. Postgrad. Med., USSR). *Sovet Med* 31(6):68-74, 1968.

8408 **ACUTE HEMORRHAGIC PANCREATITIS.** *(Fr.)* Gratadour, M. P. *J Med Bordeaux* 144(9):1336-1341, 1967.

8409 **MEDICAL TREATMENT OF ACUTE PANCREATITIS.** *(Fr.)* Paccalin, M. J. *J Med Bordeaux* 144(9):1332-1335, 1967.

8410 **PANCREATITIS.** *(Fr.)* Traissac, F. J. *J Med Bordeaux* 144(9):1329-1332, 1967.

8411 **EVOLUTION OF PANCREATITIS.** *(Fr.)* Moretti, G. *J Med Bordeaux* 144(9):1323-1327, 1967.

8412 **ENZYMATIC PROBLEMS AND ANTI-ENZYMATIC TREATMENT OF ACUTE HEMORRHAGIC PANCREATITIS.** *(It.)* Priola, I. (Maria Vittoria Hosp., Turin, Italy). *Minerva Gastroent* 13(4):145-152, 1967.

8413 **TOTAL PANCREATECTOMY FOR DIFFUSE CHRONIC PANCREATITIS.** *(Fr.)* Ryncki, P. V. ("La Source" Clin., Lausanne, Switzerland) and B. Curchod. *Gastroenterologia (Basel)* 108(1):15-30, 1967.

8414 **PANCREATIC NECROSIS WITH GAS FORMATION. [CASE REPORT]** *(E.)* Schwartz, A. (Rothschild Hadassah U. Hosp., Jerusalem, Israel) and D. Birnbaum. *Amer J Proct* 18(6):459-463, 1967.

8415 **THE ROLE OF LYMPH AND BLOOD CIRCULATION IN PANCREATIC SELF-DIGESTION AND THE DEVELOPMENT OF EXTRAPANCREATIC NECROSES.** *(E.)* Papp, M. (Inst. Exp. Med., Hungarian Acad. Sci., Budapest), S. Ormai, L. Csaki and E. Horvath. *Acta Med Acad Sci Hung* 25(3/4):433-439, 1968.

8416 **SEMIOLOGY AND DIAGNOSIS OF ACUTE PANCREATITIS.** *(Fr.)* Vayre, P. (Vaugirard Hosp., Paris, France). *Hopital* 56(767):157-164, 1968.

8417 **A RARE COMPLICATION OF ACUTE PANCREATITIS: ASCITES AND LARGE INTESTINE FISTULATION.** *(It.)* Zotti, E. F. (Munic. Hosp., Padua, Italy), M. Godina and G. F. Fasolo. *Acta Chir Ital* 23(6):683-694, 1967.

8418 **ACUTE PANCREATITIS IN A 9-YEAR-OLD GIRL.** *(Pol.)* Moczurad, B. (Wito Hanki Munic. Children's Hosp., Chorzów, Poland). *Pol Przegl Chir* 39(11): 1228-1230, 1967.

8419 **TREATMENT AND PROGNOSIS IN ACUTE PANCREATITIS.** *(Fr.)* Vayre, P. (Vaugirard Hosp., Paris, France) and J. Hureau. *Hopital* 56(787):181-1 1968.

8420 **DUODENAL HEMATOMA IN ACUTE PANCR ATITIS.** *(Fr.)* Warter, J. (Hosp. Cent., U. Strasbourg, France), L. Asch, J. L. Imbs and C. Monath. *Ar Mal Appar Dig* 56(12):1233-1242, 1967.

8421 **LIGATION OF PANCREATIC DUCTS.** *(Pol.)* Ziarek, S. (Silesian Acad. Med. Zabrze, Poland *Pol Przegl Chir* 40(2):125-128, 1968.

See also: 7836,7843,7930

LIVER AND BILIARY TRACT

GENERAL

2 **GLUCOSE TOLERANCE AND DIABETES IN
CHRONIC LIVER DISEASE.** *(E.)* Megyesi, C.
yal Free Hosp., London, England), E. Samols and V. Marks.
cet 2(7525):1051-1056, 1967.

28 unselected patients (18 males and 10 females, 12-65 yr
average 41) with well-investigated chronic liver disease
ive chronic hepatitis, alcoholic cirrhosis, biliary cirrhosis,
ptogenic cirrhosis, cholestatic hepatitis, Wilson's disease) con-
ied by liver biopsy, 32% had definite diabetes mellitus, 25%
impaired glucose tolerance, and 43% had normal glucose
rance. Diabetes occurred with all types of chronic liver
ase and could not be correlated with jaundice, ascites, or
vious administration of diabetogenic drugs or diuretics. It
elt that "hepatogenous diabetes" is a nosological entity
ch is insufficiently recognized in the English literature. It
ostulated that chronic liver disease produces endogenous
ilin resistance and hyperinsulinemia before glucose tolerance
npaired, and that diabetes occurs when β-cell decompensa-
occurs. The cause of insulin resistance and the eventual
ll decompensation remains unknown, and the best form of
tment remains undecided.

3 **HETEROTOPIC LIVER TRANSPLANTATION
UTILIZING INBRED RAT STRAINS. I.
ARACTERIZATION OF ALLOGENEIC GRAFT REJEC-
N AND THE EFFECTS OF BILIARY OBSTRUCTION
D PORTAL VEIN CIRCULATION ON LIVER REGENERA-
N.** *(E.)* Lee, S. (Scripps Clin. Res. Found., La Jolla, Calif.)
T. S. Edgington. *Amer J Path* 52(3):649-669, 1968.

otal of 176 auxiliary heterotopic liver transplants were em-
ved to study the immunohistochemical and histopathologi-
features of allogeneic liver rejection. Male Lewis rats were
as both recipients and donors for syngeneic liver grafts
male Sprague-Dawley rats were used as donors of allogeneic
transplants. Both transplant and host livers were reduced
0% of their normal mass by partial hepatectomy, and bile
ts of donor livers were uniformly ligated. Two types of
transplants were used: in 1 type, only the hepatic artery
plied the afferent circulation, while the vascular supply and
drainage of the host liver were not modified. In the other
e, both hepatic artery and portal vein afferent circulation
e reestablished and the host liver was deprived of portal
blood flow. Hepatic vein drainage into the vena cava was
tical in both types. There was a total cumulative mortality
2%; 28 recipients (15.9%) died during the initial 24-hr post-
rative period. The dependence of liver regeneration on
al vein blood flow was affirmed both in the presence and
he absence of biliary obstruction. Biliary obstruction *per*
ad little effect on the gross regenerative capacity following
atectomy, but did elicit a consistent proliferative response
he cholangiolar level. A highly reproducible and temporally
ise sequence of histopathologic changes was observed during
geneic rejection, in contrast to previous studies. Four dis-
t phases of allogeneic rejection were noted: phase I, a lag
se, the duration of which depended on histocompatibility
erences between donor and recipient; phase II, the appear-
e, after 1-3 days, of a mononuclear cell infiltrate in the
al areas in the absence of hepatocellular injury; phase III, the appearance of cells of the lymphocytic or monocytic series in the hepatic sinusoids associated with hepatocellular injury; and phase IV, progressive fulminant necrosis during the height of the rejection phenomena. Further study of this model may permit a more detailed investigation of the many physiologic and pathogenetic factors involved in allogeneic liver rejection.

8424 **SIDE EFFECTS OF STEROID TREATMENT IN PATIENTS WITH HEPATITIS.** *(Ger.)* Pabst, K. (2nd Med. Clin., U. Mainz, Germany), U. Theile and F. Orestano. *Therapiewoche* 17(47):1917-1919, 1967.

Of 164 patients hospitalized for acute and chronic hepatitis, 70 were treated with steroids. Patients selected for steroid therapy were those in poor general condition with SGPT levels of 60 U/liter or more and serum bilirubin values of more than 10 mg%. Those with acute hepatitis were given an initial dose of 60-75 mg/day of prednisolone or methylprednisolone. This dose was increased by 5 mg every 2 days after the first 5 days, and treatment was rarely continued for more than 5 weeks. Most patients with chronic hepatitis and hepatic necrosis received 50 mg/day of prednisolone. Since dosages were increased more slowly the duration of treatment averaged 8 weeks. Side effects included steroid diabetes (21 cases), a Cushing-like syndrome (13 cases), acne (8 cases), potassium deficiency (7 cases), withdrawal psychosis (4 cases), and a bleeding peptic ulcer. Although the percentage of side effects in this series of patients was high, complications were usually mild. Only a few patients who developed diabetes required oral antidiabetics or insulin, and blood sugar levels returned to normal when steroid therapy was discontinued. Serum potassium values returned to normal and clinical signs of hypokalemia disappeared within 8 days when patients were given 84 mg K/day. The bleeding ulcer responded to conservative treatment. It is concluded that the number and severity of side effects produced by steroid therapy can be reduced by using steroids only where indicated, carefully following the patients, and using appropriate prophylactic measures.

8425 **WILSON'S DISEASE IN INDIA. II. BIOCHEMICAL AND PATHOGENETIC CONSIDERATIONS IN PATIENTS, PARENTS AND SIBLINGS.** *(E.)* Manghani, D. K. (Sir J. J. Group Hosp., Bombay, India) and D. K. Dastur. *Neurology* 19(2):117-126, 1968.

Parameters of copper metabolism and urinary amino acid, calcium, phosphate and uric acid levels were studied in all or most of 23 patients with Wilson's disease (20 with hepatomegaly and 8 with osseomuscular changes) in 25 of their parents or siblings, and in 15-20 normal subjects or patients with nonmetabolic disorders. An abnormally low copper oxidase value was found in all patients with Wilson's disease (0.040 ± 0.018 extinction coefficient units (ECU) compared with both the controls (0.508 ± 0.139 ECU) and the parent-sib group (0.369 ± 0.153 ECU). The total serum copper level was significantly reduced in the patient group as a whole (147.59 ± 38.56 μg/100 ml) compared to the normal controls (180.3 ± 13.09 μg/100 ml), but was not significantly different from that in the parents and sibs (161.70 ± 33.54 μg/100 ml). The patient group differed significantly with respect to direct-reacting Cu (56.32 ± 16.69 μg/100 ml) from the control (15.17 ± 9.9 μg/100 ml) and parent-sib (16.09 ± 13.03 μg/100 ml) groups. For individual cases, an expression of the direct-reacting copper in terms of % of the total copper rather than as the actual value appeared to be more accurate. The 3 groups also differed significantly from each other with regard to indirect-reacting copper (90.50 ± 37.66, 167.63 ± 11.0 and 142.91 ± 23.59 μg/100 ml, resp.). There was a 7-fold increase of urinary copper excretion in the patient group (71.00 ± 54.13 μg/24 hr) compared with the control group (11.09 ± 11.77 μg/24 hr). Urinary copper excretion was 29.41 ± 28.25 μg/24 hr in the parent-sib group. All but 1 patient with Wilson's disease showed severe to massive (3-7 times normal) amino aciduria, with an 8- to 10-fold increase in threonine and glutamic acid, and 4- to 5-fold increases in serine, glutamine, citrulline and phenylalanine. Urinary calcium and phosphate excretion were elevated in the patient group (138.4 ± 86.16 mg/24 hr and 1.19 ± 0.56 g/24 hr, resp.) compared with the controls (24.95 ± 14.15 mg/24 hr and 0.94 ± 0.33 g/24 hr, resp.). Uric acid excretion was also higher in the patients (242.50 ± 241.6 mg/24 hr) than in the control group (172.56 ± 144.7 mg/24 hr). The 8 patients with osseomuscular changes differed from the other patients with Wilson's disease only in the significantly lower direct-reacting copper value (48.24 ± 20.64 and 61.71 ± 11.15 μg/100 ml, resp.). There was also a trend toward increased calcium and reduced phosphate excretion in this subgroup. In addition, a qualitative test for cystine was strongly positive in 4 of these 8 patients and in only 2 of 11 of the remaining subjects with Wilson's disease. Parents and sibs were comparable to the normal subjects in most determinations, significant differences being found only for copper oxidase levels. The patients and the parent-sib groups differed significantly with respect to serum copper oxidase, direct-reacting copper and urinary copper.

8426

CHOLESTYRAMINE THERAPY IN PATIENTS WITH A PAUCITY OF INTRAHEPATIC BILE DUCTS.

(E.) Sharp, H. L. (U. Minnesota Sch. Med., Minneapolis), J. B. Carey Jr., J. G. White and W. Krivit. *J Pediat* 71(5):723-736, 1967.

Five patients (3 males and 2 females, 5-23 yr old) with a deficiency of intrahepatic bile ducts were examined before and after cholestyramine therapy (6-24 g/day) to determine whether treatment with this anion-exchange resin would improve the pruritus associated with this anomaly. Elevated serum bile acid levels, with a trihydroxy-dihydroxy ratio greater than 1, were found in all 5, indicating obstructive liver disease. The dosages given to the 2 older patients had to be increased and 2-3 months were required before satisfactory clinical results were demonstrated. However, pruritus was eventually relieved, both direct and indirect serum bilirubin values returned to the normal range, liver function tests reverted toward normal, and there were dramatic increases in height and weight. In the remaining 3 patients, cholestyramine provided symptomatic relief, reduction in serum bile acids and some evidence of improvement in liver function; these 3 patients did not continue with the prescribed amount of cholestyramine which is necessary to obtain normal serum bile acid levels. Case histories of the 5 patients are contained in an addendum. It is concluded that bile acids may have a deleterious effect on hepatic metabolism.

8427

CLINICAL AND EXPERIMENTAL STUDIES ON THE RHEUMATOID-LIKE FACTOR IN CHRONIC LIVER DISEASE.

(E.) Tabe, K. (Osaka U. Med. Sch., Japan). *J Osaka City Med C* 16(5/6):387-406, 1967.

A total of 110 cases of liver disease, including 9 cases of fatty liver, 5 cases of liver cancer, 8 cases of acute hepatitis, 45 case of chronic hepatitis and 43 cases of cirrhosis, were subjected t peritoneoscopy, needle biopsy, liver function tests and the RA test for rheumatoid-like agglutinins. The results demonstrated that 67% of those with cirrhosis and 24% of those with chroni hepatitis had positive values on the RA-test. The RA-test wen from positive to negative in 6 of 10 cases of cirrhosis followin administration of corticosteroids for more than 5 months. Although the RA-test was positive in 6 cases of chronic hepatitis and 10 cases of cirrhosis, the thymol turbidity and zinc sulfate tests were within the normal range in these patients. When immunofluorescent studies were carried out on the livers of rats with experimental hepatic fibrosis induced by sensitizatior to egg yolk, FITC-labeled anti-rat γ-globulin was located at the site of infiltration of round cells, plasma cell-like cells or lymphoid cells. In addition, the rheumatoid-like factor was detect in the sera of most animals sensitized repeatedly with egg yolk for 4-6 weeks.

8428

THE INCIDENCE OF HEPATIC CIRRHOSIS AND CARCINOMA IN AUTOPSIED PATIENTS

(Ger.) Wöckel, W. (Inst. Path., Med. Acad. Erfurt, Germany) and H. Altrock. *Deutsch Gesundh* 23(4):165-169, 1968.

In a 10-yr period, autopsies were performed on 15,568 patient of all ages (8751 males and 6815 females), including 4004 on infants and children. Cirrhosis or chronic progressive heaptitis was found in 458 cases (308 males and 150 females) or 2.94% of the total; 453 of these subjects were over 15 yr old (3.92% of all adults). In both men and women cirrhosis was most common in the 6th, 7th, and 8th decades. Cirrhosis was foun fortuitously at autopsy in 64 of the 458 subjects (14.0%). Cirrhosis and its complications caused death in 235 cases (51. Of the 218 patients (47.6%) with esophageal varices, 61 (13.3 died of bleeding. Splenic hyperplasia was present in 368 cases (80.35%), ascites in 246 (53.7%), and generalized jaundice in 165 (36.0%). In the same period, 40 autopsied patients (0.26 had primary liver carcinomas and 2 had primary liver sarcomas Liver carcinoma was found in 24 men and 16 women for a sex ratio of 1.2:1. Liver carcinoma occurred most often in th 7th decade in men and in the 5th and 6th decades in women. In 11 of the 40 cases (27.5%) cirrhosis was the underlying disease. These patients made up 2.4% of all patients with cirrhosis. In 50 of the 458 cirrhotics (10.9%) primary tumors were found in sites other than the liver. These included 49 carcinomas and 1 sarcoma. Only 11 of the cirrhotics (2.4%) had liver metastases. These findings are compared with others in the literature.

8429

MICROEMBOLIC PULMONARY HYPERTENSIO IN PYOGENIC CHOLANGITIS.

(E.) Lai, K. S. (Queen Mary Hosp., Hong Kong), A. J. S. McFadzean and R. Yeung. *Brit Med J* 1(1):22-24, 1968.

a consecutive series of 728 patients with recurrent pyogenic olangitis, 15 (2.1%) developed pulmonary hypertension. How- er, the incidence among those with a history of frequent acute acerbations for more than 8 yr was 7.6%. At laparotomy, iich antedated the development of pulmonary hypertension 14, all were found to have greatly scarred livers and all had ones in the intrahepatic and extrahepatic ducts. The findings necropsy on 3 of the 15 patients indicated that the pulmo- ry hypertension may have been due to repeated pulmonary croembolism. It is concluded that the emboli had their ori- in vessels within the liver, and it is suggested that the ves- s concerned were the portal-hepatic venous shunts within rs. The portal components of these shunts have been shown be derived mainly from the peribiliary plexus, and the shunts mmonly show the histological changes of acute pylephlebitis.

30 ELECTRON MICROSCOPIC STUDIES IN BILIARY ATRESIA. I. BILE DUCTULAR PROLIFERA-)N. *(E.)* Hollander, M. (Mount Sinai Sch. Med., New York, Y.) and F. Schaffner. *Amer J Dis Child* 116(1):49-56, 1968.

ctular cell proliferation in liver biopsies from 7 patients with rahepatic biliary atresia was examined under the electron croscope in order to determine the nature of these prolifera- cells in the absence of bile flow and to investigate the ultra- uctural alteration which may be responsible for this type of ary cirrhosis. The marked accumulation of material noted hin the ductular cells suggests that the hepatocytes are se- ting bile into their canaliculi, which is then resorbed by the tular cells. The periductular inflammatory reaction noted hought to indicate leakage of an irritating substance that may the growth stimulant for ductular proliferation and may be ponsible for the fibrogenesis leading to cirrhosis.

31 ELECTRON MICROSCOPIC STUDIES IN BILIARY ATRESIA. II. HEPATOCELLULAR ALTERA-)NS. *(E.)* Hollander, M. (Mount Sinai Sch. Med., New York, Y.) and F. Schaffner. *Amer J Dis Child* 116(1):57-65, 1968.

electron-microscopic study of the parenchymal cells in extra- patic biliary atresia was undertaken to determine the extent parenchymal cell impairment, especially of organelles associ- d with biliary excretion. Liver biopsy specimens were ob- ned from 6 children with biliary atresia; 4 patients were 7-9 eks old and 2 patients were 12-13 months old. Diagnosis s proven at laparotomy or autopsy. In early congenital bili- atresia (4 younger patients) the hepatocytes were essentially rmal; however, later in the disease (2 older patients), organelle erations associated with hepatic insufficiency were noted. e biliary secretory apparatus appeared intact and it is suggested it regurgitation of bile does not occur at the heaptocellular el, but distal to the canaliculi at the level of the ductules. is supports the view that the parenchymal cells in biliary esia excrete bile.

32 TREATMENT OF CHRONIC INTRAHEPATIC CHOLESTASIS WITH PHENOBARBITONE. (E.) iompson. R. P. H. (King's Coll. Hosp. Med. Sch., London, gland) and R. Williams. *Lancet* 2(7517):646-648, 1967.

ur patients (all females, 45-67 yr old) with proven chronic in- hepatic cholestasis were treated with phenobarbitone (90-180 /day) for periods of 1-2 months. Two of the patients had primary biliary cirrhosis, 1 had post-necrotic cirrhosis and cholestasis, and 1 had sarcoidosis and primary biliary cirrhosis. All patients had generalized pruritus and jaundice. Routine liver-function tests were performed at frequent intervals. In each patient, the plasma bilirubin fell to 60% of the initial level by the 10th to the 14th day of treatment; in 1 patient, the bilirbuin level rose when treatment was discontinued and fell again when treatment was reinstituted. All patients noticed a striking decrease in pruritus and an improvement in general well-being; subjective improvement in the degree of jaundice was noted. All patients were noted to become drowsy and lethargic, but this was controlled by reducing the dose to 60 mg twice daily in 2 patients. The mechanism of the reduction in plasma bilirubin by phenobarbitone may be related to enzyme induction.

8433 OBSERVATIONS ON LIVER REGENERATION AFTER MAJOR HEPATECTOMIES PERFORMED FOR MALIGNANT TUMORS. *(It.)* Ruggieri, F. (Santo Spirito Charitable Inst., Rome, Italy) and R. Maira. *Epatologia* 13(3):717-728, 1967.

No evidence of compensatory hypertrophy was found before or during surgery in 11 patients who were subjected to removal of the left lobe of the liver or total hepatectomy on the left because of malignant neoplasms. Follow-ups of these patients with liver scinitigraphy and splenoportography showed that relatively little compensatory hypertrophy developed after surgery although, in some cases, liver function tests became normal. Regeneration appeared to be less extensive than that seen in several patients after surgery for removal of hydatid cysts. Illustrative case reports are presented.

8434 ANOMALIES OF THE LIVER AND BILIARY TRACT IN THE ADULT PATIENT. *(E.)* Morrell, J. F. (Northwest. U. Med. Sch., Chicago, Ill.), M. G. Govostis and M. C. Anderson. *Bull Soc Int Chir* 27(3):236-245, 1968.

Seven case histories are presented to demonstrate that the rare occurrence of anomalies of the liver and extrahepatic bile ducts in the adult may simulate symptoms produced by more conventional diseases and may play a role in the pathogenesis of biliary and/or pancreatic inflammation. Radiologic examination in a 39-yr-old female who complained of abdominal fullness and epigastric and back pain revealed compression of the stomach and displacement of the right kidney by a mass consistent with an enlarged liver; at laparotomy a large solitary cyst, almost completely replacing the left lobe of the liver and extending over the superior surface of the right lobe, was found. A choledochal cyst in a 17-yr-old female with normal gallbladder and intrahepatic biliary radicals was treated successfully by cholecystectomy and Roux-en-Y choledochojejunostomy. Cholecystography revealed nonvisualization of the gallbladder in a 30-yr-old female with intermittent right upper abdominal pain of 4 years duration; at operation the gallbladder was found to be absent. Subsequent to cholecystectomy, a 38-yr-old female presented with nausea, vomiting, epigastric pain and abdominal tenderness; at operation a cystic duct remnant was observed which communicated directly with a duct that coursed along the gallbladder bed to the free edge of the liver. X-ray studies in a 55-yr-old female with a 6-yr history of intermittent jaundice diagnosed as chronic biliary cirrhosis revealed a periampullary duodenal diverticulum and an extrinsic mass which produced partial obstruction of the 3rd portion of the duodenum; a preoperative percutaneous cholangiogram demonstrated an enormous dilation of the extrahepatic biliary tract. At operation the bile and pancreatic ducts were found to enter the duodenal diverticulum and a carcinoma of the pancreas (not involving the ampullary area) was observed. In a 58-yr-old female with a periampullary duodenal diverticulum, the terminal end of the common duct avulsed from its site of entry into the diverticulum and the papilla of the pancreatic duct also entered the diverticulum. In a 68-yr-old male, pancreatic inflammation and obstruction of the common bile duct and duodenum were associated with a periampullary duodenal diverticulum.

8435 **THE USE OF COMBINED PRESERVATION TECHNIQUES FOR EXTENDED STORAGE OF ORTHOTOPIC LIVER HOMOGRAFTS.** *(E.)* Brettschneider, L. (U. Colorado Sch. Med., Denver), P. M. Daloze, C. Huguet, K. A. Porter, C. G. Groth, N. Kashiwagi, D. E. Hutchinson and T. E. Starzl. *Surg Gynec Obstet* 126(2):263-273, 1968.

Orthotopic liver transplantation was performed with homografts that had been preserved for 8-25 hr after their removal from nonrelated mongrel donors. The fate of the recipients was compared to that of control dogs which received homografts transferred as rapidly as possible from cooled living donors. All dogs were treated with immunosuppressive agents postoperatively. Homografts could be preserved effectively for 8 hr and often for as long as 24 hr with a combination of hypothermia at 4C, hyperbaric oxygenation at 3.5-4.0 atmospheres, and divided perfusion with diluted homologous blood through the portal vein and hepatic artery at a total rate of 6 ml/g tissue/hr. At the end of the conservation period, the chamber was decompressed during 3-4 hr. The short-term and long-term course of 5 recipients of livers preserved for 8 hr was no different from that of controls. All dogs survived the operation, and only 1 dog died in less than 19 days. Three of 5 recipients of livers preserved for 24 hr survived the operation and 1 has lived at least 4 months. Deviations from this technique were evaluated with additional groups of 3-5 experiments in which livers were stored for 24 hr. Omission of perfusion or reduction of its rate by half, elimination of hyperbaric oxygenation, rapid chamber decompression, or failure to add homologous blood to the perfusate all resulted in less satisfactory results. The recipients of these homografts either died during operation or shortly thereafter. They usually had severe clotting abnormalities, biochemical evidence of severe hepatocellular injury, retardation of protein synthesis, and a high incidence of gastrointestinal ulceration. Histological studies showed evidence of ischemic necrosis. A significant rate of homograft survival can be attained with attention to these several important details.

8436 **FAMILIAL FORM OF BENIGN IDIOPATHIC RECURRENT CHOLESTASIS.** *(E.)* Goldberg, D. M. (Western Inf., Glasgow, Scotland) and E. B. Hendry. *Arch Intern Med* 120(5):556-564, 1967.

A family of 3 brothers with recurrent attacks of obstructive jaundice is described based on intensive investigations during 5 episodes of jaundice in 2 of these patients. No etiological factor could be found. Jaundice usually followed an influenzal illness and was accompanied by hepatomegaly and preceded by a 1-3 week interval of intense pruritus. Elevated values for serum bilirubin and serum alkaline phosphatase were noted, but other serum enzymes were unchanged, suggesting minimal liver cell damage. Complete recovery took place after each episode. Cholestyramine (12 g/day) relieved pruritus. Jaundic cleared on 2 occasions shortly after starting treatment, and pe sisted but at a lower level on a 3rd occasion. It is felt that an incipient attack may have been prevented by the drug. It is suggested that these 3 cases represent a familial form of idiopathic recurrent cholestasis, and that inheritance may be by a sex-linked recessive gene.

8437 **EXPERIMENTAL EVALUATION OF LIVER ATROPHY AND PORTACAVAL SHUNT.** *(E.)* Fisher, B. (U. Pittsburgh Sch. Med., Pa.), E. R. Fisher and S. Lee. *Surg Gynec Obstet* 125(4):1253-1258, 1967.

Studies of auxiliary liver transplantation have revived the concept that portal blood contains specific nutrients essential for the maintenance of the morphologic and functional integrity of hepatic tissue. In the present study, the venous outflow to a portion of the liver in rats with end-to-side portacaval shunts was altered by hepatic vein constriction. While the rest of the organ markedly decreased in size, the lobe with ou flow impairment maintained its normal structure and composition. These findings suggest that modification of sinusoidal pressures may be a prime determinant of the atrophy which follows deviation of the portal blood supply, in agreement wit earlier reports. These studies also minimize the primacy of po tal blood for inhibition of hepatic atrophy. This study substantiates the conclusion that the diminution in liver size after portacaval shunt in normal and cirrhotic rats is not only the r sult of parenchymal cellular atrophy, but is also due to a decrease in cell numbers.

8438 **DIRECT EXCHANGE BLOOD TRANSFUSION IN TREATMENT OF HEPATIC COMA.** *(E.)* McLean, J. A. (Alfred Hosp., Melbourne, Australia) and H. A. Luke. *Brit Med J* 4(5571):78-80, 1967.

The technique of exchange blood transfusion by the direct method and experiences with 4 patients are described. In this technique, 2 transfusion pumps are rotated simultaneously to give blood from donors and remove blood from the patient. Polyvinyl tubing and Teflon catheters are used to lessen the ri of clot formation. In 4 patients (2 men and 2 women, 31-61 yr old) suffering from hepatic coma, this method of treatment produced improved blood values and 1 patient recovered. It is postulated that unmodified blood transfusion has a place in the treatement of liver failure, particularly when associated wi severe bleeding.

8439 **PROLONGED SURVIVAL OF LIVER TRANSPLANTS IN THE PIG.** *(E.)* Calne, R. Y. (Dept. Surg. Path., U. Cambridge, England), H. J. O. White, D. E. Yoffa, R. M. Binns, R. R. Maginn, R. M. Herbertson, P. R. Millard, V. P. Molina and D. R. Davis. *Brit Med J* 4(5580): 645-648, 1967.

Initial experiments with orthotopic liver transplantation in the pig showed that this procedure is followed by relatively mild rejection phenomena; 6 of 19 pigs lived a week or more after transplantation. Further experience has shown that vagotomy plus gastroduodenostomy appears to prevent the previous hig incidence of gastrointestinal hemorrhage. Liver transplants wi out immunosuppression consistently survived for long periods

ime in immunologically mature animals even when the do-
and recipient were from widely disparate genetic sources.
behavior of the liver was in marked contrast to kidney and
transplants, which were rapidly rejected in similar pigs. Of
pigs receiving liver transplants, 4 died during the operation
12 died during the 1st 3 days, but none of these showed
lence of immunological rejection. Nine animals were alive
ime of report. Liver transplantation in the pig may prove
e a useful model for analysis of immunological mechanisms
ransplantation biology.

40 SOME PRIMARY TUMORS OF THE HEPATIC MESENCHYME: AN ATTEMPT TO CLASSIFY D DIAGNOSE SPINDLE-CELL TUMORS OF THE LIVER.

.) Balouet, G. (Maritime Hosp., Brest, France) and P.
tombes. *Ann Anat Path (Paris)* 12(3):273-286, 1967.

216 primary liver tumors examined, 16 had a spindle-cell
cture and 6 (2.77%) were true mesenchymal tumors. A
cular component was involved in tumor development in 3
hese 6 tumors. Two of these were Kupffer-cell tumors and
as an hemangiopericytoma. In 2 other cases, a mesothelioma
an embryonic sarcoma, vascular structures were present
were not important or specific. The remaining tumor, a
oblastic sarcoma, had an undifferentiated spindle-cell struc-
. Five of these tumors were found in Africans and 1 in an
b. In contrast to nonspindle-cell tumors, true spindle-cell
ors were very invasive in 4 of these 6 cases and completely
royed the parenchyma in 5. Their structure was very homo-
eous although the arrangement of the spindle cells varied
tly. Very little necrosis was seen and, with the exception
he mesothelioma, very little sclerosis.

41 TEMPORARY REPLACEMENT OF THE LIVER BY HETEROLOGOUS LIVER PERFUSION.

r.) Boeckl, O. (Provincial Hosp., Salzburg, Austria), G.
lvan, H. J. Gibitz, E. Hell, O. Staindl, H. Steiner and G.
nmermann. *Langenbeck Arch Klin Chir* 318(3):209-216,
57.

patectomies were performed on heparinized Minnesota
arf swine. After blood had been rinsed out of the livers
h physiological saline containing 2000 U of heparin/liter,
livers were attached to a perfusion machine filled with
0-1400 ml of human blood. Perfusions were carried out
4 or 25C with blood entering the liver either through the
tal vein alone or through the portal vein and hepatic artery
leaving through the inferior vena cava. Perfusions were
for 3-12 hr. Although more bile was secreted during per-
ion with homologous blood, bile was also secreted during
erologous perfusion. BSP and ^{131}I-labeled Bengal Rose
e eliminated from the blood, although not in such large
ounts as in normal humans and swine. Both dyes were
nd in the bile. Arterial blood was 98% saturated with O_2
venous blood was 70% saturated. Ammonia was rapidly
inated from the blood after loading with 0.5 g of ammo-
m citrate. During 3 hr perfusions lactate dehydrogenase and
PT levels increased and alkaline phosphatase activities de-
ased. Perfusion with blood from cancer patients with ob-
ctive jaundice for 2 hr decreased bilirubin levels from 5.3
1.5 mg%, alkaline phosphatase from 62 to 46 mU/ml, and
a from 43 to 18 mg%, while SGPT levels increased from
to 21.3 mU/ml.

8442 TRYPTOPHAN-NICOTINIC ACID METABOLISM DURING ACUTE AND CHRONIC LIVER DISEASE.

(It.) Rottini, E. (Inst. Med. Symptomatol., U. Perugia, Italy), G. Cozzolino, G. Dominici and L. Scianaro. *Ann Fac Med Chir Perugia* 59(1):79-89, 1967.

The daily urinary excretion of tryptophan metabolites was determined in 24 subjects (6 females and 18 males, aged 16-78 yr) with or without oral administration of 100 mg/kg body weight of tryptophan. Of these subjects, 14 had acute hepatitis and 10 had chronic hepatitis. In acute hepatitis urinary excretion of kinurenin and acetylkinurenin were increased and that of 3-hydroxyanthranilic acid was reduced. After loading, 100% increases occurred in the urinary excretion of all tryptophan metabolites except for 3-hydroxyanthranilic acid which was increased 75%. By 28 days after the onset of symptoms, kinurenin and acetylkinurenin values had returned to normal, but the excretion of 3-hydroxyanthranilic acid was significantly increased. In chronic hepatitis the daily urinary excretion of tryptophan metabolites was almost normal, but 3-hydroxyanthranilic acid excretion was reduced. After loading, urinary excretion of all tryptophan metabolites increased more than 100% with the exception of that of 3-hydroxyanthranilic acid which increased 60%.

8443 CHANGES IN HEPATIC BLOOD FLOW AFTER VAGOTOMY AND PYLOROPLASTY. *(It.)*

Piaggi, V. (Inst. Gen. Med. Clin., U. Modena, Italy) and P. Vassanelli. *Acta Chir Ital* 23(6):659-665, 1967.

Hepatic blood flow was measured with colloidal ^{198}Au on 16 patients, aged 20-45 yr, before and 10-20 days and 8-12 months after they had undergone vagotomy and pyloroplasty for peptic ulcers. Before surgery hepatic blood flow ranged from 802-1296 ml/min, values which did not differ significantly from those measured in normal controls. A very significant reduction, averaging 41.5%, was seen 10-20 days after surgery. After 8-12 months the hepatic blood flow averaged 67.3% lower than values measured before surgery. It is suggested that reductions in hepatic blood flow might reduce bile production and biliary tract motility and account for diarrhea and steatorrhea developing after vagotomy.

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LIVER AND BILIARY TRACT

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8537 IMMUNOELECTROPHORETIC STUDY IN LIVER DISEASES. *(Ger.)* Mondorf, W. (2nd Med. Clin., U. Frankfurt/Main, Germany) and K. H. Goggel. *Epatologia* 13(3):549-554, 1967.

8538 JAUNDICE CAUSED BY ACUTE FATTY LIVER. *(Sp.)* de Tezanos P., S. (Dr. E. Fedormes Hosp., Valparaiso, Chile), F. Olavarria G. and Silva R. *Hosp Vina Mar* 23(3-4):102-113, 1967.

See also: 7601,7707,7730.7738,7739,7778,7791,7837,7840,7841,7842,7844,7851,7852,7860,7864,7877,7890,7891, 7892,7893,7897,7898,7899,7904,7908,7913,7914,7922,7925,7926,7928,7931,7938,7941,7942,7943,7947, 7948,7949,7950,7957,7962,7965,7969,7971,7975,7976,7978,7980,8088,8095,8196

539 **LIVER ULTRASTRUCTURAL AND BIOCHEMICAL CHANGES IN THE PRENEPHROTIC STAGE F AMINONUCLEOSIDE NEPHROSIS.** *(E.)* Lewin, P. K. anting Inst., U. Toronto, Ontario, Canada) and M. A. oscarello. *Exp Molec Path* 8(1):21-33, 1968.

ght- and electron-microscopic observations of the liver during e early stages of experimental nephrosis produced in Wistar bino rats by a single i.v. injection of 1% aminonucleoside solu- on were correlated with the incorporation of glucosamine-1- ^{4}C into plasma glycoproteins 6.5, 17.5 and 24 hr following e administration of the drug. Hepatocytic changes were ob- rved as early as 6 hr after injection of aminonucleoside and least 48 hr prior to the occurrence of any ultrastructural nal glomerular abnormalities, and 5 days prior to the onset proteinuria. These early ultrastructural changes in the hepato- tes consisted essentially of a loss of the regular arrays of doplasmic reticulum in the cytoplasm associated with reticu- r vesiculation. At 24 hr following aminonucleoside administra- on prominent cisternal dilatation of the endoplasmic reticulum, ten associated with small vesicular and tubular cytoplasmic ructures, was observed. Free and attached ribosomes were creased in number. Mitochondrial structural deformities oc- rred, but were an inconstant finding. Four days after amin- ucleoside injection most hepatocytes showed vesiculation the rough endoplasmic reticulum with increased numbers of ee ribosomes, lysosomes and microbodies. During the nephro- c state (7 days after aminonucleoside), changes in hepatocytes ere similar to those observed prior to proteinuria, but the toplasmic disorganization of the hepatocytes was widespread d was associated with increased numbers of ribosomes and lyribosomes in the hyaloplasm. Incorporation of ^{14}C-gluco- mine into plasma glycoproteins was depressed slightly, but gnificantly, at 6.5 and 17.5 hr compared with control animals. owever, by 24 hr after aminonucleoside administration a sig- ificant increase in the specific activity of the plasma glycopro- ins was observed. The reason for the initial slight depression glycoprotein synthesis is not understood.

540 **NO CHEMICAL EVIDENCE OF HEPATIC LIPID PEROXIDATION IN ACUTE ETHANOL TOXI- ITY.** *(E.)* Hashimoto, S. (Case Western Reserve Sch. Med., leveland, Ohio) and R. O. Recknagel. *Exp Molec Path* 8(2): 25-242, 1968.

an effort to determine whether acute ethanol intoxication volves peroxidative decomposition of rat liver lipids, the micro- mal, mitochondrial and nuclear lipid fractions from Wistar t liver 1, 9, 18 and 24 hr after administration of ethanol .57 ml of 50% solution/100 g by stomach tube) were studied ectrophotometrically. As expected, diene conjugation absorp- on (λ max=233 mμ), characteristic of peroxidized lipids, was monstrated in rat liver microsomal lipids peroxidized *in vitro* d after carbon tetrachloride poisoning (250 μl/100 g). How- er, no evidence of diene conjugation absorption could be de- cted in any subcellular fraction 1-24 hr after ethanol admin- ration. Using a semimicro iodimetric titration procedure, all amounts of lipid peroxides were found in peroxidized fflower oil and in rat liver microsomal lipids peroxidized *in ro.* In contrast to previously reported findings, no lipid peroxides were detected in total liver lipids or in lipids of isolated microsomes or mitochondria in either normal or ethanol-fed rats at 1 or 18 hr following ethanol administration. This failure to detect any evidence of lipid peroxidation at any time during the 1st 24 hr after acute ethanol intoxication greatly complicates current molecular concepts of acute ethanol toxicity.

8541 **STUDIES ON THE EFFECT OF AZATHIOPRINE IN CHRONIC LIVER DAMAGE.** *(Jap.)* Hiroshima, A. (Osaka City U. Med. Sch., Japan). *J Osaka City Med C* 17(1/2):89-99, 1968.

The effects, side-effects, and rebound phenomena of azathioprine were evaluated by liver function tests during the treatment of 14 cases of chronic hepatitis and 16 cases of cirrhosis. The drug was given for 5-26 weeks, at an initial dose of 100-200 mg/day and a maintenance dose of 50-100 mg/day. A marked improvement was noted in the serum transaminase activity during the treatment and BSP retention was also improved somewhat in some cases. After discontinuation of azathioprine, a rebound phenomenon in terms of a rise in serum transaminase activity was noted in 1/3 of the cases. Mild gastrointestinal disturbances were noted in 11 cases; the mild leukopenia noted in 6 cases disappeared within 2-3 weeks after the cessation of treatment. In rats given injections of egg yolk, azathioprine suppressed the increase in serum transaminase activity and reduced the increase in hepatic transaminase activity as compared to a control group receiving egg yolk alone; azathioprine also ameliorated the histological changes produced by the egg yolk injection.

8542 **THE CELLULAR ANALYSIS OF LIVER CARCINOGENESIS: ULTRASTRUCTURAL ALTERATIONS WITHIN HYPERPLASTIC LIVER NODULES INDUCED BY 2-FLUORENYLACETAMIDE.** *(E.)* Merkow, L. P. (U. Pittsburgh Sch. Med., Pa.), S. M. Epstein, B. J. Caito and B. Bartus. *Cancer Res* 27(9):1712-1721, 1967.

The fine structure of 2-fluorenylacetamide-induced hyperplastic liver nodules and the nonnodular surrounding liver was studied in male Wistar rats following a 24-hr fast. Within the nodule, annulate lamellae were frequently observed. These organelles were associated with an increase in smooth endoplasmic reticulum frequently arranged in cluster formation. In addition, within the nodule, there was a concomitant decrease in parallel-arrayed rough endoplasmic reticulum and numerous small, lightly staining vacuoles. On the other hand, the adjacent nonnodular liver did not contain annulate lamellae, but did show large osmiophilic lipid bodies. The surrounding liver displayed preservation of rough endoplasmic reticulum and less clustered smooth endoplasmic reticulum. The combination of ultrastructural features imparted a characteristic appearance which specifically differentiated the hyperplastic nodule from adjacent liver tissue.

8543 **CELLULAR ANALYSIS OF LIVER CARCINOGENESIS: THE INDUCTION OF LARGE HYPERPLASTIC NODULES IN THE LIVER WITH 2-FLUORENYLACETAMIDE OR ETHIONINE AND SOME ASPECTS OF THEIR MORPHOLOGY AND GLYCOGEN METABOLISM.** *(E.)* Epstein, S. (U. Pittsburgh Sch. Med., Pa.), N. Ito, L.

Merkow and E. Farber. *Cancer Res* 27(9):1702-1711, 1967.

In experiments on male Wistar rats weighing 150-200 g, administration of diets containing either 0.05% 2-fluorenylacetamide or 0.25-0.80% ethionine for 2-8 weeks produced large hyperplastic nodules in over half the rats, with a mortality rate of 22.4% and 6.1%, resp. These nodules were composed predominantly of hepatocytes which showed morphologic and biochemical differences from the surrounding nonhyperplastic liver. The glycogen or its metabolic control were also different in the nodule in that a significant amount of glycogen was present even after a 48-hr period of fasting and little breakdown was induced by glucagon administration. The nodules showed a consistent and progressive decrease in glucose-6-phosphatase activity as well as in glycogen phosphorylase activity. Evidence implicating the hyperplastic nodule as a step in the carcinogenic process is presented and discussed.

8544 INFLUENCE OF 3-METHYLCHOLANTHRENE ON LIVER CARCINOGENESIS IN RATS INGESTING DL-ETHIONINE, 3′-METHYL-4-DIMETHYLAMINOAZOBENZENE, AND N-2-FLUORENYLACETAMIDE. *(E.)*

Marugami, M. (Nara Med. U., Kashihara, Japan), N. Ito, Y. Konishi, Y. Hiasa and E. Farber. *Cancer Res* 27(11):2011-2019, 1967.

In order to determine whether the administration of 3-methylcholanthrene (3-MC) would inhibit the development of liver cancer induced by ethionine in the same way that it prevents cancer induction by 3′-methyl-4-dimethylaminoazobenzene or N-2-fluorenylacetamide, male Wistar rats were fed diets containing 0.25% ethionine, 0.06% 3′-methyl-4-dimethylaminoazobenzene or 0.025% N-2-fluorenylacetamide, with or without 0.0067% 3-MC, or 0.0067% 3-MC alone, for periods up to 5 months. Unlike its protective effect against cancer induction by the other 2 carcinogens, 3-MC had no evident effect on the development of preneoplastic or neoplastic changes induced in the liver by ethionine. At 5.8 months, liver cancer was found in 54.5% of rats given ethionine alone, 62.5% of rats given ethionine plus 3-MC, and 50-70% of rats given the other 2 carcinogens alone, but in none of the other groups. These findings are consistent with the conclusion that metabolism of ethionine by microsomal enzymes is not essential for liver carcinogenesis with this methionine analog.

8545 QUENCHING MOUSE LIVER IN ISOPENTANE AND PROPANE AT -155C: SOME MORPHOLOGIC AND CHEMICAL EFFECTS. *(E.)*

Young, D. E. (Armed Forces Inst. Path., Washington, D. C.), C. C. Griffin, E. A. Arnold and R. E. Stowell. *Arch Path* 84(5):499-508, 1967.

A comparative structural and biochemical study of the effects of isopentane and propane on mouse liver rapidly frozen at -155C and thawed at 24C showed similar types and degrees of damage by electron microscopy, although no changes were observed by light microscopy. Quenching in propane at -175C produced more severe damage than propane at -155C, primarily due to the increased cooling rate. Lipid was not extracted from tissues by either quenching agent. The acid hematein stain for phospholipids showed preservation of staining at the periphery of slices and loss of it centrally, possibly caused by denaturation of lipoprotein in the deeper areas. Biochemically, slightly more succinoxidase activity was lost during quenching in propane than in isopentane at -155C.

8546 HISTOCHEMICAL LOCALIZATION OF GLYCOGEN-SYNTHESIZING ENZYMES DURING PARASITIC INFECTIONS. *(E.)*

Mercado, T. I. (Natl. Inst. Allergy Infect. Dis., NIH, Bethesda, Md.) and T. Von Brand. *Exp Parasit* 21(3):325-335, 1967.

Polysaccharide synthesis by amylophosphorylase, glycogen synthetase, and branching enzyme was generally periportal in the livers of rats infected with *Plasmodium berghei*. In animals with low-grade infections synthesis was more uniform throughout the liver lobule, resembling that of normal rats, while in very highly infected animals (parasitemia over 70%) no significant activity was demonstrable. As in malaria, synthesis during hemobartonellosis was essentially periportal. In severe trypanosome infections periportal synthesis was also observed but not as regularly as in malaria or hemobartonellosis; centrilobular activity was observed frequently. A periportal deposition of suga corresponding to the areas of glycogen synthesis in the malaria-infected animals was not observed. In both infected and normal animals, sugar appeared distributed more or less evenly throughout the liver lobule. These findings parallel closely those obtained in previous *in vivo* studies on glycogenesis and indicate that a faulty enzyme mechanism is the factor responsible for the changes in glycogenesis during these infections.

8547 ULTRASTRUCTURAL ALTERATIONS OF DOG LIVERS DURING ENDOTOXIN SHOCK. *(E.)*

Boler, R. K. (U. Mississippi Sch. Med., Jackson) and A. J. Bibighaus III. *Lab Invest* 17(5):537-561, 1967.

Four adult mongrel dogs were given an i.v. injection of *Escherichia coli* endotoxin which produced profound shock. Sections of liver were taken prior to and at intervals of 15, 75, and 135 min after injection of endotoxin and were examined by electron microscopy. The earliest alterations observed were loss of glycogen from hepatocytes, which became more severe during later intervals, desquamation of sinusoidal endothelium, and vacuolation of hepatocytes. The large vacuoles subsequentl increased in quantity and size and were found to contain bundles of fibers having axial cross-striations with a periodicity of approximately 175 Å. These vacuoles were thought to contain fibrinogen and the fibrous material was considered to be fibrin. These vacuole-like cytosomes increased in density and size during the period of study and at 135 min some had ruptured limiting membranes. Considerable proliferation of agranular endoplasmic reticulum was noted beginning at 75 min. In addition, numerous alterations of granular endoplasmic reticulum mitochondria, and cell membranes and the formation of myeli figures were observed and considered to be nonspecific alterations related to the near necrotic condition of hepatocytes at 135 min. Sinusoids became congested to various degrees at 75 and 135 min with cellular debris, platelets, fibrin thrombi, erythrocytes, and leukocytes. The cell membranes of leukocytes were often broken and their specific cytoplasmic granule were released into the sinusoids.

8548 LIPID TRANSPORT IN EXPERIMENTAL DIETARY HEPATIC INJURY IN RATS. *(E.)*

György, P. (Philadelphia Gen. Hosp., Pa.), E. Cardi, C. S. Rose, M. Hirooka and B. W. Langer Jr. *J Nutr* 93(4):568-578, 1967.

The effect of lipotropic factors on lipid transport was studied in male Sprague-Dawley rats fed hypolipotropic diets rich in protein (extracted peanut meal with or without added small amounts of casein). With basal diets permitting at least initially satisfactory growth, the serum levels of cholesterol and lipoproteins fell precipitously. This effect was prevented by methionine and choline when mixed with the diet. However, given separately, the effect of methionine was not seen and that of choline was, in general, significantly reduced. Vitamin B_{12} was not able to prevent an initial drop in the serum levels of cholesterol and lipoproteins, which gradually returned to normal levels. Homocystine mixed with the diet, especially in combination with other lipotropic factors, produced significant hypercholesterolemia. Supplements of vitamin B_{12} greatly reduced the amount of total lipids with proportionate increases of the phospholipid fraction in the liver. In contrast, the total lipids and phospholipids were increased in the serum. Choline, and methionine, when given separately, were unable to prevent the development of fatty liver in long-term experiments. In the perirenal fat, in contrast to the lipids in liver and serum, polyunsaturated fatty acids characteristic of cod liver were missing. A somewhat higher concentration of linoleic acid was found in the groups with supplements of choline and vitamin B_{12}.

8549 AN EVALUATION OF THE EFFECT OF HALOTHANE ON LIVER FUNCTION AND DISEASE.

E.) Thompson, D. S. (VA Hosp., Little Rock, Ark.), C. N. Eason and B. W. Thompson. *Amer J Surg* 114(5):658-664, 1967.

A retrospective study of 545 patients subjected to halothane nesthesia between 1959 and 1965 did not produce any evidence of liver disease attributable to anesthesia. Only 3 of the 520 survivors showed any sign of liver disease, and hepatic pathology was no more common among the 25 fatalities than would be expected. A prospective study of liver function was than carried out on 63 patients, 31 receiving halothane and 32 receiving other anesthetics. Analysis of the results of liver function tests performed the day before, on the day of, and 2 and 7 days after surgery revealed no significant difference beween the 2 groups in the incidence of jaundice and elevated SGPT levels, although patients receiving halothane had a higher ncidence of elevated SGOT and those receiving nonhalothane nesthetics had a higher incidence of elevated alkaline phosphaase. It seems that upper abdominal surgery *per se* rather than alothane may be the principal mechanism leading to liver dysunction after surgery.

8550 DOSE-RESPONSE CHARACTERISTICS OF AFLATOXIN B_1 CARCINOGENESIS IN THE RAT.

(E.) Wogan, G. N. (Dept. Nutrit., Massachusetts Inst. Technol., Cambridge) and P. M. Newberne. *Cancer Res* 27(12): 370-2376, 1967.

Highly purified aflatoxin B_1 was administered to male and female Fischer rats by intubation, or by feeding, to induce hepaocellular carcinoma and other liver lesions; lesions were also bserved in other tissues. Male animals surviving a single LD_{50} ose (5 mg/kg) of the compound developed persistent liver esions, but no hepatocellular carcinoma, within 69 weeks after dosing. In males dosed with 400 μg administered in 10 equal daily doses, 4 of 24 animals developed hepatocellular carcinomas 35-82 weeks after treatment. A similar dose was ineffective in inducing carcinomas in females within an 82-week period, although preneoplastic liver lesions occurred with a high incidence. When aflatoxin B_1 (1.0 ppm in the diet) was fed continuously, hepatocellular carcinomas were induced in 18 of 22 males within 3-41 weeks, but did not appear in females until 64 weeks. At a dietary level of 0.015 ppm, carcinomas were induced in 12 of 12 males after 68 weeks and in 13 of 13 females killed after 80 weeks. Feeding of the 1.0 ppm diet for only the 14 initial days of the experiment resulted in minimal carcinoma incidence in both sexes 80 weeks after withdrawal.

8551 ALLERGIC LIVER DAMAGE IN RATS: WITH A COMPARISON BETWEEN ANOXIC AND CONGESTIVE HEPATOCYTIC VACUOLATION. *(E.)*

Papadimitriou, J. M. (Dept. Path., U. Western Australia, Perth) and J. B. Blackwell. *J Path Bact* 94(2):397-407, 1967.

Intracytoplasmic globules produced in the liver by i.v. injections of antigen (human serum albumin) into sensitized Wistar rats were compared by electron microscopy and histochemical studies with the type of liver damage that occurs in anoxia, ischemia and in combined congestion and anoxia. The large eosinophilic globules seen in the hepatocytes were shown to be lysosomal in origin. Morphological and histochemical differences were noted between the immunologically damaged livers and those subjected to ischemia, hypoxia and congestion. These results support the hypothesis that the direct toxic effect of antigen-antibody complexes, rather than mechanical obstruction of the hepatic sinusoids, is the cause of liver damage in sensitized rats.

8552 COMPARATIVE STUDIES OF THE NUCLEOLI OF MORRIS HEPATOMAS, EMBRYONIC LIVER, AND AFLATOXIN B_1-TREATED LIVER OF RATS.

(E.) Unuma, T. (Baylor U. Coll. Med., Houston, Texas), H. P. Morris and H. Busch. *Cancer Res* 27(11):2221-2233, 1967.

The nucleoli of the adult and embryonic rat liver, Morris 9121, 8995, 7787 and R-7 hepatomas, and the liver after prolonged aflatoxin B_1 treatment were compared with respect to size and ultrastructure. Embryonic liver cells had enlarged nucleoli with well-demarcated fibrillar and granular components. Cells of the Morris hepatomas had enlarged nucleoli. The demarcation of fibrillar and granular components in the nucleolonemas was similar to that of the normal liver in the Morris hepatoma 7787 and was progressively less in hepatomas 9121, R-7 and 8995. The ratios of the area containing fibrillar components to the total nucleolar area of the normal liver, embryonic liver and Morris hepatomas were within the same range despite differences in the size of the nucleoli. The morphologic resemblance of the nucleoli of the 7787 hepatoma to that of the normal liver may be related to the greater similarity of the base composition of rapidly labeled nucleolar RNA of this tumor to that of normal liver than that found for other tumors. In liver cells of aflatoxin B_1-treated rats, the enlarged nucleoli resembled those of regenerating liver. From these studies, it appears that the Morris hepatomas contain a spectrum of variation of nucleolar morphology ranging from those of the rapidly growing transplantable tumors to that of normal liver. These data support the view that studies on ultrastructure do not provide diagnostic criteria for tumors, at least with the method of fixation and staining employed in this study.

8553 **ASSOCIATION OF EMBRYONIC ANTIGENS WITH EXPERIMENTALLY INDUCED HEPATIC LESIONS IN THE RAT.** *(E.)* Stanislawski-Birencwajg, M. (Inst. Scient. Res. Cancer, CNRS, Villejuif, France), J. Uriel and P. Grabar. *Cancer Res* 27(11):1990-1997, 1967.

Short-term hepatic injuries were produced in rats by single i.p. injections of N-dimethylnitrosamine, carbon tetrachloride, and cadmium sulfate, or by partial hepatectomy. In all cases the animals responded with varying serum titers of embryonic α_2-glycoprotein which could be detected as early as 24 hr after the treatment. Antigen LA, another embryonic constituent, was never detected under the above conditions. Long-term hepatic injuries were produced by oral ingestion of carcinogenic aminoazo dyes, N-dimethylnitrosamine and aflatoxin B_1, and by noncarcinogenic analogs of aminoazo dyes. The lesions induced were those classically obtained following such treatments: hepatocarcinoma, bile-duct carcinoma, cirrhosis, hepatitis, etc. The embryonic α_2-glycoprotein was associated with these lesions, whereas Antigen LA was specifically associated with rats bearing hepatocarcinomas. Neither Antigen LA nor the α_2-glycoprotein were found in rats bearing tumors of organs other than the liver. Lipoprotein-esterase was increased in the majority of sera of rats with these various hepatic lesions.

8554 **LYSOSOMAL CHANGES ASSOCIATED WITH HYPEROXIA IN THE ISOLATED PERFUSED RAT LIVER.** *(E.)* Abraham, R. (Brit. Indust. Biol. Res. Assoc., Carshalton, Surrey, England), W. Dawson, P. Grasso and L. Golberg. *Exp Molec Path* 8(3):370-387, 1968.

Changes in lysosomes associated with hyperoxia were studied in livers isolated from male Carworth Farm E rats and perfused by the method of Mayes and Felts (1966). Increased O_2 tensions in the perfused blood resulted in the formation of enlarged lysosomes and autophagic vacuoles. Demonstration of acid phosphatase (both by light and electron microscopy), β-glucuronidase, aryl sulfatase, and E_{600}-resistant esterase in these organelles identified them as lysosomes. Electron-microscopic observations showed that except for changes in the lysosomes, none of the other cellular organelles were distinctly affected. The formation of autophagic vacuoles and the cell damage were prevented when perfusion was carried out with blood oxygenated with air, or when the liver was derived from a rat treated with chloroquine. These findings are attributed to alteration in the permeability of the lysosomal membrane resulting from hyperoxia. In turn, this permeability change releases hydrolytic enzymes into the cytoplasm leading to cell death, particularly in the centrilobular areas of the liver in fed and fasted rats. A special role in this process is envisaged for the smaller pericanalicular lysosomes, as indicated by a reduction in their number and a change in their distribution.

8555 **HEPATOCARCINOGENICITY OF GRISEOFULVIN FOLLOWING PARENTERAL ADMINISTRATION TO INFANT MICE.** *(E.)* Epstein, S. S. (Harvard Med. Sch., Boston, Mass.), J. Andrea, S. Joshi and N. Mantel. *Cancer Res* 27(10):1900-1906, 1967.

Random-bred infant Swiss mice were given subcutaneous injections of suspensions of griseofulvin in tricaprylin (2.5-50 mg/ml) according to various dosage schedules. Doses of griseofulvin in excess of 0.25 mg on day 1 of life produced acute toxicity. Following a total dosage of 3.0 mg in infant mice, or of 0.5, 0.5, 1.0, and 1.0 mg at ages of 1, 7, 14, and 21 days, resp., a high incidence of hepatoma developed (44% of 16 male mice alive at 49 weeks, in comparison with 8% of 48 male solvent controls.

8556 **THE EFFECT OF GONADECTOMY ON THE DEVELOPMENT OF HEPATOMAS INDUCED BY URETHAN.** *(E.)* Vesselinovitch, S. D. (Chicago Med. Sch., Ill.) and N. Mihailovich. *Cancer Res* 27(10):1788-1791, 1967.

C57BL x C3H F_1 mice of each sex (7 days old at time of 1st injection) were given 6 urethan injections (0.5 mg/g body weight) at 3-day intervals. Three weeks after the last urethan treatment, one half of the mice were gonadectomized and the other half were sham-operated. At 52 weeks of age all animals were killed. The sham-operated males developed hepatomas in 96% and the females in 20%. The orchidectomized mice showed a significantly lower (62%) and the ovariectomized group a significantly higher (67%) incidence, as well as size and multiplicity of hepatomas than their corresponding non-gonadectomized controls. Morphology of the kidneys of gonadectomized mice primarily indicated a mixed male and female hormonal environment, which was associated with the presence of adrenal cortical hyperplasia and in a few instances with the appearance of adenomas, both of the β-cell type. Gonadectomy thus practically abolished the sex difference both in the host hormonal environment and in hepatoma development. The results provide indirect evidence that male hormones enhance and female hormones suppress the development of hepatomas, although not of the other tumors (malignant lymphomas, Harderian gland tumors, and lung adenomas) initiated by urethan.

8557 **ALTERATIONS IN THE LIVER DUE TO ACUTE AMINO ACID DEFICIENCY: AN ELECTRON MICROSCOPIC STUDY OF YOUNG RATS FORCE-FED A THREONINE-DEVOID DIET.** *(E.)* Shinozuka, H. (U. Pittsburgh Sch. Med., Penna.), E. Verney and H. Sidransky. *Lab Invest* 18(1):72-85, 1968.

Hepatic parenchymal cells of 5-week-old female Sprague-Dawley rats force-fed a complete or a threonine-devoid diet for 1-7 days were studied by electron microscopy. In all experiments, the findings in the animals force-fed the threonine-devoid diet were compared with those in animals force-fed the complete diet or fed commercial diet *ad libitum*. In the nuclei of the experimental animals, striking nucleolar enlargement development after 1 day on the deficient diet and became progressively marked. This enlargement was accompanied by ovoid or rod-like elongation, and frequently there was apparent attachment of one or more portions of the nucleolus to the nuclear membrane. After primary glutaraldehyde fixation followed by osmium fixation, there appeared to be a direct connection between peripherally localized nucleoli and nuclear pores. This finding of localized contiguity of nucleoli to the nuclear membrane suggested that under certain conditions it may serve as a pathway for the direct transfer of ribonucleic acid from the

cleolus to the cytoplasm. In the cytoplasm, a large amount
glycogen and lipid accumulated in the hepatic cells of ani-
ls force-fed the threonine-devoid diet. Concurrently, there
s a marked increase in the number and size of lysosomes,
ny of which contained large amounts of glycogen. An in-
ase in the number of free polysomes in the cytoplasm was
ed.

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**URINARY COPROPORPHYRIN EXCRETION AND HEPATIC FUNCTION IN WOMEN TAK-
G ORAL CONTRACEPTIVES.** *(E.)* Burton, J. L. (Royal
rm., Edinburgh, Scotland), N. B. Loudon and A. T.
son. *Lancet* 2(7530):1326-1328, 1967.

nary total coproporphyrin concentrations, measured by the
id screening test of Donath, were determined in 50 women
had been taking oral contraceptives for more than 2 yr
compared to those in 50 women of the same age who used
lusive pessaries and no oral contraceptives. Liver function
s were performed in all women who had increased copro-
phyrins and in a corresponding number of women taking
l contraceptives who had normal coproporphyrins. It was
ed that 13 (26%) of the women taking oral contraceptives
elevated coproporphyrins in their urine, compared with
e in the control group. An increase in BSP retention was
ed in 5 of the 13 with elevated urinary values and in 3 of
13 women with normal coproporphyrin concentrations;
association of increased urinary coproporphyrin concentra-
with BSP retention was not significant. There was no sig-
cant correlation between urinary coproporphyrin concentra-
and age, although the mean BSP retention was higher in
nen aged 40 and over (7%) than in women below 40
5%); this difference was also not significant.

9

**LOCAL AND GENERALIZED EFFECTS OF LOCAL RADIATION. PART I. EFFECTS OF
CAL RADIATION TO THE LIVER AND UPPER INTES-
IES IN PARTIALLY HEPATECTOMIZED, SHAM-OPER-
ED, AND NORMAL RATS.** *(E.)* Looney, W. B. (U.
ginia Sch. Med., Charlottesville) and L. O. Chang. *Radiology*
4):906-918, 1967.

series of experiments were performed: 1) operated, sham-
rated, and normal rats were irradiated at the peak of DNA
thesis (21 hr following partial hepatectomy) with 94 or
0 r of X-radiation; and 2) 94, 750, or 1500 r were given
artially hepatectomized rats 21 hr after surgery. In both
es, the effects of radiation were evaluated for up to 48 hr.
al X-radiation to the liver and underlying intestines resulted
he immediate depression of DNA synthesis, continuing over
48-hr study period. Maximum depression occurred at 8 hr,
h recovery in individual cells at 24 hr after doses ranging
n 94-1500 r. Depression of DNA synthesis at times later
n 8 hr appeared to be from the inhibition of cells initiating
A synthesis. Depression of DNA synthesis was also found
he shielded thymus and lower intestines. There was greater
ression and less recovery of DNA synthesis following irra-
ion in the partially hepatectomized rats compared to the
m-operated and normal rats. This could be the result of
combined effect of the operative procedure and the radia-
. The results of other investigations with regenerating liver
thymus, however, suggest that regenerating organs may
npete for some unknown factor necessary for DNA synthe-

8560

THE PROTECTIVE ACTION OF SOME PHENOTHIAZINES AGAINST THE *IN VITRO* TOXICITY OF CARBON TETRACHLORIDE. *(Fr.)* Boyer, J. B. (Fac. Med. Pharm., Clermont-Ferrand, France), P. Bastide, P. Tronche and G. Dastugue. *C R Soc Biol (Paris)* 161(1):1089-1091, 1967.

Liver slices from male rats were incubated for 1 hr at 37C in an atmosphere of O_2 with 2 ml of buffer, 2 ml of buffer containing 10 μg/ml of CCl_4, or 2 ml of buffer containing 10 μg/ml of CCl_4 and 10^{-7} M phenothiazine. Phenothiazines tested included promethazine, chlorpromazine, thiazinamium, oxomemazine, and diethazine. CCl_4 alone increased the protein, RNA, and DNA contents and increased 5′-nucleotidase activity in the incubation medium. All of the phenothiazines definitely counteracted these effects. Promethazine and oxomemazine were the most active. In almost all cases they reduced the values obtained to control levels.

8561

MORE STUDIES ON THE PRODUCTION OF SPECIFIC ANTIBODIES FOR HOMOLOGOUS AND AUTOLOGOUS LIVER TISSUE IN EXPERIMENTAL ANIMALS. I. SEROLOGICAL FINDINGS. *(Ger.)* Götz, H. (Med. Clin., U. Erlangen, Germany), F. Scheiffarth, W. Warnatz and W. Christmeier. *Z Gastroent* 5(5):299-303, 1967.

Under hexobarbital anesthesia, 10 rabbits were given transduodenally 5 ml of an emulsion containing 5 parts of 10% ethyl alcohol to 1 part of CCl_4. Half of these animals then received 5 injections of homologous liver extract and Freund's adjuvant at 1 week intervals and the rest served as controls. After immunization all animals were given CCl_4 with or without homologous liver extract s.c. at 12 hr intervals to rule on the effect of nonspecific stimulation. No changes in total serum proteins were seen in either group, but sensitized animals did have dysproteinemia with increases in albumin and smaller increases in globulin fractions, particularly β- and γ-globulins. A fraction intermediate between albumin and α_1-globulin was found in 3 sensitized rabbits. Serum lability tests were normal in all animals of both groups. Serum lactate dehydrogenase activities were normal in the control group. Among sensitized animals they increased continuously until 8 weeks after administration of CCl_4 and then decreased slightly. Hemagglutination titers were never higher than 1:32 in rabbits receiving CCl_4 alone, but in sensitized animals they were as high as 1:256 and 1:512 at 11 weeks after administration of CCl_4. No evidence of precipitates with homologous liver extracts were seen in either group with 2-dimensional immunodiffusion in agar gels.

8562

COMPARATIVE STUDIES OF CHANGES IN SGOT AND SGPT LEVELS IN THE GOLDEN HAMSTER WITH EXPERIMENTAL, EXTRA-INTESTINAL AMEBIASIS. *(Ger.)* Raether, W. (Farbwerke Hoechst AG., Frankfurt/Main-Höchst, Germany), G. Scheubert and O. Vanderbeke. *Z Tropenmed Parasit* 18(3):304-320, 1967.

Significant increases in SGOT and SGPT levels were found in golden hamsters which were given 0.05 ml of a dilute culture of *Escherichia coli* and *Pseudomonas aeruginosa* i.p. and, 3 and 10 days later, 30,000 ameba (hamster-passaged *Entamoeba histolytica*, strain F) in gelatin between the 2 lobes of the liver. No increases in transaminase activity occurred in hamsters injected with the bacteria alone, immunized and injected with bacteria, immunized i.p., or treated with sterile Ringer's solution in gelatin placed between the lobes of the liver. Necrotic changes, which were often extensive and associated with abscesses containing trophozoites, were found in 65 of 69 infected hamsters. Although ameba were isolated from the livers of 3 of the 4 remaining animals, their livers showed no signs of necrosis but had fat deposits spreading out from the periphery. These 4 animals also had elevated transaminase levels. No evidence was found of a quantitative relation between SGOT activities or the rate SGOT entered the bloodstream and the extent of cell damage or the rate of cell destruction. Strain M of *E. histolytica* was less virulent and produced only a gradual increase in enzyme activities and less extensive necrosis.

8563 **LIVER FUNCTION IN WORKERS ENGAGED IN THE PRODUCTION OF CERTAIN CHLORINATED ORGANIC CHEMICAL POISONS.** *(Rus.)* Paramonchik, V. M. (Kiev Inst. Occup. Hlth. Dis., USSR). *Sovet Med* 31(3):62-65, 1968.

8564 **JAUNDICE AND INTRAHEPATIC CHOLESTASIS DUE TO ORAL CONTRACEPTIVES.** *(Ger.)* Piesbergen, H. (Gen. Hosp., Hamburg-Eilbek, Germany). *Med Welt* 18(7):451-453, 1968.

8565 **CLINICAL AND FUNCTIONAL CONDITION OF THE LIVER IN CHRONIC ALCOHOL POISONING.** *(Rus.)* Zakharov, V. N. *Sovet Med* 31(3):57-62, 1968.

8566 **OPEN AND CLOSED LIVER TRAUMA.** *(Rus.)* Stoianov, A. (Inst. First Aid, Sofia, Bulgaria) and M. Khristov. *Khirurgiia (Moskva)* 44(3):103-106, 1968.

8567 **OPERATIONS FOR LIVER INJURIES TAKING INTO CONSIDERATION DAMAGE TO INTRAHEPATIC VESSELS AND BILE DUCTS.** *(Rus.)* Shapkin, V. S. (Vladivostok Med. Inst., USSR) and P. I. Luk'ianov. *Khirurgiia (Moskva)* 44(3):109-115, 1968.

8568 **THE TREATMENT OF OPEN AND CLOSED INJURIES TO THE LIVER.** *(Rus.)* Babichev, S. I. (A. A. Ostroumov Munic. Clin. Hosp. No. 33, Moscow, USSR) and N. V. Dement'ieva. *Khirurgiia (Moskva)* 44(3):106-108, 1968.

8569 **GRANULOMATOUS HEPATITIS OF UNKNOWN ETIOLOGY: AN ETIOLOGIC AND FUNCTIONAL EVALUATION.** *(E.)* Guckian, J. C. (Div. Infect. Dis., U. Texas - Med. Br., Galveston) and J. E. Perry. *Amer J Med* 44(2):207-215, 1968.

8570 **CLINICAL TRIAL OF 'FLAGYL' IN AMOEBIC LIVER ABSCESS.** *(E.)* Harinasuta, T. (U. Med. Sci., Bangkok, Thailand), P. Wiriyawit, D. Bunnag and S. Tejavanija. *Indian Pract* 21(10):732-736, 1968.

8571 **METRONIDAZOLE IN AMOEBIC LIVER ABSCESS.** *(E.)* Banerji, R. N. (Safdarjang Hosp., New Delhi, India), A. K. Basu and R. D. Ayyar. *Indian Pract* 21(10):737-741, 1968.

8572 **METRONIDAZOLE IN THE TREATMENT OF AMOEBIC LIVER ABSCESS.** *(E.)* Powell, S. (Amebiasis Res. U., U. Natal, South Africa). *Indian Pract* 21(10):728-731, 1968.

8573 **THE TREATMENT OF HEPATIC AMEBIASIS WITH METRONIDAZOLE.** *(E.)* Chhetri, M. K (S. S. K. Hosp., Calcutta, India), N. C. Chakravarty, B. Bhattacharyya and S. K. Sarkar. *Indian Pract* 21(10):742-74 1968.

8574 **BEHAVIOR OF LIVER MITOCHONDRIA AN LYSOSOMES FOLLOWING ACUTE INTOXIC TION WITH TRITON X-100: A BIOCHEMICAL AND ELE TRON MICROSCOPIC STUDY.** *(It.)* Baccino, F. M. (Inst. Gen. Pathol., U. Turin, Italy), P. Pani and L. Congiu. *Sperimentale (Firenze)* 116(5):293-311, 1966.

8575 **LIVER CELL ABNORMALITIES INDUCED IN MICE BY THE FUNGAL TOXIN CAUSING LUPINOSIS.** *(E.)* Gardiner, M. R. (Animal Hlth. Lab., Dept Agric., South Perth, Western Australia). *J Path Bact* 94(2): 452-455, 1967.

8576 **FATAL LISTERIOSIS INDUCED BY ETHIONI IN ALBINO RATS.** *(E.)* Bertók, L. (Vet. Med. Res. Inst., Hungarian Acad. Sci., Budapest) and J. Szemerédi. *J Path Bact* 94(1):194-196, 1967.

8577 **UNCOUPLING OF OXIDATIVE PHOSPHORYLATION IN RAT LIVER AND SPLEEN MITOCHONDRIA BY EXPOSURE TO TOTAL-BODY IRRADIATION.** *(E.)* Yost, M. T. (Dept. Biol., Amherst Coll., Mass.), H. H. Robson and H. T. Yost. *Radiat Res* 32(2):187-199, 1967.

8578 **EXPERIMENTAL PRODUCTION OF BILHARZIAL PIPE-STEM FIBROSIS IN THE CHIMPANZEE.** *(E.)* von Lichtenberg, F. (Peter Bent Brigham Hosp., Boston, Mass.) and E. H. Sadun. *Exp Parasit* 22(2):264-278, 1968.

8579 **THE ROLE OF LIPID PEROXIDATION AND ANTIOXIDANTS IN ETHANOL-INDUCED LIPID ALTERATIONS.** *(E.)* Di Luzio, N. R. (U. Tennessee Med. Sch., Memphis). *Exp Molec Path* 8(3):394-402, 1968.

8580 **INCIDENCE OF ANICTERIC HEPATITIS IN ASYMPTOMATIC YOUNG MEN: I. PILOT STUDY.** *(E.)* Riegel, N. (USAF Hosp., Keesler AFB, Biloxi, Miss.) and R. A. Sanowski. *Southern Med J* 60(11):1254-125 1967.

8581 **REGENERATION OF THE LIVER AFTER HEPATECTOMY. [CASE REPORT]** *(E.)* DeLand, F. H. (Johns Hopkins Med. Inst., Baltimore, Md.) an H. N. Wagner Jr. *J Nuclear Med* 9(11):587-589, 1968.

8582 **SOME ASPECTS OF PATHOLOGY AND CLIN CAL FEATURES OF AMOEBIC LIVER ABSC**

.) Subramaniam, R. (Madras Med. Coll., India). *Indian Pract* (10):724-726, 1968.

583 AMOEBIC LIVER ABSCESS: ANALYSIS OF AUTOPSY AND BIOPSY MATERIAL. *(E.)* kat, B. K. (Nehru Post-Grad. Inst. Med. Ed. Res., Chandigarh, dia). *Indian Pract* 21(10):747-748, 1968.

584 HEPATIC AMOEBIASIS. *(E.)* Charmot, G. (Dept. Therapeut. Res., SPECIA, Paris, France). *dian Pract* 21(10):759-762, 1968.

585 TRIAL OF METRONIDAZOLE IN EXTRA-COLONIC AMOEBIASIS: A PRELIMINARY RE-)RT. *(E.)* Subramaniam, R. (Madras Med. Coll., India), N. adanagopalan, T. Vijayalakshmi and M. Shantha. *Indian Pract* (10):785-791, 1968.

586 LIVER DYSFUNCTION WITH MESTRANOL BUT NOT WITH NORETHYNODREL IN A PATIENT ITH ENOVID-INDUCED JAUNDICE. *(E.)* Urban, E. (U. olorado Med. Ctr., Denver), B. W. Frank and F. Kern Jr. *Ann tern Med* 68(3):598-602, 1968.

8587 THE EFFECT OF ETHER ANESTHESIA ON THE GLYCOGEN CONTENT IN THE LIVER. *(Rus.)* Zhdanov, G. G. (Kalinin Med. Inst., USSR). *Eksp Khir Anest* 13(4):79-80, 1968.

8588 DRUG-INDUCED CHOLESTATIC HEPATITIS. *(Fr.)* Caroli, J. and P. Opolon. *Rev Prat* 18(15): 2279-2289, 1968.

8589 ALCOHOLIC NECROSIS OF THE LIVER. *(Fr.)* Bertrand, L. (Saint Éloi Hosp., Montpellier, France) and H. Michel. *Rev Prat* 18(15):1313-2330, 1968.

8590 MODERN OUTLOOK ON THE TREATMENT OF LIVER INJURIES. *(Pol.)* Wąsowski, J. (Acad. Med., Gdańsk, Poland). *Pol Przegl Chir* 40(1):94-97, 1968.

8591 CYTOLYTIC HEPATITIS CAUSED BY DRUGS. *(Fr.)* Girard, M. *Rev Prat* 18(15):2267-2277, 1968.

See also: 7716,7722,7725,7729,7736,7740,7748,7749,7771,7779,7780,7781,7802,7804,7805,7870,8050,8654

8592 TRANSFUSION HEPATITIS ARISING FROM ADDICT BLOOD DONORS.

(E.) Cohen, S. N. (New Jersey State Dept. Health, Trenton) and W. J. Dougherty. *JAMA* 203(6):139-141, 1968.

Between April 1 and June 30 of 1962, a number of narcotics addicts and suspected addicts sold blood at a local proprietary blood bank. The effect of this source on the incidence of hepatitis in recipients was determined in a follow-up study carried out by the New Jersey State Department of Health and involved more than 1000 patients. Seventeen cases of serum hepatitis were detected, all but one of which were in the study group receiving blood from the blood bank under investigation. Although the incidence of hepatitis was no higher than usual among the recipients of this bank's blood (2.6%), it was 6 times as high as in the control group, and 70 times as high among those specifically receiving addict and suspected-addict donations. The narcotics addict thus seems to be a significant source of homologous serum hepatitis.

8593 URINARY EXCRETION OF FORMIMINOGLUTAMIC ACID (FIGLU) DURING VIRAL HEPATITIS IN CHILDREN.

(It.) Gomirato-Sandrucci, M. (Pediat. Clin., U. Turin, Italy), N. Nigro, G. Bonenti and L. Benso. *Epatologia* 13(3):497-518, 1967.

Formiminoglutamic acid (FIGLU) determinations were made by the method of Tabor and Wingarden on 24-hr urine specimens from 44 children, aged 3-13 yr, with viral hepatitis. In the early stages of hepatitis all of these patients had elevated serum bilirubin and transaminase levels and abnormal liver function tests, erythrocyte sedimentation rates, and serum protein fractions. Urinary FIGLU excretion ranged from 2.4 to 224.20 μM/hr (mean 75.99 μM/hr). During convalescence all other values tended to normalize while urinary FIGLU excretion increased even further in most subjects (mean 157.75 μM/hr). The difference between urinary FIGLU excretion during the early stages of hepatitis and that during convalescence is statistically significant. It is suggested that increased FIGLU excretion during convalescence is due to a relative deficiency of folic acid which is required for its further breakdown. Urinary FIGLU excretion may serve as a possible test for liver regeneration.

8594 CLINICAL AND FUNCTIONAL FINDINGS ON THE REGENERATIVE EFFECT OF ANABOLIC STEROIDS IN THE TREATMENT OF VIRAL HEPATITIS.

(It.) Laterza, G. (Children's Hosp., Bari, Italy). *Epatologia* 13(3):601-610, 1967.

The duration of the initial fever, serum transaminase values, the results of MacLagan's and Hanger's tests, and bilirubin levels were compared for 30 children with viral hepatitis who were given norandrostenolone decanoate and 30 who were not. Children ranged in age from 2-12 yr. Normalization of SGOT levels and MacLagan's test occurred significantly sooner and the febrile period was somewhat shorter among the treated children. Recurrences of jaundice and fever occurred in 2 untreated patients. No symptoms of intolerance, complications, or virilization were seen in the treated group.

8595 CLINICAL AND ULTRASTRUCTURAL FEATURES OF ANICTERIC VIRAL HEPATITIS.

(It.) Teodori, U. (Inst. Spec. Med. Path., U. Florence, Italy) and P. Gentilini. *Fegato* 13(2):127-150, 1967.

Of 4 patients (3 teenagers and a 58-yr-old man) with histologically confirmed anicteric viral hepatitis, only 2 had manifest clinical symptoms. Liver function tests were rarely abnormal although serum transaminases were elevated. Electron microscopic examination of biopsied material showed considerable changes in the Disse spaces, Kupffer's cells, and the vascular pole of the hepatocytes. Except for some areas of cytoplasmi necrosis, the rest of the hepatocyte was almost normal. The Golgi apparatus on the side of hepatocytes bordering on the biliary tract was normal or, more often, hypertrophic; the cana liculi were normal. The absence of jaundice in these patients is attributed to the retention of the Golgi apparatus, even in cells with cytoplasmic necrosis. Slight increases in indirect bilirubin can be traced to inadequate absorption in the altered vascular pole. The presence of active fibrillogenesis in the Diss spaces and profound but reversible intercellular diastasis may precede the development of pericellular fibrosis in patients wh later develop chronic hepatitis.

8596 VIRUS-INDUCED PELIOSIS HEPATIS IN RATS

(E.) Bergs, V. V. (U. Miami Sch. Med., Florida) and T. M. Scotti. *Science* 158(3799):377-378, 1967.

Inoculation of newborn Fischer, W/Fu, or Lewis rats with the 9H virus recently isolated from leukemic rat tissues resulted in the development of peliosis hepatis. The virus was recoverable from the peliotic livers. The cystic lesions were of the parencl mal peliosis type seen in humans and appeared to be the result of focal hepatic necrosis followed by hemorrhage. These findings suggest a possible new etiological factor for consideration in further investigations on peliosis hepatis in other species, an also provide a model system for studies in rats on the pathogenesis of this disease and the nature of the inducing virus.

8597 CHANGES IN ERYTHROCYTE G6PD DURING INFECTIOUS HEPATITIS IN CHILDREN.

(It.) Rosmino, G. C. (Pediat. Clin., U. Turin, Italy) and M. T. Norelli. *Minerva Pediat* 19(27):2322-2325, 1967.

Erythrocyte glucose-6-phosphate dehydrogenase activities were determined in 26 children, aged 6-13 yr, with confirmed infectious hepatitis 15-20 days after the appearance of jaundice. These activities were below normal limits in 15 cases and were normal in 11. It is suggested that low enzyme activities might be due to high concentrations of phosphate ions which inhibit union of the apoenzyme with the coenzyme.

8598 THE IMMUNOGLOBULIN PATTERN IN ACUT INFECTIOUS HEPATITIS IN CHILDREN.

(It.) Bottone, E. (Pediat. Clin., U. Pisa, Italy), R. Corda, P. A. Macchia, A. Falorni, G. Baldini and V. Badas. *Minerva Pediat* 19(27):2311-2320, 1967.

Immunoglobulins were determined by radial immunodiffusion i serum from 80 children, aged 3 months to 12 yr, with clinical and biochemical symptoms of viral hepatitis. All patients wer were given the same treatment, except that 50 children were given 1 mg/kg/day of prednisone for 2 weeks and 30 were not. In both groups IgM levels were significantly increased

d were still increased, although not as much, 3-4 weeks after
e onset of symptoms. Increases in IgM do not have a pro-
ostic value since there is no relation between the IgM level
d the severity of hepatitis or changes in bilirubin and transa-
nase levels. Significant increases in IgG levels were found in
e first week. Since IgG has a half-life of 23 days, levels must
rease during the incubation period and might be of value
ring epidemics for identifying individuals who have contracted
e infection without developing clinical symptoms. IgA values
re not increased uniformly in all cases. In determinations
1 3-4 weeks after the onset of symptoms, increased IgA
ues had returned to normal. There were no differences be-
een immunoglobulin levels in children who received predni-
ne and those who did not.

99 **ASPECTS OF IMMUNITY IN VIRAL HEPA-
TITIS.** *(Fr.)* Constantinesco, N. (Inst. Hyg.,
si, Romania), O. Zavate, I. Micu, I. Buzdugan and G. Virna.
esse Med 75(38):1879-1882, 1967.

idemiological studies in Romania showed that the annual
orbidity of poliomyelitis, rubella, measles, and mumps was
ich higher among children under 15 yr than among adults,
ile the morbidity of infectious hepatitis was only 1-3 times
gher among children than among adults. Between 1953 and
65, infectious hepatitis occurred only sporadically among
manian children. Recurrences occurred in 5.4% of 2123
tients with infectious hepatitis (5.0% in children, 5.7%
adults). Of 116 recurrences, 56 (48.3%) developed less
an 3 yr after the initial infection and 60 (51.7%) developed
12 yr later and are considered true recurrences. A study of
milies in which members developed recurrences of hepatitis
owed no evidence of crossed immunity between infectious
d serum hepatitis or homologous immunity during the initial
fection of the recurrence. The recurrence was not consistently
s severe than the initial attack in 85 patients with recurrences
hepatitis. These findings suggest that the first episode of
patitis does not necessarily confer immunity to subsequent
fections.

00 **RECENT FINDINGS ON THE ECOLOGY OF
VIRAL HEPATITIS. STATISTICAL STUDY.**
.) Rondanelli, E. G. (Inst. Gen. Med. Clin., U. Pavia, Italy),
Gorini, R. Moratti and G. F. Lanzarini. *Minerva Med*
(79):4147-4156, 1967.

uring the period 1960-1965, 806 cases of viral hepatitis
ere reported in the province of Pavia, Italy. These included
9 subjects under 12 yr old. The annual morbidity increased
om 97 cases (18.71 cases/100,000 inhabitants) in 1960 to
6 (67.46/100,000) in 1965, for an increase of more than
0%. Until the end of 1963, a decrease occurred in the
orbidity among children while the adult morbidity remained
nost stationary. The morbidity was relatively higher in more
nsely populated areas. At the beginning of 1964, the mor-
idity began to increase, particularly among adults, and to
read to subjects living in less densely populated regions,
rticularly in sparsely populated hilly and mountainous re-
ons. The morbidities from 1964 on are higher than those
r Italy as a whole.

01 **VIRAL HEPATITIS AND PREGNANCY.** *(It.)*
Di Nola, F. (Amedeo di Savoia Hosp. Infect. Dis., Turin, Italy), G. Bracco, A. Pasquale and P. Moiraghi. *Minerva Med* 58(79):4156-4160, 1967.

Between January 1, 1965 and March 31, 1967, 15 pregnant patients had viral hepatitis. Of these, 4 were in the 1st trimester of pregnancy, 5 in the 2nd trimester, and 6 in the 3rd trimester. There were no significant differences between total bilirubin, SGOT, and SGPT values in these patients and 75 nonpregnant women, aged 19 to 35 yr, with viral hepatitis. Infants born to the 6 women who were followed to term were all normal. One patient in the 3rd trimester gave birth to a normal infant 20 days prematurely.

8602 **VIRAL HEPATITIS AND AGE. A STATISTICAL STUDY.** *(It.)* Rondanelli, E. G. (Inst. Gen. Med. Clin., U. Pavia, Italy), P. Gorini, R. Moratti and D. Dionisi. *Minerva Med* 58(79):3955-3962, 1967.

The χ^2-test was used to analyze 806 cases of viral hepatitis reported in the province of Pavia, Italy, between 1960 and 1965. The distribution of viral hepatitis reached a maximum in children from birth to 9 yr and then decreased exponentially with age. This distribution was almost exactly the same for males and females but differed significantly in different geographic regions. The mean age of patients was inversely proportional to the population density so that in sparsely populated hilly and mountainous regions the morbidity was highest among subjects from 10-19 yr old. In 1960 the ratio of children/adults with viral hepatitis was 4.38, while in 1965 this ratio was 1.59.

8603 **IMMUNOLOGICAL ASPECTS OF ACTIVE CHRONIC HEPATITIS. IMMUNOELECTROPHORETIC OBSERVATIONS.** *(It.)* Scalise, G. (Clin. Infect. Dis., U. Siena, Italy), P. M. Lecchini and C. A. Boggiano. *Minerva Med* 58(79):4563-4572, 1967.

Sera from 27 patients with chronic hepatitis were subjected to agar immunoelectrophoresis. The IgA and IgG fractions were increased and the IgM fraction was normal in 3 patients who had a previous episode of viral hepatitis confirmed histologically. Of 11 patients with chronic hepatitis and a history of acute hepatitis, the IgA and IgG fractions were increased in 10 and the IgM fraction in 3. The IgG fraction was increased in 8, the IgA fraction in 6, and the IgM fraction in 5 of 9 patients with chronic hepatitis and a history of alcoholism. Of 4 patients with subacute hepatitis, only 1 had increased IgM and IgA fractions, while IgG levels were increased in 3 cases and reduced in 1.

8604 **CLINICAL FEATURES OF VIRAL HEPATITIS. STATISTICAL STUDY.** *(It.)* Rondanelli, E. G. (Inst. Gen. Med. Clin., U. Pavia, Italy), P. Gorini, R. Moratti and D. Dionisi. *Minerva Med* 58(79):3962-3973, 1967.

A clinical study was made of 406 patients, aged 2 months to 95 yr, with viral hepatitis. The duration of symptoms ranged from 11-95 days and was significantly longer in adults than in children under 14 yr. The preicteric period was significantly longer in adults than in children. In the vast majority of cases jaundice lasted from 2-6 weeks and lasted significantly longer in adults than in children. Fever in the preicteric and icteric periods, gastrointestinal symptoms, hepatomegaly, and urinary hyperchromatism were found significantly more often among children than among adults.

8605 **THE FUNCTIONAL STATE OF THE ADRENAL CORTEX IN CHILDREN WITH INFECTIOUS HEPATITIS.** *(Rus.)* El'kina, Iu. A. (Minsk Med. Inst., USSR) and I. A. Savitskaia. *Pediatriia* 46(9):47-49, 1967.

8606 **PATHOGENETIC BASIS FOR THE USE OF PREDNISOLONE IN THE TREATMENT OF CHILDREN WITH INFECTIOUS HEPATITIS.** *(Rus.)* Nisevich, N. I. (N. I. Pirogov 2nd Moscow Med. Inst., USSR) and A. G. Bokovoi. *Pediatriia* 47(1):7-11, 1968.

8607 **THE IMPORTANCE OF KOUMISS AS A DIETARY FACTOR IN THE COMPLEX THERAPY OF CHILDREN WITH INFECTIOUS HEPATITIS (BOTKIN'S DISEASE).** *(Rus.)* Tuktarova, Kh. M. (Bashkir Med. Inst., Ufa, USSR). *Pediatriia* 47(1):23-25, 1968.

8608 **THE OCCURRENCE AND FREQUENCY OF INOCULATION HEPATITIS, BASED ON MORPHOLOGICAL AND HISTOLOGICAL STUDIES.** *(Ger.)* Eichholtz, W. (Inst. Path., U. Göttingen, Germany) and W. Eger. *Med Welt* 18(2):119-124, 1968.

8609 **INFLAMMATION OF THE BILIARY TRACT DURING VIRAL HEPATITIS IN CHILDREN.** *(Rus.)* Shilenok, I. G. (Krasnoyarsk Med. Inst., USSR) and V. P. Politova. *Pediatriia (Moskva)* 46(10):67-68, 1967.

8610 **STUDIES OF HEMAGGLUTINATION IN CHILDREN WITH HEPATITIS, USING TANNIN-TREATED ERYTHROCYTES.** *(Hun.)* Dóbiás, G. (Inst. Postgrad. Med., Budapest, Hungary), E. Perényi, G. Vadász and T. Fodor. *Kiserl Orvostud* 20(3):248-251, 1968.

8611 **THE CLINICAL PICTURE, PROPHYLAXIS AND PROGNOSIS OF INFECTIOUS HEPATITIS AND OTHER VIRAL DISEASES OF THE LIVER.** *(Rus.)* Tareiev, Ie. M. (Inst. Ther., Acad. Med. Sci. USSR, Moscow). *Sovet Med* 31(3):3-14, 1968.

8612 **DYNAMICS OF THE ACTIVITY OF SOME ISOENZYMES IN PATIENTS WITH INFECTIOUS HEPATITIS.** *(Rus.)* Fedianin, Iu. P. (Crimean Med. Inst., Simferopol', USSR). *Sovet Med* 31(3):44-47, 1968.

8613 **THE ETIOPATHOGENESIS OF POST-HEPATITIC PATHOLOGY.** *(E.)* Turchetti, A. (Med. Clin., U. Palermo, Italy). *Panminerva Med* 9(12):570-584, 1967.

8614 **NEUROLOGIC COMPLICATIONS DURING VIRAL HEPATITIS.** *(It.)* Arciello, G. (D. Cotugno Hosp., Infect. Dis., Italy), F. Pandico and G. Tallarino. *Riforma Med* 81(36):988-990, 1967.

8615 **HERPES SIMPLEX VIRUS HEPATITIS IN MICE: AN ELECTRON MICROSCOPIC STUDY.** *(E.)* Rabin, E. R. (Baylor U. Coll. Med., Houston, Texas), A. B. Jenson, C. A. Phillips and J. L. Melnick. *Exp Molec Path* 8(1): 34-48, 1968.

8616 **RAT VIRUS DISEASE, AN EXPERIMENTAL MODEL OF NEONATAL HEPATITIS.** *(E.)* Margolis, G. (Dartmouth Med. Sch., Hanover, N. H.), L. Kilha and P. R. Ruffolo. *Exp Molec Path* 8(1):1-20, 1968.

8617 **PROTEIN, SUGAR AND CHOLESTEROL CONCENTRATION IN THE BLOOD OF PREC NANT WOMEN WITH INFECTIOUS HEPATITIS.** *(Rus.)* Yakobova, A. N. (Samarkand Med. Inst., USSR). *Akush Gin (Moskva)* 43(11):40-42, 1967.

8618 **OVARIAN-ADRENAL RELATIONS IN FEMA PATIENTS SUFFERING FROM INFECTIOUS HEPATITIS.** *(Rus.)* Yatsenko, L. Ya. (Crimean Med. Inst., Simferopol, USSR). *Akush Ginek (Moskva)* 44(2):69-71, 19

8619 **EPIDEMIC VIRAL HEPATITIS IN THE INFA** *(Fr.)* d'Oelsnitz, M. (Pasteur Hosp., Nice, Fran V. Robini, M. Plissier, A. Borja and J. M. Vaillant. *Pediatrie* 22(8):903-909, 1967.

8620 **VIRAL HEPATITIS IN PATIENTS WITH MALIGNANT TUMORS AND SYSTEMIC DISEASES.** *(Rum.)* Gavrilă, I. (Inst. Med. Pharmacol., Cluj Romania), N. Opreanu and R. Ardeleanu. *Med Intern (Bucu* 19(10):1205-1212, 1967.

8621 **RELAPSES OF VIRAL HEPATITIS.** *(Rus.)* Aleksandrova, V. R. (Sanit. Hygiene Med. Inst. Leningrad, USSR), S. A. Smirnova and K. G. Kapetanaki. *Ter Arkh* 39(11):118-121, 1967.

8622 **EPIDEMIOLOGY OF VIRAL HEPATITIS.** *(S* Ramirez S., E. (Obrero Hosp., Cerro de Pasco, Peru). *Rev Med Hosp Obrero* 16(3-4):229-240, 1967.

8623 **THE RESORPTION OF ^{131}I-LABELLED ALBUMIN IN INFECTIOUS HEPATITIS.** *(Rum.* Kelemen, L. (Inst. Med. Pharm., Târgu-Mureş, Romania), S. Csőgőr, I. Hirschfeld and R. Ionescu. *Rev Medicochir Iasi* 71(3):559-562, 1967.

8624 **THE INCIDENCE OF VIRAL HEPATITIS WITH REGARD TO ABO BLOOD TYPES AN RHESUS FACTOR.** *(Rum.)* Pruteanu, P. (Inst. Med. Pharr Iasi, Romania), A. Viţă, T. Wexler, M. Ulianov, G. Cuciurea T. Hurmuzache and T. Schwartzenberg. *Rev Medicochir Ias* 71(4):941-947, 1967.

8625 **TREATING PATIENTS WITH RESIDUAL MANIFESTATIONS OF VIRAL HEPATITIS AT A PYATIGORSK RESORT.** *(Rus.)* Osipov, Yu. S. (Ins Balneol. Physiother., Pyatigorsk, USSR). *Vop Kurort Fiziot* (1):11-15, 1968.

8626 **TRANSAMINASE ACTIVITY IN THE BLOOI OF PATIENTS WITH RESIDUAL MANIFEST TIONS OF VIRAL HEPATITIS DURING TREATMENT AT HEALTH RESORT.** *(Rus.)* Kulikova, G. F. (Inst. Balneol. Physiother., Pyatigorsk, USSR). *Vop Kurort Fizioter* (1):16 21, 1968.

8627 **EVOLVED FORMS OF VIRAL HEPATITIS.** *(Por.)* Vasconcellos, D. (Inst. Gastroent., Reci

razil), F. L. Castro and F. M. Reis. *Arq Gastroent* 4(2):111-
19, 1967.

628 **HEPATITIS, PROBABLY DUE TO TRANSFUSIONS, IN TWO YOUNG INFANTS, A TWIN
OY AND GIRL.** *(Fr.)* Monnet, P. (E. Herriot Hosp., Lyon,
rance), D. Rosenberg and A. Jouvenceaux. *Pediatrie* 22(6):
99-703, 1967.

8629 **EPIDEMIOLOGICAL ASPECTS OF INFECTIOUS HEPATITIS IN THE MILAN PROVINCE.** *(It.)* Vezzoso, B. (Min. Sanit., Milan, Italy) and V. Molese. *Gazz Sanit* 38(6):266-272, 1967.

8630 **EPIDEMIOLOGICAL COURSE OF VIRAL HEPATITIS IN ITALY.** *(It.)* Graziano, F. (Min. Sanit., Rome, Italy) and C. Cocchini. *Gazz Sanit* 38(7/8):349-355, 1967.

See also: 7867,7883,7963,8587

8631 **TISSUE IRON AND HEMOCHROMATOSIS: COMPARATIVE STUDIES IN DENVER AND BOSTON.** *(E.)* MacDonald, R. A. (U. Colorado Sch. Med., Denver). *Arch Path* 84(5):543-551, 1967.

Hemosiderin was studied histologically and measured chemically in the organs of 150 unselected autopsied persons over the age of 15 (16-91 yr old) at the University of Colorado Medical Center during a 4-month period in 1966. Comparisons were made with previous studies in Boston. In Denver, 69% of persons had stainable iron in the liver, comparable to the 70% in Boston. Patients in Denver had more total hepatic iron than patients in Boston: those without cirrhosis in Denver had an average of 372 mg (in Boston, 226 mg). Patients with cirrhosis in Denver had 380 mg; in Boston, 250 mg. Although cirrhotics did not have more hepatic iron than persons without cirrhosis, they more often had the histological features of hemochromatosis: stainable iron deposits in the parenchymal cells of the pancreas and heart. This was found in 18% of cirrhotics compared with 2% of noncirrhotics in Denver and in 33% compared with 2% in Boston. Patients with cirrhosis should be studied further to elucidate factors responsible for abnormal iron storage.

8632 **EXTRACORPOREAL PORTOSYSTEMIC SHUNT FOR PORTAL HYPERTENSION.** *(E.)* White, J. J. (Royal Victoria Hosp., Montreal, Quebec, Canada), M. Slapak and L. D. MacLean. *Surgery* 63(1):17-28, 1968.

Extracorporeal portosystemic shunts were constructed in 2 patients with portal hypertension: a 64-yr-old atherosclerotic male and a 53-yr-old male with massive, gastrointestinal bleeding. After large-bore catheters placed in the portal vein via the umbilical vein and in the superior vena cava via the external jugular vein had been connected with silicone rubber tubing, portal pressures were reduced by 15-20 cm H_2O with shunt blood flows of 250-400 ml/sec. Unshunted portal pressure, central venous pressure and shunted portal pressure were consistently parallel. However, there was little correlation between these pressures and the pulse and blood pressure, which generally reflected the current central hemodynamic status. The minimal morbidity, the ease of construction and the promising preliminary observations with extracorporeal portosystemic shunts for portal hypertension encourage further attempts at this procedure.

8633 **CRITICAL ASSESSMENT OF THE RATIONALE OF THORACIC DUCT DRAINAGE IN THE TREATMENT OF PORTAL HYPERTENSION.** *(E.)* Warren, W. D. (U. Miami Sch. Med., Fla.), J. J. Fomon and C. A. Leite. *Surgery* 63(1):7-16, 1968.

Possible effects of thoracic duct drainage were studied in 6 patients presenting with liver cirrhosis, ascites and portal hypertension; these included 3 patients in whom the ducts were occluded and 3 in whom the catheter was introduced through the internal jugular vein and passed through the junction of the 2 structures into the duct. The hepatic vein was also catheterized, and the wedge hepatic vein pressure and central venous pressure (CVP) were measured. In the 3 patients with occluded thoracic ducts the portal hypertension was not relieved by drainage. The average CVP was 8 mm Hg and the thoracic duct pressure was 34 mm Hg. In the 3 unoccluded patients the average CVP was 12 mm Hg and the thoracic duct pressure was also 12 mm Hg; no drainage was attempted. The theoretical basis of thorac duct drainage for the treatment of portal hypertension is discussed and concluded to be erroneous. No evidence was found to support the hypothesis of a relative obstruction at the thoracic duct-subclavian junction, there was no pressure gradien across this junction, and there was no correlation between thoracic duct pressures and portal vein or intrahepatic pressures

8634 **EFFECT OF THORACIC DUCT DECOMPRESSION ON ASCITES.** *(E.)* Fish, J. C. (U. Texas Galveston), H. E. Sarles, A. R. Remmers Jr. and R. D. Williams *JAMA* 203(2):98-102, 1968.

Four patients (1 male and 3 females, 38-59 yr old) with cirrhosis and ascites were studied under careful conditions of metabolic balance during thoracic duct drainage and reinfusion of lymph. Decompression alone did not change the degree of ascites. When a negative water and sodium balance was produced, such as by dialysis of the lymph, a corresponding reduction in ascites was noted. This effect was similar to the results of paracentesis, diuretics, or ascites autotransfusion. In 1 patient, ascites autotransfusion resulted in a 4-fold increase in the lymph flow rate. It is postulated that the accelerated lymph flow resulted from an expansion of the plasma volume by ascites, which resulted in an increased blood flow to the liver and an increased transudation of lymph at the postsinusoidal block.

8635 **THE PROGNOSTIC IMPLICATIONS OF ALTERED PHYSIOLOGY IN OPERATIONS FOR PORTAL HYPERTENSION.** *(E.)* Siegel, J. H. (Albert Einstein Coll. Med., Bronx, N. Y.), M. Greenspan, J. D. Cohn and L. R. M. Del Guercio. *Surg Gynec Obstet* 126(2):249-262, 1968.

Cardiovascular hemodynamics, oxygen consumption, and acid base balance were evaluated in 51 patients with cirrhosis and portal hypertension and compared with those previously reported from 30 patients in septic shock, 16 in nonseptic shock and 13 noncirrhotics without shock. The common circulatory abnormality complicating surgical therapy was the occurrence of a decreased cardiac output. However, in patients with septic shock and in those with cirrhosis and portal hypertension, a hyperdynamic state was often found, characterized by a decrease in net vascular tone, diminished peripheral oxygen extraction in the presence of an increased cardiac output, increased pulmonary venoarterial admixture, and the development of some degree of myocardial depression. The data suggest that the increased, but ineffective, circulation is secondary to arteriovenous shunts in both systemic and pulmonary circulations. Evaluation of the level of effective oxygen transport as a function of the degree of abnormality in net vascular tone revealed a zone of maximum risk, which was associated with a high mortality. Survival of cirrhotics undergoing electiv portal decompression was shown to depend on the level of their hyperdynamic state. Inclusion in the zone of maximum risk was associated with a 70% operative mortality rate, as

ontrasted with a 5% mortality rate for those patients outside
ie zone. The hyperdynamic group of patients, from either
iuse, are prone to develop high-output cardiac failure. The
se of an inotropic agent can influence prognosis by enabling
iese patients to compensate for the demands upon their ab-
ormal peripheral circulation.

636 **THE INFLUENCE OF KIDNEY OR LIVER
DISORDERS ON THE SERUM CONCENTRA-
ION AND URINARY EXCRETION OF ^{3}H-PERUVOSIDE,
TRITIUM-LABELLED CARDIAC GLYCOSIDE.** *(E.)*
ahrtz, H. (Dept. Pharmacol., Christian-Albrechts U., Kiel,
ermany) and P. A. Van Zwieten. *Europ J Pharmacol* 3(2):
47-152, 1968.

he elimination of ^{3}H-peruvoside was studied in 3 different
ypes of patients: 1) those with normal renal, hepatic, and
ırdiac functions; 2) those with renal failure; and 3) those with
ver disease (either cirrhosis or hepatitis). After i.v. injection
f ^{3}H-peruvoside, total radioactivity of serum and urine was
etermined during a 48-hr period. In control patients 2 differ-
nt half-lives (20 min and 50-60 hr, resp.) were established for
ie elimination of serum radioactivity. In patients with liver
isease, serum radioactivity elimination was no different from
iat in the controls. Renal failure, however, caused a consider-
ɔle slowing in the elimination of radioactivity from the serum.
fter 48 hr, about 40% of the administered radioactivity had
een excreted via the kidney in control patients. Patients suf-
ering from liver disease, however, excreted about 70% of the
jected radioactivity in the urine. The pathologically dimin-
hed excretion of the drug in the bile of these patients is prob-
ɔly compensated for by an increased urinary excretion. The
rinary excretion was reduced to 12% in patients with renal
ilure.

637 **LIPIDS IN THE SERUM, LIVER, AND
ADIPOSE TISSUE IN HEPATIC CIRRHOSIS.**
t.) Guala, L. (Inst. Med. Semeiotics, U. Naples, Italy), T.
e Leo and I. Caccia-Perugini. *Fegato* 13(2):179-195, 1967.

as-liquid chromatography was used to determine total fat,
hospholipids, neutral fats, free cholesterol, steroids, glycerides,
ıd the percentages of the principal fatty acids in total fats in
ie serum, liver, and mesenteric fat in a patient with atrophic
rrhosis and splenogenic panmyelophthisis and in a normal con-
ol. Lipids in the liver and mesenteric fat were determined on
iaterial biopsied during surgery. In addition to a decrease in
ɔtal serum lipids, the liver of the cirrhotic patient had low
ɔntents of phospholipids, free cholesterol, and some of the es-
ential fatty acids, particularly linoleic and arachidonic acids,
hile increases occurred in steroids and glycerides. Parallel
ianges occurred in serum, liver, and mesenteric fat deposits.
hese disorders in lipid metabolism are probably responsible
ɔr the development of cirrhosis from fatty liver.

638 **TREATMENT OF HEPATIC COMA IN CIR-
RHOSIS BY PLASMAPHORESIS AND PLASMA
NFUSION (PLASMA EXCHANGE).** *(E.)* Sabin, S. (Boston
ity Hosp., Mass.) and J. A. Merritt. *Ann Intern Med* 68(1):
-7, 1968.

lasma exchange was performed in 3 patients (2 females and
male, 49-64 yr old) with severe hepatic coma secondary to proved cirrhosis of the liver. The patients were in coma for 72 hr despite therapy consisting of protein restriction, high carbohydrate intake, neomycin sulfate, and electrolyte replacement. Plasma was exchanged by removing blood from a catheter in the brachial artery and infusing the patient's own red cells and fresh-frozen plasma through a venous catheter in the opposite arm. The amount of plasma exchanged varied from 1-3½ liters/day. All 3 patients showed marked improvement after plasma exchange, but died a few days to several weeks later of complications of their chronic liver disease. One patient underwent this procedure on 3 separate occasions and responded each time. The 2nd patient, who also had the hepatorenal syndrome, demonstrated striking improvement in mental status after plasma exchanges although his renal status continued to deteriorate. The 3rd patient, who demonstrated decerebrate posturing, fixed and dilated pupils, and extensor plantar reflexes before plasma exchange, became oriented and alert after exchange. Blood ammonium levels showed a reduction after plasma exchange in the 1 patient in whom these determinations were made. The blood urea nitrogen, electrolytes, calcium, and phosphorus were unchanged, but a decrease in the serum bilirubin and alkaline phosphatase was noted after exchange. Plasma exchange was not associated with complications in any of the 3 patients.

8639 **THE STATE OF THE BLOOD COAGULATION SYSTEM IN CHILDREN WITH CIRRHOSIS OF THE LIVER.** *(Rus.)* Ryzhkova, L. A. (Inst. Pediat., Acad. Med. Sci. USSR, Moscow). *Pediatriia* 47(2):29-33, 1968.

8640 **PORTAL HYPERTENSION DURING BILIARY CIRRHOSIS: REPORT OF SIX SURGICAL CASES.** *(Fr.)* Viard, H. (Ed. Herriot Hosp., Lyon, France), E. Saubier and P. Mikaeloff. *Ann Chir* 21(23/24):1401-1407, 1967.

8641 **THE INDICATIONS FOR ANABOLIC STEROIDS IN THE TREATMENT OF CHRONIC HEPATIC LESIONS.** *(Fr.)* Blahniková, L. (Inst. Res. Human Nutrit., Prague, Czechoslovakia) and J. Horký. *Gastroenterologia* 107(6):354-359, 1967.

8642 **JUVENILE HEPATIC CIRRHOSIS SIMULATING CONGENITAL HEART DISEASE. [CASE REPORT]** *(E.)* Desbaillets, P. (Pediat. Clin., U. Lausanne, Switzerland), A. Merminod, R. Mosimann and J. L. Rivier. *Cardiologia* 51(3):170-176, 1967.

8643 **STUDY OF FIBRINOLYSIS INHIBITORS IN PATIENTS WITH CIRRHOSIS.** *(Rum.)* Teodorescu, P. (Inst. Med. Pharmacol., Bucharest, Romania), M. Lilis, A. Câmpeanu, R. Ionăşescu and F. Ionescu-Stoian. *Med Intern (Bucur)* 19(11):1283-1290, 1967.

8644 **ADRENAL AND THYROID AUTOANTIBODIES IN A PATIENT WITH "IDIOPATIC" ADDISON'S DISEASE AND LIVER CIRRHOSIS ASSOCIATED WITH CANCER: CASE REPORT.** *(It.)* Ziliotto, D. (Inst. Med. Symptomatol., U. Padua, Italy), I. Mastrogiacomo and P. Borini. *Policlinico [Med]* 74(5):235-243, 1967.

8645 **DISEASES ASSOCIATED WITH AND CAUSES OF DEATH IN HEPATIC CIRRHOSIS.** *(Ger.)* Theuer, D. (Charité Hosp., Berlin, Germany), H. B. Zimmermann and I. Jacobus. *Deutsch Gesundh* 22(45):2116-2121, 1967.

8646 **A CASE OF *CRYPTOCOCCUS* INFECTION SUPERIMPOSED ON CIRRHOSIS OF THE LIVER.** *(Turk.)* Koçak, N. (Med. Fac., U. Istanbul, Turkey), M. Yenerman, Y. Ergün and E. Özdoğan. *Turk Tip Cem Mec* 33(10):651-654, 1967.

8647 **LIVER SHUNTS.** *(E.)* Viamonte, M. Jr. (U. Miami Sch. Med., Fla.), L. Martinez, R. E. Parks, W. D. Warren and J. Fomon. *Amer J Roentgen* 102(4):773-775, 1968.

8648 **PANCREATIC INVOLVEMENT IN LIVER CIRRHOSIS.** *(Jap.)* Sato, T. (Tonan Hosp., Sapporo, Japan), A. Hirayama, T. Higuchi, M. Sawada, M. Kudo, T. Nishikawa and K. Ichikawa. *Med J Mutual Aid Ass* 17(3):3-36, 1968.

8649 **ESTRADIOL METABOLISM IN CIRRHOSIS.** *(E.)* Zumoff, B. (Montefiore Hosp., New York, N. Y.), J. Fishman, T. F. Gallagher and L. Hellman. *J Clin Invest* 47(1):20-25, 1968.

8650 **SPONTANEOUS HEMOPERITONEUM IN LIVER CIRRHOSIS. (PATHOGENIC ASPECTS).** *(Rum.)* Schächter, A. (Bucharest Munic. Emerg. Hosp., Rumania), N. Mihăilescu, F. Köppich and G. Vlad. *Med Inte (Bucur)* 19(10):1213-1216, 1967.

8651 **CIRRHOSIS OF THE LIVER. REVIEW OF 79 CASES.** *(Sp.)* Sabonge, R. (Santo Tomás Hosp Panama, Rep. Panama). *Arch Med Panamen* 15(1-2-3):23-33, 1966.

8652 **CLINICAL AND BIOCHEMICAL STUDY CONCERNING THE THERAPEUTIC EFFECT OF THE DIURETIC DRUG TRIAMTEREN IN ASCITOGENIC CIRRHOSIS.** *(Rum.)* Páunescu-Podeanu, A. (Carol Davila Hosp., Bucharest, Romania), H. Demirian, C. Ioniță, D. Georgescu, A. Tarachiu, R. Anghel and L. Rubingher. *Med Intern* 20(5):523-530, 1968.

8653 **A CLINICAL AND STATISTICAL STUDY OF 238 CASES OF CIRRHOSIS OF THE LIVER.** *(Sp.)* Meeroff, M. (Natl. Ministry Soc. Assistance, Buenos Ai Argentina), A. N. Pelizzeeri, R. Celestino, BDO. Feldfeber, L. Pinchuk, A. Folino, A. Alicino, J. Cestoni, BDO. Bekier, J. Goschinsky, S. Aschkenazi, A. M. Luis, J. C. Meeroff, M. Rodovsky, O. Mayorga, J. M. Gregorio and J. Mandelman. *Medicina (Mex)* 47(1024):543-547, 1967.

8654 **CIRRHOSIS OF THE LIVER RESULTING FROM CARBON TETRACHLORIDE: THE POSSIBILITY OF REVERSION AT VARIOUS INTERVALS BY MEANS OF PARTIAL HEPATECTOMY.** *(It.)* Marrano, D. (Inst. Spec. Surg. Path. Clin. Training, U. Bologna, Italy), R. Bazzocchi and F. Maghetti. *Acta Chir Ital* 22(Suppl. 3): 9-139, 1966.

See also: 7722,7761,7858,7984,7996,8428,8541

55 **GALL STONE DYSPEPSIA.** *(E.)* Rhind, J. A. (Pontefract Gen. Infirm., England) and L. Watson. *t Med J* 2(1):32, 1968.

order to determine whether gallstones commonly cause flatu- t dyspepsia, 67 patients presenting with dyspepsia were di- ed into 3 groups: 1) those with gallstones who complained y of dyspepsia and were treated by cholecystectomy; 2) those h gallstones who complained of both dyspepsia and attacks biliary colic and were treated by cholecystectomy; and 3) se with dyspepsia only and no radiological evidence of gall- nes, who were treated only symptomatically. Patients were iewed 1 yr after surgery. The results showed that cholecys- tomy for gallstones cured flatulent dyspepsia in more than f the cases, while less than 25% were not improved. The sence of a hiatus hernia does not lessen in probability of cure less the dominant symptoms indicate gastroesophageal re- x.

56 **ORIGIN OF BACTERIA IN BILEDUCT BILE.** *(E.)* Scott, A. J. (Royal Free Hosp., London, gland) and G. A. Khan. *Lancet* 2(7520):790-792, 1967.

e samples were obtained by direct puncture during cholangi- aphy or laparotomy from 4 groups of patients: 25 patients h chronic calculous biliary disease and jaundice due to cho- ocholithiasis (A) or postoperative biliary stricture (B), and patients with no biliary disease but with jaundice due to nors of the lower common bile duct (C) or hepatic duct (D). cteria were found in 83% of the patients in group A and in in group B, but in none of groups C or D. It is suggested t the cholangitis that complicates choledocholithiasis is re- ed to the preexisting biliary disease. When bacterial cho- gitis has developed, the concentration of aerobic organisms comparable to that in the feces.

57 **BILE ACID CONJUGATION OF LIVER TISSUE FROM PATIENTS WITH ACUTE CHOLECYSTI- S.** *(E.)* Gottfries, A. (Sahlgren Hosp., U. Göteborg, Sweden). *and J Clin Lab Invest* 21(3):249-256, 1968.

he synthesis of bile acid conjugates was studied in liver homog- ates obtained from operative biopsies taken in 25 patients men and 18 women, 18-80 yr old) operated on for acute olecystitis. Eleven of these cases were given preoperative fusions of carbohydrates. In the 14 patients not given carbo- ydrates a significant decrease in the capacity to synthesize le acid conjugates was found as compared to the control oup. In the 11 patients treated with carbohydrates the syn- esizing capacity was almost restored to the level of the con- ols. For patients not given carbohydrates, a significant nega- ve correlation was found between the synthesizing capacity nd the retention of BSP. It is concluded that at least one liver nction is affected in acute cholecystitis. The degree of reten- on of BSP can serve as a measure of the impairment of this nction. Preoperative infusion of carbohydrates improves the onjugating capacity of the liver.

658 **EXPERIMENTAL ANASTOMOSES OF THE COMMON BILE DUCT.** *(E.)* Klopper, P. J. Wilhelmina Hosp., U. Amsterdam, Netherlands), K. A. Kelly nd A. Vos. *Surgery* 63(3):459-463, 1968.

The common bile duct of 30 dogs was transected midway between the duodenum and its junction with the duct from the right lateral lobe of the liver. Continuity was restored by end-to-end anastomosis performed with the American vascular stapler, Nakayama's instrument for small-vessel anastomosis, or by hand with catgut, stainless steel wire, or vascular silk. The results of the operations were assessed by means of liver function tests during surgery, and by cholangiographic, histologic, and vascular injection techniques 3-6 months later. The results showed the superiority of the American vascular stapler in achieving the best functional and morphologic results. The use of silk was also satisfactory, but poor results were obtained with stainless steel wire, catgut, and Nakayama's instrument. Minimum interference with vascularization of the anastomosis appeared to be an important factor in the success of the American vascular stapler.

8659 **PANCREATOGENIC CHOLECYSTITIS.** *(Rus.)* Mamamtavrishvili, D. G. (Tiflis Inst. Postgrad. Med., USSR). *Sovet Med* 31(6):79-81, 1968.

Among 120 patients with acute and chronic cholecystitis but no symptoms of pancreatitis, 38 had sterile bile and many of these had elevated biliary amylase levels. Based on a detailed clinical, anatomo-pathological and bacteriological study of such patients, aseptic cholecystitis is concluded to be a concrete form of the cholecysto-pancreatic syndrome which is favored by biliary hypertension and the reflux of pancreatic enzymes into the bile ducts. When both pancreatic enzymes and infection are present, various complications involving the liver and bile ducts often occur. In bacterial forms of cholecystitis immediate surgery is indicated; in simple pancreatogenic cholecystitis, however, intravenous administration of procaine (30 drops/min of a 0.2% solution) and Trasylol often yields good results. Procaine decreases the reflex spasm in the bile ducts, normalizes the function of the sphincter of Oddi; reduces biliary hypertension, and improves tissue nutrition in the gallbladder, liver and bile ducts. Trasylol inhibits pancreatic enzyme action, reduces abdominal shock, and prevents bleeding around damaged tissues. Opium derivatives are to be avoided since they retard evacuation of the stomach and increase spasm in the sphincter of Oddi. Antibiotics are indicated only if chills and fever are present. Among 52 patients treated recently for aseptic cholecystitis, only 12 were operated on immediately (of whom 5 died of bile peritonitis) while 40 were treated with procaine and Trasylol; 35 of these 40 could be discharged as cured, while 5 required subsequent surgery. It is felt that conservative treatment can reduce mortality by 50% in pancreatogenic cholecystitis patients who would formerly have been operated.

8660 **CHRONIC CHOLECYSTITIS: EXPERIENCE IN A LARGE CHARITY INSTITUTION.** *(E.)* De Marco, A. (Louisiana State U. Sch. Med., New Orleans), F. C. Nance and I. Cohn Jr. *Surgery* 63(5):750-756, 1968.

A review of the records of 695 patients (91 males and 604 females, 9-95 yr old, average 50.5 yr; 374 Caucasians and 321 Negroes) undergoing elective cholecystectomy at Charity Hospital in New Orleans during the period 1957-60 revealed that symptoms had been present for an average of 2.8 yr. Stones were found in the gallbladder in 96% and in the common bile in 13.8%, and were revealed by cholecystography in 61%. Cholecystectomy was used alone in 394 cases and was combined with common bile duct exploration in another 201, 48% of whom had stones. Complications developed postoperatively in only 18%, and the overall mortality was 1.3% (0.8% for cholecystectomy alone and 2.5% for combined procedures). The low incidence of bile duct injuries and low rate of retained stones are emphasized. The mortality rate for patients over 60 (3.2%) was 8 times as high as that for patients under this age (0.4%), emphasizing that younger patients tolerate this type of surgery better, have fewer complications, and should have better results. Of the 686 survivors, 654 had no further symptoms and only 12 had serious recurrence. A comparison of the results of emergency versus elective cholecystectomy for cholecystitis indicated the superiority of the elective procedure. In view of the natural history of the disease, the low mortality rate, and the satisfactory long-term results, early cholecystectomy is strongly recommended for patients with chronic cholecystitis.

8661 MORBIDITY OF SURGICAL TREATMENT FOR NONMALIGNANT BILIARY TRACT DISEASE. *(E.)*

Glenn, F. (New York Hosp.-Cornell Med. Ctr., N. Y.), C. K. McSherry and P. Dineen. *Surg Gynec Obstet* 126(1):15-26, 1968.

A retrospective analysis of 3217 biliary tract operations performed over a 16-yr period revealed an incidence of nonfatal complications of 6.9%; 120 or 46.2% of the complications were directly related to the biliary tract. The leading cause of morbidity after biliary tract surgery was infection, principally of the wound. Pulmonary disorders, cardiovascular disease, urologic complications, and retained common duct stones accounted for the other principal sources of morbidity.

8662 CHOLEDOCHAL DYNAMICS IN MAN. *(E.)*

Schein, C. J. (Montefiore Hosp. Med. Ctr., New York, N. Y.) and T. C. Beneventano. *Surg Gynec Obstet* 126(3):591-596, 1968.

In 20 cholecystectomized patients, the intubated common bile duct was studied by cinemanometric cholangiography; this permitted observation of the dynamic aspects of sphincter function, the mechanism of pancreatic duct filling, ductal size, passage pressure, and pain production under normotensive and acutely produced hypertensive conditions. The passage pressure is defined as the minimal intracholedochal tension required to activate the contractile systolic and filling diastolic functions of the distal portion of the common bile duct. Each phase of the cycle was prolonged in the hypertensive state. Retrograde pancreatic duct filling was accomplished in 9 patients; the pancreatic filling pressure was always greater than that patient's own passage pressure, but less than that required to produce maximal ductal size or to induce pain. As a function of intraductal tension, the common bile duct could be reversibly distended to a width 250% of its base-line size. Acute abdominal pain could be induced in all persons when the intracholedochal pressure was raised to produce maximal ductal dilatation. The dynamic changes observed in acutely produced choledochal hypertension were promptly reversed when the intraductal tension was reduced to the basal level.

8663 CHOLECYSTECTOMY IN THE AGED. *(E.)*

Ibach, J. R. (Taylor Hosp., Ridley Park, Pa.), H. A. Hume and W. H. Erb. *Surg Gynec Obstet* 126(3):523-528, 1968.

A total of 151 patients over 60 yr of age underwent cholecystectomy during the 5-yr period 1962-1966. Approximately half the patients were operated on for chronic cholecystitis and half for acute cholecystitis. Six deaths resulted, for an overall mortality of 4%. Elective operations were tolerated well, with a 2.3% mortality rate, while emergency operations carried a much higher mortality rate of 16.7%. No deaths resulted in patients with uncomplicated chronic calculous cholecystitis. Deaths were confined to patients who underwe an emergency cholecystostomy for acute, perforated, or gangrenous cholecystitis and to those patients with common bile duct stones. The results of this study indicate that in the elderly, elective cholecystectomy for chronic calculous cholecystitis is a safe operation with a low mortality rate; however, when an emergency operation of the biliary tract is required, the mortality rate rises alarmingly.

8664 EXPERIMENTAL CHOLELITHIASIS IN THE RABBIT INDUCED BY CHOLESTANOL FEEDING: EFFECT OF NEOMYCIN TREATMENT ON BILE COMPOSITION AND GALLSTONE FORMATION. *(E.)*

Hofmann, A. F. (Rockefeller U., New York, N. Y.), V. Bokkenheuser, R. L. Hirsch and E. M. Mosbach. *J Lipid Res* 9(2):244-253, 1968.

It is well known that dietary cholestanol is converted by the rabbit to 5α-bile acids which coprecipitate with the normally occurring 5β-bile acids to form gallstones composed of calciun and sodium glycoallodeoxycholate and glycodeoxycholate. A study in cholestanol-fed (1%) rabbits showed that the oral administration of large doses (0.5 g/day p.o.) of neomycin prevents gallstone formation and reduces the elevated concentration of allodeoxycholic acid in bile, with a reciprocal increase in allocholic acid concentration. The reduction in the concentration of allodeoxycholic acid and in the incidence of gallsto was proportional to the dose of neomycin; at a concentration of allodeoxycholic acid below 20% of total bile acids, gallston formation did not occur. Neomycin probably exerts its actio by modifying the anaerobic intestinal flora which dehydroxyl allocholic acid to allodeoxycholic acid; this suggests that both hepatic and bacterial transformations are essential steps in the pathogenesis of cholestanol-induced cholelithiasis.

8665 EXPERIMENTAL BILIARY LITHIASIS PRODUCED BY SLOW STENOSIS OF THE TERMINAL PART OF THE COMMON BILE DUCT. *(It.)*

Mattioli, F. (Inst. Gen. Surg. Clin., U. Genoa, Italy), L. Guerr and G. C. Torre. *Pathologica* 59(875/876):207-218, 1967.

A comparison was made of biliary lithiasis produced in male rabbits by wrapping the common bile duct, just before it ente the duodenum, with cellophane soaked in a saturated aqueous solution of sodium dicetylphosphate or with a ring of Ameroi a plastic which swells on contact with water or body fluids, i mersed in mineral oil. One animal in each group died of unknown causes 17 and 20 days after surgery. Autopsy and hist logical findings were the same in both groups. Cholelithiasis developed in 3 of 5 animals treated with cellophane and sodiu etylphosphate and in 4 of 5 animals treated with Ameroid. e gallstones consisted of a protein nucleus covered with a ntle of bile salts and pigments. Gallstones were either spheri- and unattached to the gallbladder or polypoid and attached its mucosa. It appears that gallstones, when first formed, anchored to the wall of the gallbladder and are then freed. pronounced signs of inflammation were seen in either group, cept for granulomatous areas secondary to necrosis.

66 **PAPILLOTOMY IN THE TREATMENT OF DISEASES OF THE DESCENDING BILIARY ACT.** *(Ger.)* Müller-Beissenhirtz, P. (Munic. Hosp., unschweig, Germany), H. J. Berger and P. C. Alnor. *ngenbeck Arch Klin Chir* 318(3):217-232, 1967.

574 operations performed on the biliary tract between 1963 1 1965, 156 (27.2%) were papillotomies with T-tube drainage. nong the 131 patients undergoing surgery for the 1st time, pillary stenosis was combined with cholelithiasis and choledo- olithiasis in 79, with cholelithiasis in 49, and with cholecysti- in 3. Associated pancreatitis was present in 27 (17.3%) of ese 156 patients. Papillary stenosis was found in 25 of 39 tients who had undergone two or more operations on the iary tract, including 3 patients with choledochoduodenosto- es. All 3 became symptomatic after papillotomy and repair the common bile duct. Histological examination of tissue noved during surgery showed inflammatory changes in 50%, generative changes in 25%, and both kinds of changes in 25%. e operative mortality after papillotomy was 6.9% for patients dergoing surgery for the 1st time, 8.8% in patients with ncreatitis, and 16.0% for patients undergoing repeated surgery. nce the operative mortality was 5.2% among patients who re not subjected to papillotomy during their 1st operation, is concluded that papillotomy does not entail increases in the rgical risk. Nonfatal operative complications included 5 cases cholangitis, 1 subphrenic, and 2 complications not directly ated to surgery. None of these patients developed pancreati- after papillotomy.

67 **ANOMALIES IN THE MAIN BILE DUCTS. 506 CASES OF BILIARY DISEASE STUDIED ITH SURGICAL CHOLANGIOGRAPHY.** *(It.)* Bellucci, M. nst. Gen. Surg. Clin., U. Perurgia, Italy). *Ann Fac Med Chir rugia* 59(1):105-116, 1967.

f 506 patients with biliary diseases who were subjected to rgical cholangiography, 16 (3.1%) had accessory hepatic cts and 2 (0.39%) had cystic ducts with anomalous openings. ccessory hepatic ducts opened into the common hepatic duct 12 cases, into the gallbladder in 2 cases, and into the cystic ct in 2 cases. In one case the cystic duct was double, open- ; into the common bile duct and duodenum and in another se the cystic duct had an anomalous opening into the right patic duct. The angle between the cystic duct and common e duct was obtuse instead of acute in 0.39% and was very ute so that the cystic duct ran almost parallel to the common e duct in 11%. In another 20.31% the cystic duct was isted and convoluted. The common bile duct opened norm- y in 78% but opening into the duodenum near the superior odenal flexure in 5%, into the descending duodenum in 10%, l into the 3rd part of the duodenum in 7%.

8 **PERSONAL EXPERIENCE WITH LIPOIDOSIS OF THE GALLBLADDER.** *(Fr.)* Leonard, P. (157 Brand Whitlock Blvd., Brussels, Belgium). *Actualites Hepatogastroent Hotel Dieu* 4(1):B1-B10, 1968.

8669 **THE SURGICAL TREATMENT OF INTERNAL BILIARY FISTULAS.** *(Rus.)* Topchiashvili, Z. A. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Khirurgiia (Moskva)* 44(3):51-55, 1968.

8670 **THE MANAGEMENT OF ACUTE INFLAMMATORY DISEASES OF THE BILIARY TRACT.** *(Rus.)* Ivanov, P. A. (Novosibirsk Med. Inst., USSR). *Khirurgiia (Moskva)* 44(3):37-41, 1968.

8671 **ACUTE CHOLECYSTITIS AND BILIARY PERITONITIS PRODUCED BY *TAENIA SAGINATA*.** *(Rus.)* Adamiia, G. (Tiflis Inst. Postgrad. Med., USSR) and T. G. Gogotishvili. *Sovet Med* 31(3):126-127, 1968.

8672 **INCOMPETENCE OF VATER'S PAPILLA AS A REASON FOR UNSATISFACTORY RESULTS OF CHOLECYSTECTOMY.** *(Rus.)* Grishkevich, E. V. (A. V. Vishnevskii Inst. Surg., Acad. Med. Sci. USSR, Moscow), M. V. Danilov and A. M. Grishkevich. *Sovet Med* 31(3):92-96, 1968.

8673 **EXPERIENCE WITH THE COMBINED USE OF TETRACYCLINE AND GLUCOSAMINE IN THE TREATMENT OF CHOLECYSTITIS.** *(Rus.)* Galkin, V. A. (Central Inst. Postgrad. Med., Moscow, USSR), V. R. Sobolev, V. A. Maksimov and I. S. Molochaieva. *Sovet Med* 31(3):76-78, 1968.

8674 **STENOCARDIA IN PATIENTS WITH OBSTRUCTION OF THE NECK OF THE GALLBLADDER.** *(Rus.)* Dzhavadian, A. M. (N. I. Pirogov 2nd Moscow Med. Inst., USSR) and A. B. Galitskii. *Sovet Med* 31(3):69-75, 1968.

8675 **CARCINOMA OF THE GALLBLADDER: EVALUATION OF 39 CASES.** *(It.)* Proscia, N. (Munic. Hosp., Asti, Italy) and F. Sorisio. *Minerva Chir* 22(19):1074-1077, 1967.

8676 **THE CYSTIC STUMP SYNDROME IN THE ETIOLOGY OF POST-CHOLECYSTECTOMY PAIN.** *(It.)* Rapisarda, L. M. (Inst. Gen. Surg. Clin., U. Palermo, Italy) and M. Impellizzeri. *Minerva Chir* 22(24):1351-1363, 1967.

8677 **FORMATION OF GALLSTONES. II.** *(E.)* Hultén, O. (Dept. Surg., U. Uppsala, Sweden). *Acta Chir Scand* 134(7):561, 1968.

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ANATOMOPATHOLOGICAL STUDY. *(Fr.)* Albot, G. (Hotel Dieu, Paris, France) and P. Delavierre. *Actualites Hepatogastroent Hotel Dieu* 3(2):79-92, 1967.

8681 DYSPLASIAS AND ADENOMAS OF THE GALLBLADDER: SURGERY. *(Fr.)* Hess, W. (Med. Fac., Basel, Switzerland). *Actualities Hepatogastroent Hotel Dieu* 3(2):99-114, 1967.

8682 STONE IN THE COMMON DUCT. *(Rev.) (E.)* Le Quesne, L. P. *J Roy Nav Med Serv* 54(3):190-199, 1968.

8683 A CASE OF DUPLICATION OF THE GALLBLADDER. *(Pol.)* Milik, J. (Distr. Hosp., Nisko, Poland). *Wiad Lek* 21(11):971-973, 1968.

8684 CHOLECYSTOSIS: A CLINICAL AND RADIOLOGICAL SYNDROME. *(Sp.)* Magaldi, P. M. A. R. (Ital. Hosp., Buenos Aires, Argentina), F. Fabrykant, P. Giordano and L. Martino. *Medicina (Mex)* 48(1041):385-391, 1968.

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8686 SURGICAL TREATMENT OF GALLBLADDER LIPOIDOSIS. *(Fr.)* Hepp, J. (Bichat Hosp., Paris, France) and J. Moreaux. *Actualities Hepatogastroent Hotel Dieu* 3(2):34-38, 1967.

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8688 GALLBLADDER LIPOIDOSIS: PATHOLOGICAL ANATOMY AND ETIOPATHOGENESIS. *(Fr.)* Rauber, G. (U. Hosp. Ctr., Nancy, France). *Actualites Hepatogastroent Hotel Dieu* 3(2):14-33, 1967.

8689 CYSTS OF THE EXTRAHEPATIC BILE DUCTS. *(Rus.)* Milonov, O. B. (Sci.-Res. Inst. Clin. Exp. Surg., Min. Hlth. USSR, Moscow), R. L. Dubrovskii and A. A. Umbruminants. *Khirurgiia (Moskva)* 44(3):84-88, 1968.

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8692 THE SURGICAL TREATMENT OF HIGH TRAUMATIC STRICTURES OF THE HEPATIC DUCT. *(Rus.)* Smirnov, Ie. V. (S. M. Kirov Acad. Milit. Med., Lening USSR). *Khirurgiia (Moskva)* 44(5):137-143, 1968.

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8697 HYPERPLASTIC CHOLECYSTOSIS. *(E.)* Murdock, M. G. (Jefferson Med. Coll. Hosp., Philadelphia, Pa.). *J Albert Einstein Med Ctr* 15(3):253-255, 1967.

8698 OBSTRUCTIVE LESIONS OF THE BILIARY TRACT. *(E.)* Meyer, J. (Dayton, Ohio). *J Abd Surg* 10(4):122-123, 1968.

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8703 GALLBLADDER PAPILLOMATOSIS. *(Fr.)* Toulet, J. (Int. Hosp., U. Paris, France). *Actualit Hepatogastroent Hotel Dieu* 3(2):39-54, 1967.

8704 GRANULAR CELL MYOBLASTOMA OF THE CYSTIC DUCT: REPORT OF A CASE WITH ELECTRON MICROSCOPE OBSERVATIONS. *(E.)* Mackay

G. (Dept. Path., U. British Columbia, Vancouver), G. B. ot and J. A. MacDougall. *Canad J Surg* 11(1):44-51, 1968.

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07 **THE POSTOPERATIVE PERIOD IN CHOLECYSTECTOMY.** *(Sp.)* Machicado Z., E. (Fac. d., Cayetano Heredia U., Lima, Peru) and M. Araujo Y. *ad Peru Cir* 20(4):231-236, 1968.

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09 **PSEUDOLITHIASIS IN THE FORM OF PARAVESICULAR CELOMIC CYST.** *(Sp.)* Aguirre, M. (Surg. Dept., San Martin Inst., Buenos Aires, Argentina), Castelletto, C. Castilla and C. Defelitto. *Rev Argent Chir* (3-4):99-101, 1968.

710 **SURGERY OF THE SPHINCTER OF ODDI DUE TO CALCULI OF THE COMMON BILE UCT.** *(It.)* Mattioli, F. (Inst. Gen. Clin. Surg., U. Genoa, aly). *Rass Arch Chir* 5(5):195-212, 1967.

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8713 **THE PROBLEM OF THE INDICATIONS OF CHOLEDOCO-DUODENOSTOMY.** *(Rum.)* Ciobanu, S. (Inst. Med., Timisoara, Romania). *Rev Medicochir Iasi* 71(4):923-930, 1967.

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8715 **PRODUCING BILIARY CALCULI EXPERIMENTALLY BY THE SLOW AND INCOMPLETE STENOSIS OF THE BILE DUCT. I. STENOSIS BY SODIUM DIKETYLPHOSPHATE.** *(It.)* Mattioli, F. (Inst. Gen. Clin. Surg., U. Genoa, Italy), L. Guerra and G. C. Torre. *Rass Arch Chir* 4(4, Suppl. 3):61-63, 1966.

8716 **SURGICAL TREATMENT OF EXTRAHEPATIC OBSTRUCTIVE JAUNDICE.** *(Sp.)* Meriño G., H. (Jose Joaquin Aguirre Hosp. Clin., Vina del Mar, Chile), R. Soumastre C., J. A. V. P. Weitz R., R. Vargas D. and R. Vargas M. *Hosp Viña Mar* 23(3-4):72-76, 1967.

See also: 7878,7884,7886,7894,7932,7964,7973,7974,7977,7979,8051,8058,8152,8156,8166,8171

8717 CLINICAL EXPERIENCE WITH THE USE OF A SINGLE-LAYER INTESTINAL ANASTOMOSIS.

(E.) Olsen, G. B. (U. Alberta Hosp., Edmonton, Canada), E. Letwin and H. T. G. Williams. *Canad J Surg* 11(1):97-100, 1968.

Clinical experience with a technique for single-layer intestinal anastomosis is reported in patients subjected to gastroduodenal (189 cases, mean age 55 yr), small-intestinal (52 cases, mean age 48 yr) or large-intestinal (112 cases, mean age 69 yr) surgery. In this technique, the mesentery is incised so that a pulsating vessel passes to the intestine at the line of resection. The vessels in the submucosa are ligated with fine silk and a single layer of simple silk sutures is placed to include all layers of the intestinal wall. After placing each suture, the bowel edges are inverted by gentle pressure; sufficient tension is applied so that the edges are just approximated without causing ischemia. Other technical factors (choice of suture material, hemostasis, bowel preparation and peritoneal toilet) are important in limiting the intensity of the inflammatory reaction and obtaining optimal healing conditions. Among the patients who had gastroduodenal anastomoses for peptic ulcer, 2 developed major complications, but none of the anastomoses leaked. In the group with small-intestinal anastomoses for a variety of indications, the anastomosis broke down on the 3rd day in 1 patient who died from extensive perihepatic abscess formation. Leakage of the anastomosis occurred in 1 man with large-intestinal surgery (most resections for carcinoma). There was no bleeding from the anastomsis in any of the 3 groups. The combination of one-layer anastomosis and a decompressing tube gastrostomy appeared to be effective in preventing postoperative obstruction. This surgical technique is especially useful in situations where access is difficult.

8718 SOME CLINICAL AND PHYSIOPATHOLOGICAL FEATURES OF LAMBLIASIS (GIARDIASIS): A STUDY ON ADULT PATIENTS.

(Sp.) Olmos, A. (Dept. Med., Concepcion U., Chile), R. Oyarce, G. Domke, F. Bull, F. Biel and G. Torrejón. *Bol Chile Parasit* 23(1/2):48-56, 1968.

Giardia lamblia infestations were present in 58% of 13,000 Chilean patients admitted with gastrointestinal disorders during 1959-64; 90% of these, however, were children. Detailed clinical, radiological, and biochemical studies were made on 67 adults (45 men and 22 women) with lambliasis confirmed by stool examinations. Patients infested with other parasites and those who had other gastrointestinal disorders were excluded. The most common symptoms were diarrhea (42 cases), weight loss (34 cases), asthenia (34 cases), high abdominal pain (20 cases), meteorism (17 cases), and nausea and vomiting (5 cases). Radiological examinations, performed in 31 cases, showed severe duodenitis in 29%, a malabsorption pattern in 15%, and changes in motility in 14%. Findings were normal in 56% and all 31 patients had normal cholecystograms. In determinations made on 27 patients, fat balances were less than 90% in 7 cases and radioiodine-labeled fat absorption ranged from 76.7-86.3%. Two patients excreted more than 1% of ^{131}I-labeled Tolpovidone in their stools. Plasma carotene values were below the normal range in 7 of 29 cases. The results of Schilling's test (vitamin B_{12} absorption) were low in 2 of 18 patients. One of these patients responded to administration of intrinsic factor; he was an alcoholic with atrophic gastritis and histamine-resis tant achlorhydria. Blood hemoglobin values were low in 9 o 17 patients. Intestinal ^{59}Fe absorption was reduced in 4 of 13 patients. Two patients with bleeding hemorrhoids had occult blood in their feces.

8719 SARCOMAS OF THE DIGESTIVE TRACT.

(C Wildner, G. P. (Inst. Cancer Res., Berlin, Germa and K. Klein. *Arch Geschwulstforsch* 32(4):358-379, 1968.

Between 1956 and 1960, 6824 new cases of sarcoma and 80, of carcinoma were reported in the German Democratic Repu The sarcomas included 3110 in males and 3714 in females. If sarcomas of the oral cavity and throat are included, digesti tract sarcomas represented 15% of the total. Of these, 394 (about 40%) were located in the oral cavity and throat; 432 between the esophagus and anus; 81 in the liver, pancreas, gallbladder, and biliary tract; and 72 in the peritoneum, mes tery, and omentum. Of the sarcomas located between the es agus and anus, 55% were found in the stomach, 25% in the small intestine, 11% in the large intestine, and 2.1% in the es agus. The ratio of carcinomas to sarcomas was 380:1 in the rectum, 236:1 in the esophagus, 192:1 in the large intestine, 171:1 in the stomach, 3:1 in the small intestine, 63:1 in the liver, 381:1 in the gallbladder and biliary tract, and 182:1 in the pancreas. Sarcomas of the gallbladder, pancreas, and live occurred more often in women than in men. The incidence sarcomas of the lower gastrointestinal tract generally increase with age, but a relatively large percentage (5 of 38 cases) of liver sarcomas were found in children. Liver hemangiomas d to blood group dyscrasias were found in 10 these 38 patients The relatively large percentage of reticulum cell sarcomas and lymphosarcomas, including undifferentiated round cell sarcon suggests that the lymphatic system plays an important role in the pathogenesis of gastrointestinal sarcomas.

8720 ELECTRON MICROSCOPE STUDY OF ALPH CELL ISLET TUMORS IN THE ZOLLINGER-ELLISON SYNDROME.

(Fr.) Thiery, J. P. (Fac. Sci., Paris, France) and J. P. Bader. *Actualities Hepatogastroent Hotel Dieu* 3(3):184-209, 1967.

Electron microscope studies were made of pancreatic tissue f normal controls and 10 patients with Zollinger-Ellison syndr including 3 with macroscopically visible pancreatic tumors, 4 with small lymph node metastases, 2 with hepatic metastases and 1 with a tumor in the duodenal submucosa. Three patie with lymph node metastases and 1 with a pancreatic tumor typical homogeneous type A_1 cell populations; 2 with hepat metastases had greatly changed homogeneous type A_1 cell p ulations; 1 with lymph node metastases and 2 with pancreati tumors had heterogeneous cell populations in which A_1 cells predominated; and 1 with a duodenal tumor had a homogen eous cell population with a different type of A_1 cells. The ultrastructure for each of these types of A_1 cell populations described in detail. In 9 of these 10 cases A_1 cells were arg rophilic with characteristic dense oval granules. Two other patients with the clinical and biological symptoms of Zolling Ellison syndrome had no tumors, but did have abnormally l Langerhans' islets and an increase in the number of A_1 cells

se findings suggest that A_1 cells in the pancreas secrete a
rin-like hormone.

1 **MICROBIOLOGICAL CHANGES IN THE HUMAN FECAL FLORA FOLLOWING THE AD-
ISTRATION OF TETRACYCLINES AND CHLORAM-
NICOL.** *(E.)* Sompolinsky, D. (Asaf Harofe Govt. Hosp.,
fin, Israel), V. Yaron and W. J. Alkan. *Amer J Proctol*
):471-478, 1967.

effect of various tetracyclines and chloramphenicol on the
flora was studied in 74 hospitalized patients and 15
hospitalized adult subjects. Neither group had received
biotics for at least 6 months prior to the experiment and
ents with a history of gastrointestinal disease were excluded.
drugs were administered p.o. for 5 days, demethylchlorte-
ycline at a dose of 150 mg q.i.d.; chlortetracycline, chloram-
icol, oxytetracycline and tetracycline at 250 mg q.i.d. Im-
iately prior to treatment, 26.2 and 11.3% of the strains
feces of hospitalized and nonhospitalized patients, resp.,
resistant to tetracyclines (considered collectively, since the
ct of all the tetracycline drugs was virtually identical) and
and 2.9%, resp., were resistant to chloramphenicol. Two
of treatment with tetracycline caused almost complete
ppearance of strains sensitive to these drugs from the hos-
ized patients; 2 weeks and even 2 months after treatment,
o 80% of the isolated microorganisms remained resistant to
cyclines. During this treatment, the number of strains re-
nt to chloramphenicol rose from 14 to 70%, but these re-
nt cultures disappeared after treatment. In the nonhospi-
ed subjects treated with tetracyclines, comparable numbers
tracycline-resistant cultures appeared in 2 days as in the
italized group, but resistance rapidly disappeared after
ment. Resistance to chloramphenicol rarely occurred dur-
etracycline therapy. At the end of chloramphenicol treat-
t, over 40% of the cultures were still susceptible to this
in hospitalized subjects and marked cross-resistance to the
cyclines was apparent. The effects of this antibiotic were
rent even 2 months after treatment. Comparison with
hospitalized subjects was not made because of the small
of the latter group. In 20 nontreated hospitalized con-
, the incidence of resistant strains on the 1st day was 16.6
6.3% to the tetracyclines and to chloramphenicol, resp.;
2 weeks of hospitalization, these figures increased to 29.4
12.5% resp. Two months later nearly all organisms were
itive to the drugs studied. Studies on fecal strains of 19
ed hospitalized patients revealed transferable multiresis-
e factors in 16 cases. These studies call for renewed atten-
to the problems of hospital hygiene.

2 **CLINICAL EVALUATION OF COMBINED RADIATION AND CHEMOTHERAPY IN GASTRO-
ESTINAL MALIGNANCIES.** *(E.)* Henderson, I. W. D.
ntreal Gen. Hosp., Quebec, Canada), B. Lipowska and M. N.
heed. *Amer J Roentgen* 102(3):545-551, 1968.

clinical efficacy of i.v. 5-fluorouracil alone (15 mg/kg x
ys, then 500 mg biweekly until toxicity appeared), radiation
py alone (3000 to 7000 r in 20-30 days, then adjusted to
atient's tolerance), and combined radiation and 5-fluoroura-
erapy (3000-7000 r over an average of 45 days; 500 mg
e drug biweekly during the course of radiation therapy
provided no toxicity developed) was evaluated in 61 patients with advanced gastrointestinal cancer (rectum, colon, stomach). The majority of patients who reported subjective improvement also showed objective changes in tumor growth patterns. Statistical studies demonstrated a positive correlation between the amount of delivered radiation and the response of gastrointestinal malignancy, but no significant correlation was found between clinical response and dose/day of ionizing radiation delivered within the patient's tolerance. Among 10 patients treated with 5-fluorouracil alone for cancer of the colon, 7 showed subjective improvement and 6 of these 7 had objective improvement. There was a positive correlation between the amount of drug delivered in a specific period of time and the clinical response. Three of 4 patients with stomach cancer and 1 of 4 with rectal cancer showed objective benefit from drug therapy alone, but the small numbers of patients precluded statistical analysis. The response achieved with combined radiation and drug therapy in 14 patients with colonic lesions (6 subjective and 7 objective improvements) were analyzed as being due to additive effects and not synergism. Statistical analysis was not performed on the small groups of 8 patients with stomach cancer (6 objective and 5 subjective improvements) and 7 patients with rectal cancer (4 subjective and 4 objective improvements). All patients showed varying degrees of gastrointestinal toxicity, including diarrhea, nausea, vomiting and stomatitis. It is concluded that combined therapy with radiation and 5-fluorouracil produces the same response as radiation alone, provided the radiation is delivered in sufficient quantity. However, the required dose of radiation may be significantly less in patients given the drug in combination.

8723 **OBSERVATIONS ON AMOEBIASIS AND ITS TREATMENT.** *(E.)* Srivastava, B. N. (Med. Coll., Jabalpur, India), G. P. Vyas and B. Sarkar. *Indian Pract* 21(10): 763-772, 1968.

The clinical records of 501 patients (291 males and 210 females, 62.8% 21-40 yr old) treated for amebiasis during the period Jan. 1966-April 1968 were evaluated. Abdominal pain (65.4% of the cases), flatulence (47.3%) and diarrhea (32.7%) were the most frequent symptoms; 6 patients were asymptomatic. The most common physical signs included hepatomegaly (58.8%), hepatic tenderness (46.7%), tender cecum and colon (48.5%), and tachycardia (24,3%). Anemia, leukocytosis and elevated erythrocyte sedimentation rates occurred in 64, 39 and 119 cases, resp. Administration of 1.2 g/day of metronidazole for 5, 7, or 10 days to 32 patients with hepatic, 11 with intestinal and 7 with both types of amebiasis resulted in symptomatic improvement in 47 cases, complete parasitological reversal of stools within 4-10 days in 48 cases, and relief of hepatic tenderness in 38 of 39 patients with amebic hepatitis. No appreciable regression in hepatomegaly was noted. The value of adding a contact amebicide (900 mg/day of diiodohydroxyquinoline) to the metronidazole could not be evaluated because of the small number of patients (14). Broxyquinoline (350, 450 or 500 mg tablets, 2 tablets t.i.d. for 10 days) given to 105 patients with intestinal and/or hepatic amebiasis produced parasitological reversal in 4-7 days with symptomatic improvement in most cases. The 500 mg tablet proved to be most effective. However, the drug was without effect on hepatic amebiasis. Ambilhar was administered to 6 patients (500 mg b.i.d.) but had to be withdrawn on the 3rd day because of toxicity.

8724 **THE CYST PASSERS.** *(E.)* Chhuttani, P. N. (Nehru Postgrad. Inst. Med. Educ. Res., Chandigarh, India), M. S. Grewal and S. M. Ashraf. *Indian Pract* 21(10): 714-718, 1968.

One-day stool examinations for cysts of *Entamoeba histolytica* were carried out in 3982 subjects (hospital patients, employees and nurses). Positive results were obtained in 284 cases, of which 193 cases (67.9%) were symptomless cyst passers. The medical and surgical patient group (1532 males, 1598 females) showed cysts in 199 cases (6.36%); of which 138 were symptomless cyst passers. Similar screening of 548 hospital employees (301 males, 247 females) and 304 nurses revealed positive stool rates of about 8% in symptomless subjects (31 and 26 cases, resp.). When the number of stool examinations was increased to 3, the incidence of positive findings increased significantly from 8.55% to 13.35% in the nurse group. Sixty-three of the 193 cyst passers were treated with metronidazole (400 mg t.i.d. for 5 days). Negative results occurred within 48 hr in 75% of the cases and within 72 hr in 93%. This treatment produced a clearance rate of 92%. Two of the 5 failures became negative when a second course of therapy was given. Sigmoidoscopy, performed in 58 of 63 patients prior to treatment, demonstrated normal mucosa in all instances. The need for treating every cyst passer is supported by the results of experiments in which 11 of 34 dogs fed stools from symptomless cyst passers showed signs of acute amebic dysentery. All of the infected dogs passed mucus and blood and showed *E. histolytica* trophozoites.

8725 **CLINICAL STUDIES ON ACUTE UPPER ABDOMINAL PAIN. PART I.** *(Kor.)* Kim, S. K. (Han-Il Hosp., Seoul, Korea), J. D. Choi, C. S. Lee, S. O. Ahn, I. Y. Choi and J. D. Yoon. *Korean J Intern Med* 11(3):37-46, 1968.

The differential diagnosis of acute upper abdominal pain was determined by clinical, laboratory, and physical findings in 113 patients (62 men and 51 women, 20-50 yr old) seen during a 9-month period. Pain was attributed to peptic ulcer in 21 (18.6%), gallstones with cholecystitis in 18 (15.9%), acute pancreatitis in 13 (11.5%), acute gastritis in 8 (7.1%), acute gastroenteritis in 5 (4.4%), intestinal ascariasis in 7 (6.2%), hepatic malignancy in 6 (5.3%), other types of hepatic disease in 6, gastric cancer in 6, acute appendicitis in 5, ileus in 4, ulcerative colitis in 3, subphrenic abscess, perforated ulcer or pylorospasm in 2 each, and gastric polyps, shigellosis, pyelonephritis, angina pectoris or myocardial infarction in 1 case each. The pain was located in the epigastrium in 78 cases, the right upper quadrant in 24, and the left upper quadrant in 6; radiation to the back was noted in 15 cases, to the right shoulder in 12 cases, and to the left shoulder in 2 cases.

8726 **EPIDEMIOLOGY AND ETIOLOGY OF SEVERE INFANTILE DIARRHEA.** *(E.)* Moffet, H. L. (Children's Mem. Hosp., Chicago, Ill.), H. K. Shulenberger and E. R. Burkholder. *J Pediat* 72(1):1-14, 1968.

Among 632 children hospitalized for diarrhea over a 2-yr period, 261 (41%) were less than 2 yr old and required i.v. therapy. Throat and rectal swabs were taken for bacterial and viral studies on admission. *Shigella* and *Salmonella* were recovered from 3% of the 261 infants (average age 6 months) with severe diarrhea, compared to 12% of infants with mild diarrhea and 21% of older children with severe diarrhea. Enteropathogenic
coli were recovered from 16% of infants less than 4 mont
of age, but from none of the infants older than 7 months
Candida albicans was recovered from 18% of infants witho
previous antibiotic therapy. No statistical evidence was fo
for an etiological association of other bacteria with diarrh
adenovirus, ECHO virus, or Coxsackie virus was recovered
23% of infants hospitalized with diarrhea, compared to 11
normal infants. An etiological association of adenovirus
diarrhea may explain the greater frequency of diarrhea in
winter months and the observation of respiratory sympto
with diarrhea in some cases. Among the 261 patients req
i.v. therapy, there were 9 deaths; in only 2 of the 9 were
teric pathogens found (*Salmonella* and *E. coli).* In the m
ity of cases of sporadic severe diarrhea, no viral or bacter
pathogen was found.

8727 **INTRACTABLE DIARRHEA IN EARLY I
FANCY.** *(E.)* Avery, G. B. (Children's Hos
Dist. Columbia, Washington), O. Villavicencio, J. R. Lilly
J. G. Randolph. *Pediatrics* 41(4):712-722, 1968.

Clinical experience with 20 infants (all less than 3 month
at onset) treated for intractable diarrhea of more than 2
duration; and having 3 or more stool cultures negative fo
Salmonella, Shigella, pathogenic *E. coli,* ova and parasites
presented. All patients received supportive therapy inclu
i.v. fluids (colloids where indicated), electrolytes, and vita
Twelve had identifiable pathological entities sufficient to
plain their protracted diarrhea. In 8 of the 20 infants, n
identifiable etiology could be established, and these cases
termed nonspecific enterocolitis. Six of these 8 infants d
at autopsy, widespread enterocolitis with destruction of t
mucosa and inflammatory infiltration was noted. In the
survivors, 1 had a colostomy and steroid treatment and 1
colostomy alone; abrupt improvement in the clinical cour
noted after these measures were instituted. It is felt that
gardless of the initial cause of the diarrhea, certain perpet
factors come into play which produces intractability. Co
and perhaps adrenal corticosteroids, may be useful in the
apy of refractory enterocolitis if applied before the termi
stages of the disease.

8728 **ULCERATIVE ENTEROCOLITIS IN INFA
AND CHILDREN DETECTED AT AUTOPS**
(Pol.) Lawiński, M. (Dept. Path. Anat., Acad. Med. Wroc
Poland), J. Szacki and J. Miniewski. *Pat Pol* 18(3):351-3
1967.

Among 4084 infants and children (up to 10 yr of age) wh
to autopsy during 1953-65 at the Wroclaw Medical Acade
458 died of gastroenteritis and 147 of these (84 boys and
girls) were found to have ulcerative enterocolitis. The dis
was localized to the ileum in 19.7%, to the jejunum in 3.
and to the small bowel as a whole in 23.6%, while in 52.3
affected both the small and large intestine. It was most c
mon in infants below the age of 6 months, and only 12 o
147 cases were over 1 yr old. Peritonitis was noted at au
in 39 cases, including 17 in which there was intestinal per
tion. Central nervous system pathology was found in 21.
compared to 10.6% of the 311 children who died of gastr
enteritis without ulceration, and myocardial disease was fo
in 39.4% (11.5% of the other 311). Pneumonia and midd

nfections were seen in 75.5-82.3% and 74.2-76.8% of the
e group of 458 children, resp. Fatty degeneration of the
was found in 288 cases, including 95 of the 147 with ul-
ive enterocolitis, and 40 of these also had splenomegaly.
concluded that the principal etiological factors in ulcera-
enterocolitis of infancy are cardiovascular disorders with
tant intestinal ischemia, central nervous system disorders,
a general deficiency in the defense mechanisms of the body.

9 GASTROINTESTINAL BLEEDING IN CHILDREN.

(Rus.) Kushch, N. L. (A. M. Gor'ki Med. Inst.,
etsk, USSR), R. M. Knishevitskii and G. A. Sopov.
urgiia (Moskva) 44(7):73-76, 1968.

88 children with manifest gastrointestinal bleeding, 267 had
l and sigmoid polyps, 69 had intussusceptions, 29 had por-
ypertension and gastric or esophageal varices, 8 had Meckel's
ticulum, 7 had hemorrhagic vasculitis, 3 had duodenal ulcers,
5 had bleeding of unknown etiology. A total of 18 patients
(9 with intussusceptions, 6 with bleeding varices, 1 with
cel's diverticulum, and 2 with hemorrhagic vasculitis). Of
hildren with portal hypertension, 29 had massive hemor-
es before a diagnosis was established. The procedure of
ier and Linton was performed in 2 cases and splenectomies
suturing of esophageal veins in 2 cases. All of these pa-
s died of progressive bleeding shortly after surgery. Two
ie 3 patients with duodenal ulcers responded to conserva-
treatment. A duodenotomy was performed on the other.

30 ADENOMAS OF ALPHA-CELL ISLETS: CLINICAL AND BIOLOGICAL FINDINGS. *(Fr.)*

er, J. P. (Bichat Hosp., Paris, France), S. Bonfils and A.
ibling. *Actualites Hepatogastroent Hotel Dieu* 3(3):147-157,
7.

10 patients (6 men and 4 women, aged 17-65 yr) with
inger-Ellison syndrome, 4 had histories dating back 4 yr or
e. Nine of these patients had diarrhea which was associated
ı ulcer symptoms in 8 cases. Steatorrhea was present in 5
severe hypokalemia in 2. One patient with diarrhea and
re hypokalemia died in anuria despite treatment. Finsterer
rectomies performed on 2 of these patients resulted in an
ediate recurrence in 1 case and, after gastric resection and
otomy, a 2nd relapse; the other patient developed an anasto-
ic ulcer 5 yr after surgery. Severe hemorrhages occurred in
ises and perforations in 2. Pancreatic tumors were associated
ı a parathyroid adenoma and kidney stones in 1 case and
ı pituitary disturbances in 2. Only 1 patient had a history
amilial ulcers. Ulcers were located in the esophagus in 2
s, in the jejunum in 2, and in the duodenal bulb in 5; in
ises bulbar ulcers were associated with ulcers in the lower
denum. Lymph node or hepatic metastases were found in
ises. Four patients who were subjected to total gastrectomy
hemipancreatectomy on the left were alive and well 14-36
iths after surgery. Tests for the hourly flow of free acid,
maximum free acid concentration, K_{25} values, and secretion-
lucing activity of urine in rats were found to be of diagnos-
value for patients with intact stomachs. These results were
h less characteristic in patients who had undergone gastrec-
ies.

1 CHARACTERISTICS OF THE CLINICAL COURSE OF ENTEROVIRAL DIARRHEA IN CHILDREN.

(Rus.) Vail', L. V. (Tadzhik Med. Inst., Dushanbe, USSR), K. T. Kasymov, A. N. Pavlovich and R. M. Litvinenko. *Pediatriia (Moskva)* 47(7):43-47, 1968.

Virological and bacteriological studies were carried out daily for 10 days on the feces of 785 children admitted for severe diarrhea in 1964. Cytopathogenic viruses were isolated in 9.5% of 252 cases of acute dysentery, 24.6% of 77 cases of chronic dysentery, and 35% of 154 cases of undefined gastroenteritis and dyspepsia, compared to 11.2% of 871 healthy children examined during the same year. Dysentery bacilli were isolated from 45.4% and 20.7% of cases of acute and chronic dysentery, resp., while both dysentery bacilli and cytopathogenic viruses were isolated from 10.4% and 6.4%, resp. The proportion of positive stool specimens fluctuated widely, however, in relation to age, final diagnosis, and principal symptoms. It is concluded that the intestinal form of enteroviral infection in children is less serious and of shorter duration than bacterial dysentery. In very young children (under one year), however, the intestinal manifestations of enteroviral diarrhea are more marked, especially in combination with bacterial infection. In young children with a combination of viral and bacterial diarrhea, blood occurred in the stools 5 times more frequently than in purely viral diarrhea, and in older children the most pronounced pathogenic changes in the stools (presence of mucus and blood) occurred in bacterial dysentery. Enteroviral infections assume less severe forms as the children grow older, since immunities are built up to the majority of enterovirus types and a greater range of nonspecific protective reactions apparently develops.

8732 THE ROLE OF ENTEROPATHOGENIC *ESCHERICHIA COLI* IN THE DEVELOPMENT OF ACUTE GASTROINTESTINAL DISEASE IN ADULTS. *(Rus.)*

Sobolev, I. N. and V. P. Akimov. *Sovet Med* 31(6):110-112, 1968.

Food poisoning caused by an enteropathogenic strain of *E. coli* was diagnosed in 30 adults who had eaten macaroni with meat sauce; 19 of the 30 had to be hospitalized. The incubation period was 2-4 hr, after which signs of gastroenteritis and gastritis appeared along with the usual symptoms of poisoning–weakness, headache, dizziness, nausea and vomiting. Some patients experienced chills and abdominal pain. In 5 cases muscle spasms of the extremities were noted. Blood studies revealed normal leukocyte counts in all but 2 patients and urinalysis showed no pathological changes. *E. coli* 026 was isolated from the food itself, from the dishes, from vomited material, from the blood of 2 patients, and from the feces of 16. Recovery took 2-4 days with the usual supportive treatment. Although the main role in the disease process must be ascribed to the bacterial toxin, the positive blood cultures obtained in 2 patients indicate that a secondary role may be played by bacteremia.

8733 SARCOIDOSIS IN CHILDREN, WITH SPECIAL EMPHASIS ON THE NATURAL HISTORY AND TREATMENT.

(E.) Jasper, P. L. (U. North Carolina Sch. Med., Chapel Hill) and F. W. Denny. *J Pediat* 73(4):499-512, 1968.

The epidemiology, clinical characteristics, diagnosis, natural history and effects of conservative management and corticosteroid therapy in childhood sarcoidosis are discussed on the basis of reports in the literature and 25 children (14 boys and 11 girls; 18 Negro, 5 Caucasian and 2 Indian; average age 13 yr with 24 of 25 between 10 and 15 yr old at time of diagnosis) followed for an average of 2.8 yr between 1954 and 1968. The cases were scattered over central and eastern North Carolina and showed no significant epidemiological pattern. The most common symptoms, which were present for an average of 7 months before diagnosis, were weight loss, cough, fever, abdominal pain, ocular pain and anorexia. Hypercalcemia, hyperproteinemia and eosinophilia were the usual laboratory findings, and pulmonary function was abnormal in 6 of 11 tested. Although 14 had hepatomegaly, none had abnormal liver function tests. Pulmonary parenchymal and/or hilar node involvement was seen in all 25, and 23 cases had involvement of at least one other organ (up to 7 other organs). There were no cases of gastrointestinal involvement, but lymph nodes were affected in 18 cases and 12 had ocular disease. Among the 18 who were followed for at least 1 yr, 10 were treated with corticosteroids. These 10 showed resolution of symptoms within a few months, along with resolution or decrease in severity of organ involvement, while in the other 8 cases some new symptoms appeared and a few showed progression of the disease to new organs. Recurrences after withdrawal of corticosteroid therapy were rare.

8734

A STUDY OF BACTERIA AND LETHAL FACTORS IN FLUIDS FROM EXPERIMENTAL INTESTINAL OBSTRUCTION IN DOGS. *(E.)*

Nagy, I. (U. Glasgow Vet. Sch., Scotland) and W. L. Weipers. *J Path Bact* 95(3):199-210, 1968.

Experiments are reported which were designed to study the incidence of death or survival in relation to the bacterial flora present in isolated intestinal loops constructed in mongrel dogs, and to assay quantitatively the lethal components in peritoneal fluid, loop and gut content of dogs with isolated loops and in the gut content of normal dogs. No association of death in experimental intestinal obstruction with any single species or specific group of bacteria was found. However, the lethal factors present in the peritoneal fluid or loop content could not be transferred to recipient animals if the bacteria present were first removed. The toxicity of these fluids was greatly diminished if the bacteria were prevented from propagating by the use of antibiotics. It appeared that the lethal nature of the fluids did not arise from any significant quantity of preformed bacterial toxins, but was associated with the invasion of the peritoneal cavity by multiplying bacteria. Direct contact of bacteria with host tissues also appeared to be important, because when their products were freely able to diffuse from chambers placed within the peritoneal cavity, but the bacteria were retained, the lethal effect could not be demonstrated.

8735

A FOOD-BORNE INSTITUTIONAL OUTBREAK OF ENTERITIS DUE TO *SALMONELLA BLOCKLEY*. *(E.)*

Morse, L. J. (St. Vincent Hosp., Worcester, Mass.) and A. D. Rubinstein. *JAMA* 202(10):115-116, 1967.

An outbreak of food-borne salmonellosis involved 167 cases at the Memorial Hospital in Worcester, Massachusetts during May-June of 1966; the peak period was the 1st 10 days of June. Approximately 40% of the people had actual symptoms while in the remaining 60% the infection was revealed by mandatory stool cultures taken on all dietary personnel, house
officers, and certain nursing personnel. The illness was ch
terized by moderate fever, diarrhea, nausea, and abdomina
cramping lasting 2-3 days. There was no prolonged morbi
or mortality in the 43 hospitalized patients. Treatment w
supportive and the use of antibiotics was discouraged. Th
source of infection was traced to frozen, unpasteurized eg
yolks used in the preparation of ice cream. This is furthe
evidence for the need to exclude frozen, unpasteurized eg
products from human consumption.

8736

A SEVEN-YEAR FIELD TRIAL OF TWO TYPHOID VACCINES IN GUYANA. *(E.)*

Ashcroft, M. T. (M.R.C. Epidemiol. Res. Unit, U. West In
Kingston, Jamaica), B. Singh, C. C. Nicholson, J. M. Ritcl
E. Sobryan and F. Williams. *Lancet* 2(7525):1056-1059,

In 1960, about 72,000 Guyanese schoolchildren (5-15 yr
were divided into 3 similar groups: 1 which acted as a co
and was given tetanus toxoid, a 2nd given an acetone-kille
typhoid vaccine, and a 3rd given a heat-killed phenolized
vaccine. Two doses of 0.5 ml of reconstituted vaccines w
given s.c. 5 weeks apart. An additional 10,000 children r
ceived only 1 dose. The incidence of typhoid fever, diagr
by the bacteriological isolation of *Salmonella typhi*, was f
for 7 yr after vaccination. Among those given 2 doses, 1
of typhoid fever occurred in the control group and 16 an
in the acetone and heat-phenol typhoid vaccine groups, yi
protection rates of 88% and 65%, resp. Among those giv
dose of vaccine, the protection was somewhat greater; 22
occurred in the control group and 1 and 4 in the groups
acetone and heat-phenol vaccines, resp. There was little d
in protection until the 5th yr after vaccination.

8737

THE PATHOGENIC SIGNIFICANCE OF SC BACTERIAL ASSOCIATIONS IN THE INT TINAL FLORA OF NEWBORNS AND INFANTS. *(It.)*

Flauto, U. (Inst. Pediat. Clin., U. Milan, Italy) and F. Rosaschino. *G Mal Infett* 20(2):205-209, 1968.

Cultures were made from the stools of 290 infants, includ
112 newborns, with enteritis caused by pathogenic *Escher*
coli and almost equal proportions of 1 other species or ge
Enteritis was almost always mild when *E. coli* was associa
with *Streptococcus*, was somewhat more severe with *E. co*
and *Aerobacter* or *Pseudomonas*, and was most severe wit
E. coli and other *Escherichia* or *Proteus* strains. While in
previous study *E. coli* 055 in pure culture accounted for
of severe cases of enteritis, when combined with other *Esc*
chia and *Proteus* strains it was responsible for 29.1% and
resp., of severe cases. Similar results were obtained when
0 125 was associated with other *Escherichia* and *Proteus* s
This increased pathogenicity is attributed to synergism bet
these species of bacteria.

8738

EFFECT OF TIMED OCCLUSIONS AT VA LEVELS OF MESENTERIC ARTERIES AN VEINS: CORRELATIVE STUDY OF ARTERIOGRAPH AND HISTOLOGIC PATTERNS OF RAT GUT. *(E.)*

N
C. D. (U. California Sch. Med., San Francisco), O. N. Ran
and A. R. Margulis. *Radiology* 90(1):99-106, 1968.

Histologic and arteriographic changes were studied in the
tinal tract of white, female, Sprague-Dawley rats (250-350

ojected to timed occlusions of the superior mesenteric artery
l/or vein. The animals were grouped according to the oc-
sion site: Group I (47 rats) had root occlusion of both the
oerior mesenteric artery and vein for 0.5-2 hr; Group II (47
s) had root occlusion of the superior mesenteric vein only
30-90 min; Group III (28 rats) had superior mesenteric
ery and vein occlusion 10 divisions proximal to the ileocecal
a for 1-29 hr; Group IV (22 rats) had preterminal occlusion
the superior mesenteric artery and vein for 30 min-3 days;
oup V (26 rats) had preterminal and collateral occlusion of
superior mesenteric artery and vein for 23-120 hr; Group
(13 rats) were kept as controls. The most striking histologic
l arteriographic changes were found in Groups I, II, III and
while the least amount of change was found in Group IV.
t survival depended on the site, level and duration of the
scular occlusion. Histologic indication of infarction was cor-
oorated by small vessel abnormalities on arteriography.

39 **TRAVELLER'S DIARRHOEA: A SURVEY OF
SYMPTOMS, OCCURRENCE, AND POSSIBLE
OPHYLAXIS.** *(E.)* Turner, A. C. (Air Corp. Joint Med.
rv., London Airport, Hounslow, Middlesex, England). *Brit
d J* 4(5580):653-654, 1967.

comparison of Streptotriad with neomycin-trisulfonamides
l a placebo in the prophylaxis of traveller's diarrhea was
de in a study involving about 1100 British Airlines personnel
l families taking trips abroad. Medication was randomly dis-
buted and given for 3 weeks, 1 tablet twice daily for the 1st
weeks and 1 daily for the 3rd week. Children under 12 yr
l were given half the dosage. The commonest places of oc-
rrence of traveller's diarrhea were Africa (25.9%) and the
ddle East (23.5%). Streptotriad was statistically beneficial
reducing both diarrhea and associated symptoms. Neomycin
sulfonamides was statistically beneficial in reducing symp-
ms, but not diarrhea; abdominal pain was the commonest
sociated symptom: 75% had a duration of 3 days or less.
lverse reactions were minimal with Streptotriad; this proved
be the most popular medication on subsequent requests.

740 **HAEMORRHAGIC NECROSIS OF THE IN-
TESTINE.** *(E.)* McKinnell, J. S. (Selly Oak Hosp.,
rmingham, England) and M. S. Kearney. *Brit Med J* 4(5577):
50-463, 1967.

he clinical and pathological features of 13 cases of acute
emorrhagic enterocolitis seen in 1 hospital during an unusually
y spell in the summer and autumn of 1964 are described.
is suggested that, while the basic cause was probably re-
iction of the blood flow through the wall of the intestine,
is was probably due to functional factors associated with
art failure and its treatment and not to anatomical lesions in
e splanchnic blood vessels. It is also suggested that usually
rmless organisms ingested in unusually large numbers due to
matic conditions, and possibly proliferating in the intestinal
ntents owing to stasis, may have precipitated this hemor-
agic necrosis of the intestinal mucosa.

41 **ASCORBIC ACID NUTRITION IN GASTRO-
DUODENAL DISORDERS.** *(E.)* Cohen, M. M.
lasgow Royal Infirm., Scotland) and A. M. Duncan. *Brit
d J* 4(5578):516-518, 1967.

Leukocyte ascorbic acid levels were estimated and compared in 14 normal healthy subjects (8 men and 6 women, mean age 22 yr), all of whom were known to have an intake of ascorbic acid of at least 30 mg/day, 14 control patients (7 men and 7 women, mean age of 50 yr) with no known alimentary tract disease, with a mean dietary intake of 39 mg/day of ascorbic acid, 14 patients with gastroduodenal disorders (7 men and 7 women, mean age 50 yr), with a mean dietary intake of 27 mg/day of ascorbic acid, and 8 patients (5 men and 3 women) with duodenal ulcer before and after surgery, with a mean dietary intake of 27 mg/day of ascorbic acid before surgery and 53 mg/day after surgery. In the 14 normal subjects, the mean leukocyte ascorbic acid level was 21.7 $\mu g/10^8$ WBC, compared to 22.9 $\mu g/10^8$ WBC in the 14 control patients, 11.0 $\mu g/10^8$ WBC in the 14 patients with gastroduodenal disorders, 8.7 $\mu g/10^8$ WBC in the duodenal ulcer patients before surgery, and 12.8 $\mu g/10^8$ WBC after surgery (vagotomy and pyloroplasty). It is recommended that patients with duodenal ulcer should have routine supplements of ascorbic acid prior to surgery and during the postoperative period.

8742 **CLINICAL EVALUATION OF 5-BROMO-5-FLUORO-6-METHOXY-DIHYDRO-2′-DEOXYURIDINE.** *(E.)* Van Dyk, J. J. (Cornell U. Med. Coll., New York, N. Y.), B. D. Clarkson, R. Duschinsky, O. Keller, E. La Sala and I. H. Krakoff. *Cancer Res* 27(11):2129-2136, 1967.

5-Bromo-5-fluoro-6-methoxy-dihydro-2′-deoxyuridine (MeOBrFUdR) was given to 22 patients with far advanced, nonresectable neoplastic disease. Of these, 16 received an adequate course of therapy. MeOBrFUdR produced biologic effects indistinguishable from those caused by 5-fluoro-2′-deoxyuridine (FUdR) at approximately equimolar doses. Two of the 16 patients (males with carcinoma and adenocarcinoma of the stomach) showed significant therapeutic responses, a response rate that would be expected with FUdR. By biochemical and microbiologic methods, appreciable levels of FUdR were found to persist in the blood and urine of patients for 16 hr after administration of MeOBrFUdR, a much longer period than that following the administration of similar doses of FUdR. The persistence of detectable levels of FUdR in the blood did not result in increased toxicity of MeOBrFUdR as compared to FUdR.

8743 **THE TREATMENT OF ACUTE INTESTINAL INTUSSUSCEPTION IN CHILDREN.** *(It.)* Bardini, T. (Queen Margherita Pediat. Hosp., Turin, Italy). *Minerva Pediat* 19(27):1498-1505, 1967.

In a 5-yr period, intussusception was diagnosed in 43 children (29 males and 14 females, aged 2 months to 4 yr). Of these children, 32 were less than 1 yr old. Intussusception accounted for 7.2% of all emergency operations and was diagnosed in 18.7% of all children between 2 months and 4 yr. The maximum incidence was among children 8-9 months old. The mean period between the onset of symptoms and hospitalization was 35 hr. Abdominal pain was present in 40 cases, vomiting in 37, melena in 41, and fever in 15. The diagnosis was established in 21 cases by palpation and in 12 by a combination of palpation and rectal examination. Intussusception involved the ileum and colon in 28 cases, the ileum and the ileocecal valve in 12, and the ileum only in 3. Associated diseases included 3 cases of Meckel's diverticulum, 1 sarcoma of the small intestine, and 1 jejunal polyp. Barium enemas were successfully used to reduce intussusceptions in 5 patients with ileocolonic involvement. Surgical treatment was employed in 38 cases (manual reduction in 32, appendectomies in 6, resection of Meckel's diverticulum in 2, resection of the ileocecal ligament in 2, and intestinal resection in 6). There was only 1 operative death caused by shock. Operative complications included fevers in all cases, sometimes accompanied by shock; intestinal paresis; and diarrhea.

8744 **WHOLE-BLOOD AMINO ACIDS IN EXPERIMENTALLY INDUCED TYPHOID FEVER IN MAN.** *(E.)* Feigen, R. D. (U. S. Army Med. Unit, Fort Detrick, Frederick, Md.), A. S. Klainer, W. R. Beisel and R. B. Hornick. *New Eng J Med* 278(6):293-298, 1968.

Typhoid fever was induced experimentally in 17 human male volunteers by the ingestion of 10^7 organisms. Blood for amino acid determinations was obtained immediately before exposure and daily for 38 days, and blood and stool cultures were also obtained. Patients who manifested symptoms were treated with either chloramphenicol or ampicillin. A significant increase in whole-blood amino acids was observed during the incubation period; 10 of the 17 subjects became ill. Retrospective analysis of the amino acid changes permitted recognition of impending illness in 9 of the 10. A significant increase was not observed in any subject in whom clinically apparent disease did not develop. A significant decrease in blood amino acid concentrations was observed to accompany and follow clinical illness in the subjects who were ill. This appeared to correlate more closely with the presence of overt illness rather than mere infection. Blood amino acid changes may represent one of the earliest detectable metabolic changes after infection, and evaluation of amino acids may provide a useful tool in the early diagnosis of typhoid fever and may yield important clues to the biochemical and physiologic factors involved in the pathogenesis of this disease.

8745 **EVALUATION OF CHLORAMPHENICOL AND AMPICILLIN IN SALMONELLA ENTERIC FEVER.** *(E.)* Robertson, R. P. (U. S. Naval Med. Res. Unit 3, Abbassiah, Cairo, Egypt), M. F. Abdel Wahab and F.O. Raasch. *New Eng J Med* 278(4):171-176, 1968.

The relative efficacy of chloramphenicol and ampicillin, given orally both separately and simultaneously, was studied in 289 patients with either salmonella enteric fever or nonspecific enteric fever. Bone marrow culture was used as the most sensitive diagnostic tool. Ampicillin was effective in 77% of the acute cases of salmonella enteric fever treated, whereas chloramphenicol and combined therapy were effective in all such cases treated. The 2 groups given ampicillin had fewer relapses and carriers than the chloramphenicol group. In the acute cases of nonspecific enteric fever each drug regimen worked equally well, with no treatment failures, relapses, or carriers. Overt hemolytic anemia occurred in 3 chloramphenicol-treated typhoid patients who were also deficient in glucose-6-phosphate dehydrogenase. There was only 1 classic typhoidal complication and no mortality. It is concluded that chloramphenicol remains the most effective drug in oral treatment of acute salmonella enteric fever and that ampicillin is an effective alternate drug.

8746 **CONGENITAL SELECTIVE MALABSORPTIO OF VITAMIN B_{12} WITH PROTEINURIA.** *(It.* Gräsbeck, R. (Maria Hosp., Helsinki, Finland) and G. Kvist. *Minerva Med* 58(79):3488-3495, 1967.

Case reports are presented for 5 patients (2 males and 3 females), 2 of whom belonged to the same family. All of thes patients had megaloblastic anemia and positive Schilling tests, but administration of human gastric juice did not normalize the values in the Schilling test. All 5 patients had mild proteinuria and 2 had membranous glomerulonephritis which wa also present in a patient with pernicious anemia who did not have proteinuria. Glomerulonephritis disappeared after paren teral administration of vitamin B_{12}. Biopsies of the jejunum and ileum were normal, indicating that the primarily lesion might be at a submicroscopic or molecular level.

8747 **ENTERITIS SYNDROMES PRODUCED BY *YERSINIA ENTEROCOLITICA.*** *(Fr.)* Wauter G. (Réga Inst., Louvain, Belgium), C. Graux and G. Bruynog *Presse Med* 75(40):1975-1977, 1967.

Brief reports are presented for 4 patients, aged 1.5-27 yr, wit diarrhea and abdominal cramps. In 1 case abdominal pain w so severe that surgery was performed and pathological lesions were noted in the ileocecal lymph nodes. 'In all cases *Yersini enterocolitica* was isolated from the stools on deoxycholate-citrate-lactose medium and agglutinin tests were positive. In addition, 1 patient had a positive intradermal reaction 6 mon after the infection. In 2 cases, other children in the family h similar, but less severe, episodes of enteritis and positive seru agglutinin tests.

8748 **INTRAABDOMINAL SCHWANNOMAS: REPORT OF TWO CASES.** *(It.)* Lamarca, S. (Inst. Clin. Surg., U. Turin, Italy), F. Moricca, F. Donadio an G. Giribone. *Minerva Chir* 22(23):1279-1286, 1967.

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07744* 08112* 08115* 08144 08149 08234* 08776 08835
08848 08901
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07600 08031 08048 08104 08203 08214
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07883 08463 08524 08593* 08594* 08597* 08598* 08599*
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07841* 07878 07879 07886 07898 07932 07938 07957
07973 07974 07978 07979 07980 08384 08454 08507
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08663* 08672 08676 08681 08686 08692 08702 08703
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08671 08673 08674 08680 08688 08690 08693 08694
08697 08706 08712 08714 08725* 08777 08837

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07824 07938 07974 08468 08668 08687
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08045*
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08782 08830 08835 08901 08914 08915 08916
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08230* 08241 08248 08256* 08328 08399* 08443* 08718*
08730* 08732* 08739* 08756 08768 08780 08798 08799
08807 08851 08863 08877 08879 08883 08898 08902
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08741*
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TARY FACTORS
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08377 08485 08543* 08548* 08555* 08557* 08576 08641
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07643* 07651 07654 07688 07783
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07843* 08166* 08388 08656* 08660* 08663* 08669 08674
08675 08682 08683 08684 08690 08700 08712
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07858* 08422* 08428* 08521 08635* 08640 08644 08645
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07836* 08374* 08376 08379 08381 08388 08396* 08397*
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07757 07758 07760 07764 07771 07778 07782 07797
07798 07799 07807 07808 07864* 08450 08489 08522
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08754 08756 08765 08766 08774 08775 08778 08781
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07683 07745* 07832 07842* 07921 07953 08366 084
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08737*
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07895 07903 07933 08218* 08221 08222 08223 08224
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07820 08258* 08263*
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07708* 07713* 07714* 07720* 07732* 07734* 07737* 07738*
07739* 07746* 07751* 07753 07754 07757 07758 07761
07766 07768 07769 07772 07777 07782 07784 07787
07788 07789 07791 07794 07796 07797 07798 07801
07804 07807 07829 07934 07946 07962 08476 08487
08495 08506 08508 08543* 08546* 08560* 08626 08643
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08642 08645 08729* 08813 08816 08836 08845 08900

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08227* 08241 08242 08245 08271 08288 08298 08315
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08746* 08749 08757 08856 08864 08912 08915
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08447 08455 08476 08501 08538 08540* 08548* 08589
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07931 08675 08680 08706 08711
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08704 08704 08709 08711 08714
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08694 08697 08703
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07997 08316
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07871 08037* 08039* 08056 08064 08072 08094 08097
08098 08101 08111 08137 08183 08196 08205 08389
08779 08815 08824 08856
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08035* 08049 08182 08184 08185
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07657* 07935 08034* 08036* 08042* 08049 08050 08051
08055 08058 08060 08061 08062 08063 08065 08074
08082 08102 08170* 08182 08184 08185 08186 08195
08213 08276 08694 08793
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07849* 08034* 08170*
STRECTOMY-INDUCED AFFERENT LOOP SYNDROME
08049 08058 08185 08195
STRECTOMY-INDUCED ANEMIA
08036* 08058 08067
STRECTOMY-INDUCED BONE DISEASES
08041* 08103 08885
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08035* 08049 08058 08062 08182 08185
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08176* 08720* 08779
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08054 08058 08098 08108 08211 08725* 08732* 08762
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07631* 07673 07818 07821 07834
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07972 08173*
STROINTESTINAL CANCER
07901 08719* 08764 08821 08876 08903
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07831 07887 08728* 08740* 08792 08817 08831 08850
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07859* 07862* 07872 07887 07901 07902 07909 07910
07911 07919 07923 07924 07953 07956 07960 07972
07981 08021 08140 08782 08794 08825 08828 08863
08909
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08804
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07972 08054 08731* 08732* 08741* 08753 08757 08771
08783 08790 08794 08837 08843 08849 08874 08888
08909 08913
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07617 07669 07830 08848
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08644 08760
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07640* 07791 07830 07833 07859* 07909 07911 07951
08067 08145 08228* 08429* 08664* 08726* 08732* 08734*
08737* 08740* 08747* 08758 08778 08796 08798 08800
08804 08838 08841 08847 08849 08852 08881 08884
08888 08898
GASTROINTESTINAL MOTILITY
07656* 07823 07901 08767 08772 08847 08858
GASTROINTESTINAL MUCOSA
07631* 07667* 07669 07670 07821 07830 08179 08189
08738* 08740* 08791 08848
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08748 08817 08824 08912
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08128 08272 08717* 08734* 08751 08763 08767 08770
08772 08792 08832 08841 08846 08895
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08740* 08792 08814 08858 08911 08912
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08189 08728* 08757 08789 08806 08895
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07872 07901 07910 07923 07924 07956 07960 07981
08021 08073 08402 08434* 08782 08807 08825 08828
08855 08874
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07656*
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08035* 08105 08215 08401 08717* 08763 08773 08782
08785 08815 08823 08825 08828 08832 08842 08852
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08790 08794 08795 08799 08801 08802 08804 08813
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08294 08303 08321 08343' 08349 08351
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07601 07705* 07722* 07723* 07730* 07736* 07751* 07755
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08463 08523 08537 08569 08580 08611
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07840 07867* 07883 07890 07893 07904 08474 08523
08592* 08593* 08595* 08598* 08610 08612
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08463 08502 08580
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07890 08592* 08596* 08599* 08599* 08600* 08602* 0860
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08050 08454 08463 08523 08569 08582 08584 0860
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07883 08463 08524 08593* 08594* 08597* 08598* 0859
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08010 08014 08019 08021 08022 08026 08029 0803
08079 08169* 08655* 08762
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07795 08423* 08433* 08445 08492 08502 08510 08536
08559* 08581 08593* 08616 08641 08654
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07672
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07671
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07755 08849
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08775 08786
SALMONELLOSIS DIAGNOSIS
07905 07951 08735*
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08735* 08749 08797
SALMONELLOSIS IN CHILDREN
08726* 08736* 08749 08775 08786
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08735* 08745* 08765 08775 08786 08803
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08744*

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7916 08080 08109 08155 08428* 08440* 08460 08476
8719*
NING,SCINTILLATION
7848* 07857* 07877 07890 07892 07907 07908 07919
7923 07924 07925 07927 07931 07941 07942 07943
7947 07948 07949 07950 07965 08077 08384 08433*
8468 08571 08581
STOSOMIASIS DIAGNOSIS
7876 08496 08578 08754 08755 08833
STOSOMIASIS EPIDEMIOLOGY
8754 08755 08868 08880
STOSOMIASIS IMMUNOLOGY
8578
STOSOMIASIS PATHOLOGY
8578 08755
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8866 08868 08880
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8754 08833 08866
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7848* 07857* 07877 07890 07892 07907 07908 07919
7923 07924 07925 07927 07931 07941 07942 07943
7947 07948 07949 07950 07965 08077 08384 08433*
8468 08571 08581
ETIN
7690* 07691* 07692* 07695 07838* 07853* 07954 08200
8375*
ETION DISORDERS,STOMACH
7839* 07846* 07856* 08044* 08168* 08176* 08458 08768
8769 08779 08780 08798 08824 08826
ETION STUDY TECHNIQUES
7694 07854*
ETION STUDY TECHNIQUES,BILE
7660*
ETION STUDY TECHNIQUES,STOMACH
7674* 07675* 07677* 07678* 07679* 07687 07839* 07845*
7857* 07896 08165* 08168*
ETION,BILE
7745* 07747* 07762 07969 08426* 08430* 08431* 08441*
8688 08819
ETION,BILIARY TRACT
7747* 08387
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7675*
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7823
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7676* 07678* 07682* 07685 07687 08059 08178* 08200
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7614 07976
ETION,GALLBLADDER
7807 08677
ETION,GASTROINTESTINAL
7656*
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7665* 07675* 07676* 07679* 07682* 07683 07684 08176*
8190 08720* 08769
ETION,KIDNEY ELECTROLYTE
8055 08227*
ETION,LARGE INTESTINE
7636* 07637* 07638*
ETION,LIVER
7615 07726* 07729* 07807 07969 08527 08563 08636*
ETION,NERVOUS CONTROL OF STOMACH
7604 07675* 07684 08165* 08168* 08172* 08188 08203
ETION,PANCREAS
7665* 07691* 07692* 07693* 07694 07696 07954 07958
7970 08375* 08377 08378 08391 08393 08780 08819
ETION,PANCREAS AMYLASE
7690* 07692* 07693* 07695 07853* 07936 07954 07958
7970 08415
ETION,PANCREAS BICARBONATE
7690* 07692* 07853* 08375*
ETION,PANCREAS CHYMOTRYPSIN
7838* 07937
ETION,PANCREAS LIPASE
7651 07954 08239 08415
ETION,PANCREAS TRYPSIN
7692* 07693* 07954 08400*
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7665* 07666* 07668* 07671 08032 08372*
ETION,SMALL INTESTINE
7631* 07976
ETION,STOMACH
7594* 07681* 07682* 07686 07688 07689 07835 07983*
8006 08033*
ETION,STOMACH ACID
7613 07674* 07675* 07676* 07678* 07679* 07680* 07681*
7682* 07683 07684 07685 07687 07688 07689 07695
7839* 07845* 07857* 08059 08072 08166* 08168* 08175*
8176* 08180 08190 08200 08204 08208 08211 08458
8730* 08779 08780 08790 08835 08871

SECRETION,STOMACH ELECTROLYTE
07665* 07678* 07688 08044* 08168*
SECRETION,STOMACH MUCUS
07688
SECRETION,STOMACH PEPSIN
07674* 07682* 07684 07688 07854* 08059 08180 08200
SECRETOR STATUS,BLOOD GROUP
08177*
SEROLOGICAL DIAGNOSIS
07754 07905 07920 07921 07939 08206 08208 08366
08402 08456 08500 08580 08610 08875 08884 08897
SEROTONIN
SEE ALSO CARCINOID SYNDROME
07659* 08498
SERUM BILE PIGMENTS
07778 07821
SERUM BILE PIGMENTS IN JAUNDICE
07809 08511
SERUM CHOLESTEROL
07810*
SERUM ELECTROLYTES
07807 08055 08072 08425* 08537
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07626* 07752* 07763 07806 07840 07842* 07864* 07867*
07870 07914 07962 08366 08424* 08425* 08441* 08474
08537 08541* 08549* 08561* 08562* 08594* 08595* 08598*
08601* 08612 08626
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08592* 08608 08628
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08525
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07956 08428* 08550* 08556* 08602* 08719* 08899
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08649
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08894
SHIGELLOSIS EPIDEMIOLOGY
08800 08838 08839 08894 08897
SHIGELLOSIS IMMUNOLOGY
08838 08839
SHIGELLOSIS IN CHILDREN
08726*
SHIGELLOSIS TREATMENT
08788 08801 08802 08838 08839 08894
SHOCK
07873 08136 08451 08547* 08743* 08776 08823 08845
08867
SHUNT,PORTACAVAL
07762 07858* 07922 07984* 08437* 08457 08458 08472
08473 08482 08488 08496 08632* 08635* 08647
SHUNT,SPLENOPORTAL
07730*
SHUNT,SPLENORENAL
08471 08472 08482 08496
SIALOLITHIASIS
07967
SIGMOID
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08319 08357
SIGMOIDOSCOPY
07917 08144 08259* 08303 08333 08337 08582 08724*
08756 08757 08807 08855 08860 08863
SKELETAL SYSTEM
08022
SMALL INTESTINE
SEE ALSO DUODENUM
SEE ALSO ENTERITIS
SEE ALSO ILEUM
SEE ALSO ILEUS
SEE ALSO INTUSSUSCEPTION
SEE ALSO JEJUNUM
SEE ALSO MALABSORPTION
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07826 08153
SMALL INTESTINE ABSORPTION
07620 07621 07623* 07624* 07625* 07626* 07627* 07628*
07629* 07630* 07634* 07635* 07639* 07641* 07642* 07647
07648 07655 07915 08077 08112* 08116* 08134 08159
08160 08230* 08232* 08234* 08236
SMALL INTESTINE ANGIOGRAPHY
07903 07906 08382
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07609 08026 08092 08124 08139 08901 08916
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08835
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07593* 07594* 07595* 07597* 07620 07621 07626* 07629*
07634* 07667* 07810* 07811* 07816* 07829 07915 07976
08225* 08226* 08233* 08237 08263*

SMALL INTESTINE BIOPSY
07611 07861* 07881 08063 08144 08229* 08233* 08234*
08243 08246 08263*
SMALL INTESTINE CANCER
08147 08149 08155 08220* 08220* 08392 08743*
SMALL INTESTINE CIRCULATION
07592* 07648 08118* 08717*
SMALL INTESTINE CYSTS
08776
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07813* 08119*
SMALL INTESTINE DIAGNOSIS
07861* 07871 07881 07903 07906 07915 07933 07952
07959 07966 08063 08101 08113* 08126 08148 08149
08218* 08222 08229* 08232* 08235 08346 08784
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07621 08231* 08237
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08218*
SMALL INTESTINE DISEASES
07744* 07915 08113* 08129 08150 08160 08162 08236
SMALL INTESTINE DIVERTICULA
07695 08121 08124 08131 08161 08176*
SMALL INTESTINE ENDOSCOPY
07861* 07871
SMALL INTESTINE ENZYMES
07595* 07620 07621 07652 07816* 07829 07833 07915
07976 08129 08225* 08237 08263*
SMALL INTESTINE EXTRACTS
07594*
SMALL INTESTINE FISTULAS
07626* 08132 08143 08221 08222
SMALL INTESTINE HISTOLOGY
07593* 07595* 07620 07621 07653 07810* 07812* 07813*
07814* 07819 08116* 08119* 08142 08150 08155 08160
08235 08263* 08268*
SMALL INTESTINE HYPERPLASIA
08144
SMALL INTESTINE IMMUNOLOGY
07620 07621 07832 08268*
SMALL INTESTINE IN CHILDREN
07744* 08112* 08115* 08144 08149 08234* 08776 08835
08848 08901
SMALL INTESTINE INJURIES
08126 08136 08822
SMALL INTESTINE METABOLISM
07626* 07629* 07813* 07816* 07825
SMALL INTESTINE MICROORGANISMS
07825 08233* 08898 08904
SMALL INTESTINE MORPHOLOGY
07592* 07609 07614 07812* 07832 08129 08142
SMALL INTESTINE MOTILITY
07657* 07658* 07660* 07691* 07959 08118* 08129 08906
SMALL INTESTINE MUCOSA
07592* 07593* 07594* 07597* 07609 07611 07620 07621
07625* 07626* 07627* 07630* 07634* 07653 07673 07812*
07813* 07819 07856* 07871 07966 08063 08116* 08119*
08134 08135 08136 08142 08160 08223 08225* 08226*
08231* 08232* 08234* 08236 08901
SMALL INTESTINE NEOPLASMS
08137 08148 08243 08762
SMALL INTESTINE NEOPLASMS,BENIGN
08101 08122 08130 08131 08151 08161 08743*
SMALL INTESTINE NEOPLASMS,MALIGNANT
08125 08149 08155 08678 08743*
SMALL INTESTINE OBSTRUCTION
08114* 08115* 08118* 08123 08124 08126 08161 08162
08195 08221 08223 08346 08382 08784 08916
SMALL INTESTINE PATHOLOGY
07933 08113* 08114* 08122 08124 08125 08129 08135
08148 08149 08150 08155 08158 08159 08160 08161
08218* 08223 08224 08232* 08263* 08346 08672 08728*
08901 08904
SMALL INTESTINE PERFORATION
08128 08157 08210 08212 08221 08222 08901 08906
SMALL INTESTINE PERFUSION
07624* 07625* 07627* 07634* 07641* 07811* 08234*
SMALL INTESTINE POLYPS
07903 08123 08144 08743* 08762
SMALL INTESTINE RADIOLOGY
07903 07906 07933 07966 08101 08126 08149 08161
08222 08229* 08346
SMALL INTESTINE SECRETION
07631* 07976
SMALL INTESTINE SURGERY
07657* 08003 08030 08056 08074 08077 08101 08112*
08114* 08115* 08117* 08118* 08122 08125 08126 08127
08132 08133 08141 08143 08144 08150 08157 08159
08163 08182 08201 08212 08213 08221 08230* 08346
08362 08403 08469 08692 08717* 08743* 08776 08784
08835 08901 08916
SMALL INTESTINE TRANSPLANTATION
08003 08815 08867
SMALL INTESTINE TRAUMA
08026 08114*
SMALL INTESTINE TREATMENT
08143 08158 08158 08222 08235 08743*
SMALL INTESTINE ULCERS
08223 08730* 08779
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08131
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07597* 07602 07609 07611 07612 07629* 07819 0783
08119* 08134 08135 08139 08223 08233* 08243 0824
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08346 08776 08835
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08159
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07639* 08118* 08157 08158
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08128
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07655
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07602 07612
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07639* 07812* 07814* 07819 08134 08135 08559*
SMOOTH MUSCLE
07602 07612
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07663 07818
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07660* 07663 07818 07986* 08767 08858
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07622* 07624* 07633* 07642* 08255*
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08837
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08023
SPHINCTER OF ODDI
SEE ALSO BILIARY TRACT
07660* 07878 07879 07977 08051 08659* 08685 0868
08696 08698 08710
SPHINCTER,ANUS
07863* 08310 08340 08341
SPHINCTER,ESOPHAGUS
07600 07982* 07983* 07986* 08010 08021
SPLEEN
07653 07877 07897 07949 08450 08472 08480 0853
08577 08870
SPLENOMEGALY
08457 08642 08645 08728* 08755
SPLENOPORTAL SHUNT
07730*
SPLENOPORTOGRAPHY
07858* 08471 08473
SPLENORENAL SHUNT
08471 08472 08482 08496
SPRUE
07625* 07838* 08158 08246 08247 08248
SPRUE,TROPICAL
08229*
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07607 07613 07614 07616 07617 07653 07869 078
08139 08146
STAPHYLOCOCCAL ENTEROCOLITIS
08785
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07758 08119*
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08113* 08116* 08226* 08228* 08232* 08243 08399* 084
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07749* 07808 08540* 08589
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08475
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07794 08011 08172* 08594* 08641
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SEE ALSO BEZOARS
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SEE ALSO PYLORIC OBSTRUCTION
SEE ALSO ULCERS
07826 07832
STOMACH ABSORPTION
08054 08077
STOMACH ACID SECRETION
07613 07674* 07675* 07676* 07678* 07679* 07680* 076
07682* 07683 07684 07685 07687 07688 07689 076
07839* 07845* 07857* 08059 08072 08166* 08168* 081
08176* 08180 08190 08200 08204 08208 08211 084
08730* 08779 08780 08790 08835 08871
STOMACH ANOMALIES
08031 08087 08092

ACH BIOCHEMISTRY
7603 07604 07667* 07687 07689 07835 08044*
ACH BIOPSY
7839* 07875 07896 08052 08068 08073 08211 08888
ACH CANCER
7850* 07869 07900 07916 08037* 08038* 08039* 08042*
8044* 08047 08049 08055 08058 08064 08066 08070
8071 08076 08078 08082 08083 08086 08089 08090
8091 08093 08096 08101 08102 08109 08111 08246
8392 08620 08669 08722* 08725* 08742* 08764 08836
8913
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8080
ACH CIRCULATION
7900 07912 07997 08033*
ACH CONTENTS
7687 08032
ACH CYSTS
8069
ACH DIAGNOSIS
7839* 07845* 07846* 07850* 07854* 07855* 07856* 07857*
7872 07885 07888 07896 07900 07912 07916 07929
7935 07952 07982* 08046 08046 08048 08052 08064
8065 08066 08068 08075 08081 08082 08084 08091
8093 08096 08101 08104 08107 08171* 08389
ACH DISEASES
8099 08104 08107 08110 08762
ACH DIVERTICULA
8762 08773
ACH ELECTROLYTE SECRETION
7665* 07678* 07688 08044* 08168*
ACH ENDOSCOPY
7845* 07849* 07855* 07872 07873 07885 07888 07896
7916 07935 08046 08052 08389 08807 08855
ACH ENZYMES
8076
ACH FISTULAS
7682* 08065 08309 08910
ACH HISTOLOGY
7665* 07839* 07849* 08069 08073 08076 08084 08093
8096 08099 08389
ACH IMMUNOLOGY
7594* 07856*
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7600 08031 08048 08104 08203 08214
ACH METABOLISM
8110
ACH MORPHOLOGY
7845* 07846* 07849* 07896 07912 08207 08790
ACH MOTILITY
7658* 07659* 07662* 07679* 07850* 08048 08110 08187
8805
ACH MUCOSA
7604 07613 07665* 07674* 07676* 07677* 07683 07845*
7872 07885 07889 07896 07916 08044* 08068 08069
8085 08091 08108 08168* 08175* 08190 08207 08762
8780
ACH MUCOSAL INJURIES
7994* 08033* 08040* 08081 08168*
ACH MUCUS SECRETION
7688
ACH NEOPLASMS
7846* 07929 08080 08096 08762
ACH NEOPLASMS,BENIGN
8046 08053 08057 08084 08101 08104 08106
ACH NEOPLASMS,MALIGNANT
8042* 08044* 08056 08058 08075 08076 08093 08096
8106 08620
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8712
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7935 07998 08081 08085 08093 08104 08106 08110
ACH PEPSIN SECRETION
7674* 07682* 07684 07688 07854* 08059 08180 08200
ACH PERFORATION
8034* 08100 08762 08789
ACH PERFUSION
7674* 07677* 07678* 07682*
ACH POLYPS
7916 08046 08084 08089 08101 08246 08389 08762
ACH RADIOLOGY
7600 07845* 07846* 07850* 07857* 07875 07888 07896
7900 07912 07916 07929 07982* 08019 08048 08065
8066 08075 08081 08082 08084 08091 08093 08101
8171* 08389 08790
ACH SECRETION
7594* 07681* 07682* 07686 07688 07689 07835 07983*
8006 08033*
ACH SECRETION DISORDERS
7839* 07846* 07856* 08044* 08168* 08176* 08458 08768
8769 08779 08780 08798 08824 08826
ACH SECRETION STUDY TECHNIQUES
7674* 07675* 07677* 07678* 07679* 07687 07839* 07845*
7857* 07896 08165* 08168*

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07676* 07678* 07682* 07685 07687 08059 08178* 08200
STOMACH SECRETION,HORMONAL CONTROL OF
07665* 07675* 07676* 07679* 07682* 07683 07684 08176*
08190 08720* 08769
STOMACH SECRETION,NERVOUS CONTROL OF
07604 07675* 07684 08165* 08168* 08172* 08188 08203
STOMACH SURGERY
SEE ALSO GASTRECTOMY
07677* 07680* 07871 07916 07984* 07990* 07995* 07997
07998 08001 08015 08016 08031 08036* 08037* 08046
08048 08049 08050 08051 08053 08055 08057 08060
08061 08062 08064 08065 08074 08084 08094 08097
08098 08099 08101 08102 08104 08105 08108 08111
08118* 08165* 08174* 08177* 08186 08187 08192 08196
08197 08201 08203 08210 08213 08214 08443* 08471
08712 08717* 08762 08779 08793 08816 08835 08845
08859
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08067
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07885 08034* 08169* 08182 08184 08276
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08041*
STOMACH TRANSPLANTATION
07677*
STOMACH TRAUMA
07677* 08081 08762
STOMACH TREATMENT
08027 08081 08094 08178*
STOMACH ULCERS
07656* 07676* 07873 07916 08059 08073 08089 08098
08100 08165* 08168* 08173* 08175* 08196 08197 08199
08200 08208 08209 08214
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07613 07665* 08068
STOMACH VARICES
07889 07900 07998 08729*
STOMACH VOLVULUS
08001
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08176*
STOMACH,DRUG EFFECTS ON
07603 07689 08040*
STOMACH,HORMONAL CONTROL OF
07659* 07835
STOMACH,NERVOUS CONTROL OF
07659* 07662* 08110 08187 08193 08805
STOMACH,TOXIC EFFECTS ON
08033*
STRANGULATION
07997 08304 08763
STRESS FACTORS
08270
STRESS FACTORS IN ULCERS
08175* 08179 08180
STRICTURE,BILIARY TRACT
08058 08666* 08691 08692 08695 08698 08701 08715
STRICTURE,ESOPHAGUS
07989* 08005 08009 08019 08030 08762
STRONTIUM ABSORPTION
07632*
STUDIES OF CIRRHOSIS,CLINICAL
08653
STUDIES OF HEPATITIS,CLINICAL
08523 08604* 08628
STUDY TECHNIQUES,ABSORPTION
07622* 07628* 07643* 07645 07647 07648 07915 07919
08054 08077 08134 08238 08648
STUDY TECHNIQUES,BILE SECRETION
07660*
STUDY TECHNIQUES,DYE
SEE MARKER STUDIES
STUDY TECHNIQUES,RADIOISOTOPE
SEE TRACER STUDIES
STUDY TECHNIQUES,SECRETION
07694 07854*
STUDY TECHNIQUES,STOMACH SECRETION
07674* 07675* 07677* 07678* 07679* 07687 07839* 07845*
07857* 07896 08165* 08168*
SUCROSE ABSORPTION
08244
SUGAR INTOLERANCE
08237 08495
SULFOBROMOPHTHALEIN
07747* 07752* 07754 07788 07851* 07852* 07866* 07941
07969 08441* 08507 08528 08558* 08586 08657*
SURGERY COMPLICATIONS
07993* 08252* 08895
SURGERY COMPLICATIONS,STOMACH
08067
SURGERY TECHNIQUES
07694 07762 07983* 07984* 07985* 07990* 07996* 08002
08003 08030 08035* 08056 08061 08097 08105 08117*
08132 08170* 08181 08183 08197 08262* 08269* 08275
08278 08282 08293 08301 08334 08335 08341 08357

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08362 08403 08413 08439* 08472 08480 08488 08504
08517 08632* 08685 08693 08695 08701 08713 08717*
08815 08842 08851 08872 08878 08908
SURGERY TECHNIQUES,STOMACH
07885 08034* 08169* 08182 08184 08276
SURGERY-INDUCED SECONDARY MALABSORPTION,STOMACH
08041*
SURGERY,ABDOMEN
07657* 07941 07990* 08117* 08118* 08128 08143 08144
08310 08334 08401 08514 08549* 08566 08568 08748
08752 08772 09829 08832 08846 08849 08855 08859
08872 08895 08907 08908
SURGERY,ANORECTAL
08310 08345
SURGERY,ANUS
08257* 08291 08292 08293 08294 08345 08351
SURGERY,BILIARY TRACT
07762 07878 07879 07938 07964 08152 08156 08405
08434* 08465 08467 08469 08470 08510 08522 08567
08640 08656* 08658* 08660* 08661* 08663* 08666* 08669
08670 08671 08676 08678 08679 08681 08682 08685
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Public Health Service
National Institutes of Health

NOVEMBER/DECEMBER 1968
VOLUME 3, NO. 11/12
8917–9999

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Abstract and Citation Nos. 8917-9999

GASTROENTEROLOGY ABSTRACTS AND CITATIONS

A monthly publication of the
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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis and Metabolic Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication of GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available, free to qualified interested investigators and practitioners, citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology has accompanying abstracts.

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Use of Funds for printing this publication
approved by the Director of the Bureau of
the Budget on October 7, 1968.

or sale by the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. ubscription price per year in the United States, Canada, and Mexico, $8.50; foreign $2.25 additional; $1.00 per ingle copy. Payment is required in advance, and check or money order should be made payable to the uperintendent of Documents.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. Fo journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medica Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mC, μC	milli-, microcurie(s)
ADP	adenosine diphosphate	mg	milligram(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
BSP	sulfobromophthalein	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
μg	microgram(s)		
hr	hour(s)	resp	respectively
i.m.	intramuscular	Rev.	review (only in citations)
i.p.	intraperitoneal	RNA	ribonucleic acid
IU	international unit(s)	s.c.	subcutaneous
i.v.	intravenous	sec	second(s)
kg	kilogram(s)	SGOT	serum glutamic-oxalacetic transaminase
LD50	median lethal dose(s)	SGPT	serum glutamic-pyruvic transaminase
LDH	lactic acid dehydrogenase	U	unit(s)
m	meter(s)	UV	ultraviolet
M	molar	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mM	millimolar	yr	year(s)
μM	micromolar		

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

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ABSTRACTS
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NATIONAL INSTITUTE OF ARTHRITIS AND METABOLIC DISEASES

U.S. DEPARTMENT OF
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Public Health Service
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VOLUME 3
INDEX ISSUE
1968

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A monthly publication of the
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LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mC, μC	milli-, microcurie(s)
ADP	adenosine diphosphate	mg	milligram(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
BSP	sulfobromophthalein	mm	millimeter(s)
C	degrees centigrade	MTD	maximum tolerated dose
cm	centimeter(s)	ng	nanogram (10^{-9})
CNS	central nervous system	pg	picogram (10^{-12})
cpm	counts per minute	p.o.	orally
DNA	deoxyribonucleic acid	ppm	parts per million
e.g.	for example	r	Roentgen
g	gram(s)	RBC	red blood cells (erythrocytes), red blood count
μg	microgram(s)		
hr	hour(s)	resp.	respectively
i.m.	intramuscular	Rev.	review (only in citations)
i.p.	intraperitoneal	RNA	ribonucleic acid
IU	international unit(s)	s.c.	subcutaneous
i.v.	intravenous	sec	second(s)
kg	kilogram(s)	SGOT	serum glutamic-oxalacetic transaminase
LD$_{50}$	median lethal dose(s)	SGPT	serum glutamic-pyruvic transaminase
LDH	lactic acid dehydrogenase	U	unit(s)
m	meter(s)	UV	ultraviolet
M	molar	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mM	millimolar	yr	year(s)
μM	micromolar		

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tarting with Volume 3, Number 1, the SUBJECT and AUTHOR INDEXES have been generated directly from omputer-stored files. The Indexes to prior volumes were manually prepared.

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02763* 02765 02766 02913* 03110* 03116* 03228* 03244*
03245* 03373* 03673* 03720* 03727* 03728* 03729* 03745
03913* 04044* 04079 04091 04092 04170* 04227* 04240
04312 04312 04313 04320 04325 04680 04713* 04715*
04721* 04736 04737 04738 04739 04863* 04959 04988*
05293 05361* 05386* 05480 05711* 05714* 05719* 05722*
05723* 05724* 05732 05780 06228 06691 06699* 06701*
06702* 06703* 06706* 06707* 06714 06717 06810 06836*

ABSORPTION,CARBOHYDRATE
CONTINUED
06851* 06881* 07112 07123 07194* 07197 07202 07203
07391 07446* 07577 07629* 07630* 07650 07652 08043*
08225* 08231* 08234* 08241 08244 08495 08508 09083
09175 09218 09325 09333 09336 09718
ABSORPTION,CAROTENE
SEE ALSO VITAMIN A ABSORPTION
09082
ABSORPTION,CHLORIDE
01412 02753* 02758* 02770 03730* 04674* 06743* 08227*
ABSORPTION,CHOLESTEROL
02757* 03741 03817* 03886* 04722* 04728* 06698* 06813
06815 07439 07492* 07811* 08133 08306 08688 08854
ABSORPTION,COPPER
00773 03736 08942 09534
ABSORPTION,D XYLOSE
09242
ABSORPTION,DRUG
00808 01624 03738 03750* 04442* 04779* 05725* 05998*
06159* 06702* 08333 08905 08943 09005 09024 09914
ABSORPTION,ELECTROLYTE
00038 00815* 01412 01740 01824* 02170 02290 02753*
02758* 02764 02770 02775 02776 02777 03002* 03674*
03722* 03730* 03751* 03913* 04023* 04331* 04674* 04703
04713* 04716* 04720* 04720* 04981* 05720* 05724* 05726
05731 06708* 06743* 07566 07622* 07623* 07624* 07627*
07630* 07633* 07636* 07637* 07638* 07642* 07688 08168*
08227* 08874 08944 09399 09737 09954
ABSORPTION,FATTY ACID
00023* 00027* 00028* 00813* 00890 01736* 02636* 02845
03744 03862 03886* 04308* 04729* 05300 05308 05834
06236* 06705* 06735* 06822 07079* 07439 07710* 07726*
07803 07919 07919 08236 08945 08991 08993 09043
09716 09867
ABSORPTION,FOLIC ACID
00036 00311* 00816* 03104* 03242* 03247 03589* 04301*
05287* 07199 08116* 09329
ABSORPTION,FRUCTOSE
04320 04688* 07647 08245 08495
ABSORPTION,GALACTOSE
00024* 01913 02759* 04227* 04313 04837* 05529* 06810
07391 07629* 08241 08245
ABSORPTION,GALLBLADDER
01648* 01824* 01825* 03743 05656 05917 09560
ABSORPTION,GASTROINTESTINAL
00029 00117* 00797 00824 00827 01104 01725* 01726*
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02773 02774 02775 03240* 03588* 03729* 03738 03750*
04718* 04728* 04731* 04738 04889 05711* 05716* 05728
05734 06039 06436 06702* 06711 06712 06713 06714
06828 07199 07555 07631* 07632* 07649 07919 08819
08941 08945 09069
ABSORPTION,GLUCOSE
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02748* 02758* 02759* 02763* 02913* 02999* 03110* 03116*
03245* 03373* 03673* 03727* 03728* 03913* 04170* 04313
04713* 04715* 04721* 04735 04736 04863* 05654 05714*
05719* 05724* 05732 06228 06691 06699* 06701* 06703*
06706* 06717 06826* 07639* 07760 08043* 08234* 08241
08245 08508 09175 09336 09452 09668 09718
ABSORPTION,HORMONAL CONTROL OF
00812* 00815* 00823 02760* 03730* 04310* 07623* 07633*
07803
ABSORPTION,IRON
00019 00026* 00039 00311* 00439 00684* 00696* 00844*
00845* 01530* 01719* 01720* 01728* 01730* 01732* 01733*
01744 02629* 02750* 02751* 02754* 02756* 02920* 03017
03077* 03240* 03241* 03301* 03588* 03592* 03723* 03724*
03726* 03733* 03739 03742 03986 04384* 04436* 04602*
04724* 04725* 04726* 04727* 04732 04733 04950 05127*
05444* 05629 05713* 05729 05733 06704* 06709 06712
06713 06714 06715 06716 07645 07646 07647 07840
08036* 08531 08941 08942 08951 09725 09845
ABSORPTION,LACTIC ACID
02771 08938
ABSORPTION,LACTOSE
00129* 00428* 00435* 00923* 02051* 02748* 02913* 03228*
03244* 03245* 04044* 04227* 04312 04325 05986* 08244
ABSORPTION,LARGE INTESTINE
00022* 00035 00798 00815* 00816* 00817* 00828 00951*
01403* 01412 01431 01729* 01740 01742 01746 02320*
02757* 02758* 02767 02777 02778 02883* 03720* 03730*
04023* 05717* 05724* 05726 05731 05733 06379* 06711
07216* 07575 07628* 07633* 08160 08255* 08306 08934
08944 09074 09327 09916
ABSORPTION,LIPID
00027* 00028* 00029 00034 00037 00431* 00813* 00821
00826 00830 00890 00911 01191 01260* 01372* 01723*
01731* 01736* 02087 02172 02279* 02289 02291 02392*
02483* 02603* 02636* 02700* 02707 02757* 02760* 02762*
02845 02851 02988* 03232* 03234* 03246* 03316* 03725*
03741 03744 03802 03886* 04000 04080 04124 04166*
04178* 04240 04242 04308* 04673* 04681 04719* 04722*
04728* 04729* 04854* 04939 04949 05031 05206 05296
05300 05304 - 05361* 05382* 05666* 05676 05725* 05834
ABSORPTION,LIPID
CONTINUED
05864* 05910 06029 06322 06698* 06705* 06713 06822
06881* 06897 07079* 07090 07112 07188* 07191* 07193*
07195 07197 07202 07290 07439 07492* 07629* 07634*
07635* 07640* 07643* 07651 07653 07654 07688 07726*
07803 07919 08116* 08229* 08230* 08236 08239 08243
08377 08548* 08938 08945 08991 08993 09017 09043
09126 09218 09329 09332 09535 09631 09716 09926
ABSORPTION,MAGNESIUM
01037* 05076 05207 07642* 08942
ABSORPTION,MANGANESE
07787
ABSORPTION,MINERAL
SEE ALSO TRACE ELEMENT AND MINERAL ABSORPTION
01732*
ABSORPTION,PHOSPHORUS
00032 00978 01315* 02802* 03401 09458
ABSORPTION,POTASSIUM
01412 02753* 04674* 05731 07633* 07642* 09737
ABSORPTION,PROTEIN
00033 00345* 00426* 01191 01742 02483* 02955 03152*
03229* 04062 04240 04242 04672* 05676 05712* 06324
06379* 06712 06820 07195 07197 07626* 07648 07653
07696 08623 08905 08979 09326 09328 09450 09967
09970
ABSORPTION,RECTUM
00030 02763*
ABSORPTION,SMALL INTESTINE
00011* 00022* 00023* 00028* 00035 00063* 00347* 00785*
00812* 00813* 00814* 00815* 00816* 00817* 00818* 00820
00823 00825 00826 00829 00830 00895* 00925* 00926*
01260* 01372* 01403* 01721* 01722* 01723* 01724* 01729*
01731* 01732* 01733* 01736* 01739 01744 01870* 02169
02172 02247 02279* 02284* 02285* 02289 02290 02291
02293 02296 02324* 02352 02483* 02603* 02702* 02734*
02748* 02750* 02751* 02753* 02756* 02757* 02758* 02759*
02760* 02762* 02765 02767 02769 02777 02829* 02988*
03002* 03150* 03166 03229* 03230* 03232* 03234* 03242*
03246* 03500 03633* 03639 03674* 03688 03720* 03725*
03726* 03727* 03728* 03731* 03733* 03735 03736 03737
03739 03744 03745 03980 04024* 04044* 04060 04189
04236 04240 04249 04305* 04306* 04308* 04312 04313
04320 04323 04442* 04672* 04676* 04710* 04711* 04712*
04713* 04715* 04716* 04717* 04718* 04720* 04721* 04723*
04729* 04730* 04732 04734 04736 04739 04782 04783
04850* 04959 04977* 04981* 05127* 05184* 05185* 05196
05198 05202 05287* 05288* 05300 05307 05361* 05666*
05676 05687* 05709* 05710* 05712* 05714* 05715* 05718*
05719* 05721* 05722* 05723* 05724* 05725* 05726 05727
05729 05730 05733 05864* 05910 06214* 06302 06686
06691 06699* 06700* 06701* 06703* 06705* 06706* 06707*
06710 06715 06717 06723* 06836* 06844* 06881* 07031*
07079* 07123 07162 07188* 07189* 07190* 07191* 07193*
07194* 07446* 07492* 07575 07620 07621 07623* 07624*
07625* 07626* 07627* 07628* 07629* 07630* 07634* 07635*
07639* 07641* 07642* 07647 07648 07655 07915 08077
08112* 08116* 08134 08159 08160 08230* 08232* 08234*
08236 08934 08935 08936 08937 08938 08943 08944
09018 09043 09074 09084 09218 09242 09322 09324
09325 09326 09327 09331 09332 09334 09336 09429
09916 09926
ABSORPTION,SODIUM
00038 00095 01412 01824* 02170 02738 02753* 02758*
02764 02770 03002* 03674* 03730* 04713* 05682* 07622*
07624* 07633* 07642* 08255* 08939 08944 09737 09989
ABSORPTION,STEROID
02757*
ABSORPTION,STOMACH
00813* 01727* 01729* 01732* 01787 02770 03733* 03736
03739 04602* 04718* 05735 06159* 08054 08077 08945
ABSORPTION,STRONTIUM
00893* 00894* 01739 02752* 02772 03679 03734* 05856*
07632* 08942
ABSORPTION,SUCROSE
04688* 08244
ABSORPTION,TOXIC EFFECTS ON
02171 02172 02297 04453*
ABSORPTION,TRACE ELEMENT AND MINERAL
00019 00022* 00026* 00030 00031 00032 00039 00311*
00396* 00425* 00439 00773 00893* 00894* 00978 01037*
01192 01315* 01530* 01640* 01719* 01720* 01726* 01728*
01730* 01732* 01737* 01739 01744 02260 02291 02629*
02738 02750* 02751* 02752* 02753* 02754* 02756* 02802*
02920* 03017 03077* 03234* 03240* 03241* 03301* 03500
03588* 03592* 03679 03723* 03724* 03726* 03733* 03734*
03736 03739 03742 03986 04219 04240 04309* 04310*
04384* 04436* 04602* 04717* 04719* 04724* 04725* 04726*
04727* 04731* 04732 04733 04950 05076 05127* 05207
05289* 05291* 05444* 05629 05729 05733 05846* 06029
06704* 06709 06712 06713 06714 06715 06716 07197
07632* 07641* 07642* 07645 08036* 08110 08236 08525
08531 08937 08942 08951 08988 09106 09330 09534
09709 09845

ABSORPTION,TRIGLYCERIDE
00028* 00426 01372* 03741 03888* 04672* 04713* 04854*
04859* 04939 04949 05296 05308 05551* 05856* 06815
06820 06922 06824 07188* 07439 07629* 07634* 07640*
07704* 08233* 08239
ABSORPTION,VITAMINS
00036 00311* 00321 00430* 00816* 00822 00825 00845*
01230 01375 01856* 02170 02171 02283* 02289 02291
02291 02664* 02749* 02761* 03104* 03107* 03230* 03237*
03242* 03247 03500 03589* 03688 03737 04060 04213
04219 04221 04240 04242 04301* 04305* 04306* 04310*
04311 04323 04710* 04724* 04850* 04950 05024 05031
05145* 05285* 05287* 05301 05302 05302 05382* 05392
05710* 05730 05779* 05867* 06029 06707* 06844* 07031*
07190* 07194* 07197 07199 07201 07635* 07644* 08041*
08077 08165* 08229* 08230* 08242 08377 08940 08961
09082 09176 09327 09329 09330 09926
ABSORPTION,VITAMIN A
SEE ALSO CAROTENE ABSORPTION
02283* 04310* 04473 05779* 06735* 06813 07635* 07644*
07774 08229* 08377 08891
ABSORPTION,VITAMIN B12
00036 00311* 00321 00430* 00822 00825 00828 00829
00845* 01230 01375 01856* 02170 02171 02289 02664*
02749* 02761* 03104* 03107* 03230* 03237* 03247 03589*
03737 04213 04242 04310* 04311 04323 04710* 04850*
04950 05031 05285* 05392 05710* 05730 05867* 06707*
06844* 07031* 07190* 07199 07201 07594* 08077 08229*
08230* 08242 08483 08746* 08940 09176 09329 09429
09926
ABSORPTION,VITAMIN C
04060 04724* 05024
ABSORPTION,VITAMIN D
02285* 03688 04219 04221 08103
ABSORPTION,WATER
00814* 00815* 00817* 00818* 00823 01412 01723* 01803*
02170 02290 02320* 02750* 02753* 02758* 02774 02777
02829* 02893* 03730* 03745 04343 04711* 04713* 04716*
05185* 05724* 06761* 06710 07197 07216* 07575 07622*
07623* 07624* 07627* 07633* 08231* 08255* 09954
ABSORPTION,XYLOSE
00025* 02283* 02284* 04091 05309 05722* 05985* 06701*
07112 07446* 09083
ABSORPTION,ZINC
01640* 05291* 08236 08942 09491 09709
ACETYLCHOLINE
00002* 00050* 00072* 00073* 00882* 02785* 03804 04749*
04780* 05760* 06722* 06738* 07542*
ACHALASIA
00985 00990 01000 01138* 01141* 01142* 01152 01157
01202 01895 02012* 02020 02025 02887* 03049 03069*
03070* 03078* 03500* 04136 04144 04145 04147 05085*
05086* 05102 05957 06110* 06111* 06115 06696 06724*
06905 06969* 06974* 07001 07875 07986* 08009 08023
08025 08762 08815 09151 09153 09966
ACHLORHYDRIA
00118* 00291* 00320 00845* 00869 00935* 00970 01034*
01166* 01361 01700* 02063 03000* 03000* 03109* 03113*
03723* 03768* 03887* 03978 04049* 04193 04989* 05131*
05418 05435* 05852* 05867* 05884* 06166 06598 06858*
06873* 06956 06958 07019* 07027* 07068 07690* 07845*
07857* 08090 09210
ACID PHOSPHATASE
00891* 00892* 01541* 03872 04810 04811 07751* 09556
ACID SECRETION,STOMACH
00042* 00045* 00051* 00056 00134 00188* 00297* 00299*
00301* 00309* 00313* 00390* 00392* 00403 00831* 00832*
00835* 00842* 00843* 00844* 00845* 00848* 00850* 00853
00856 00857 00858 00859 00862 00867 00868 00870
00871 00872 00873 00874 00876 00888 00934* 00935*
00936* 00953* 00974 00993 01001 01014 01034* 01051
01090 01166* 01172* 01174* 01177* 01190 01199 01259*
01296* 01301* 01305* 01308* 01310* 01313* 01314* 01315*
01316* 01317* 01700* 01702* 01716 01748* 01749* 01750*
01754* 01755* 01756* 01757* 01758* 01761* 01762* 01764*
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01788 01792 01810* 01859* 01872* 01924* 02011* 02046*
02197* 02204* 02212* 02235 02236 02246 02408* 02631*
02705* 02723 02779* 02780* 02785* 02786* 02789* 02790*
02792* 02793* 02794* 02795* 02796* 02798* 02799* 02800*
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03004 03063 03064 03065 03113* 03119 03122 03136
03140 03180* 03181* 03190* 03193* 03197 03198 03209
03220 03693 03705* 03732* 03747* 03748* 03749* 03754*
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03765* 03767* 03768* 03770* 03773 03776 03796* 03887*
03978 04039* 04141 04173* 04216 04224 04256* 04258*
04259* 04267* 04286 04288 04290 04291 04718* 04742*
04743* 04745* 04746* 04747* 04748* 04749* 04751* 04752*
04755 04756 04758 04761 04763 04765 04881 04914
05098 05133* 05140* 05141* 05155 05156 05213* 05215*
05218* 05221* 05228* 05229* 05236 05239 05243 05252
05268 05273 05418 05435* 05626 05736* 05741 05742*
05744* 05745* 05746* 05749* 05753* 05754* 05755* 05756*
05758* 05759* 05760* 05762* 05763* 05764* 05766 05769
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ACID SECRETION,STOMACH
CONTINUED
05884* 05928 06014 06145* 06151* 06160* 06166 06211
06213 06235* 06236* 06240* 06245* 06252* 06255* 06256*
06259* 06260* 06287 06292 06295 06298 06598 06667*
06728 06738* 06741* 06742* 06743* 06746* 06747* 06748*
06749* 06750* 06751* 06752* 06754* 06756 06757 06758
06760 06761 06762 06763 06764 06765 06767 06769
06841* 06858* 06904 06922 06934 06950 06971* 07016*
07027* 07028* 07038 07063 07132* 07151 07157 07164
07171 07172 07173 07174 07175 07176 07177 07191*
07275* 07396 07446* 07469* 07613 07674* 07675* 07676*
07678* 07679* 07680* 07681* 07682* 07683 07684 07685
07687 07688 07689 07695 07839* 07845* 07857* 08059
08072 08166* 08168* 08175* 08176* 08180 08190 08200
08204 08208 08211 08458 08730* 08779 08780 08781
08835 08871 08948 08949 08953 09045 09076 09078
09097 09107 09114 09192 09205 09294 09300 09307
09308 09313 09396
ACIDOSIS
03574 04028* 05050 05445* 07470* 07746* 08811 08812
09507 09968
ACUTE ABDOMEN
01028* 01057 01078 01084 01086 01103 01107 01248*
01285 01369 01393* 01397* 01922* 01951 01974 03020
03908* 04071 04118 04239 04347 04653 05041 05195
06036 06082 06296 06404 06621* 06911 07506 07561
07567 08818 08829 08832 08837 08840 08896 09224
09228 09289 09477 09774 09798 09873 09922 09925
09988
ACUTE ABDOMEN DIAGNOSIS
00949* 01120 03909* 03910* 03937 03944 03970 04861*
04895 04896 04982* 05026 05329* 05871* 06422* 06658
06926 07217* 07255 07945 08303 08725* 08770 08808
08869 09391 09901
ACUTE ABDOMEN ETIOLOGY
08869
ACUTE ABDOMEN IN CHILDREN
00998 01040* 01120 02128* 03050 04113 04654 04982*
ACUTE HEPATITIS
00698 00701 00931* 03374* 03389 03418 03524* 03534
03542 04593 05494 05497 05926 06488 06868* 07453*
07867* 07913 08424* 08442* 08475 08486 08573 08598*
09555 09573 09684 09686
ACUTE PANCREATITIS
00418 00582 00588 00589 00592 00594 00949* 01483*
01491* 01499 01514* 01516* 01517 01519 01522 01523
01524 01525 02413* 02443* 02444* 02446* 02447* 02450
02451 02453 02454 02455 02457 02465 02466 03001*
03331* 03332* 03334* 03336* 03337* 03338* 03340* 03342*
03343* 03348 03351 03352 03353 03354 03475 03672*
03909* 03947 03953 03965 03972 03984 04043* 04209
04299 04392* 04406 04408* 04409* 04410* 04414 04417
04418 04421 04422 04423 04425 04426 04428 04429
04430 04558* 04920 04972* 05054 05398 05404 05406
05410 05412 05414 05415 05417 05419 05420 05421
05856* 05871* 06234 06418* 06419* 06420* 06422* 06424*
06426* 06428* 06431 06432 06435 06436 06437 06441
06443 06444 06446 07289 07298* 07301* 07303* 07306*
07307* 07308* 07310 07311 07312 07315 07316 07317
07319 07324 07327 07329 07496* 07836* 07843* 07930
08379 08395* 08397* 08404 08406 08407 08408 08409
08410 08411 08412 08416 08417 08418 08419 08420
08685 08725* 09452 09469 09471 09473 09474 09477
09479 09483 09484 09731 09756 09782
ACUTE VIRAL HEPATITIS
00603* 00704 00720 00726 00927* 01612* 01621 02603*
02614 03356* 03383* 03408 03416 03520* 03521* 03530
03555 03557 03563 03576 04442* 04556 04557* 04558*
04578 04580 04584 04586 05534* 05571* 05581 05583
05586 05588 05589 06556* 06558* 06567 06568 06569
06571 06572 06579* 06787* 06957 07443* 07454* 07455*
07456 07963 08463 09610 09676 09681
ADENOCARCINOMA
01416 01418 01420 02341 02379 04126* 05087* 05104
05863* 06148* 06622* 06627* 07026* 07068 07073 07081*
07209* 07238 07240 07257 07258 07441 08071 08083
08147 08220* 08318 08742* 08856 09194 09357 09360
09376 09435 09451 09502 09720 09832
ADENOMA
02093 02325* 04331* 05078 05319* 05949 06330* 06361
06464* 07068 07223 07229 07231 07237 07242 07258
07441 07917 08130 08146 08147 08154 08315 08317
08336 08337 08388 08681 08824 08856 09199 09216
09357 09551 09559 09614
ADHESIONS
01041* 01278 01394* 01997 02433 02974* 03025 04078
04186 06959 07087* 07121 07879 08117* 08221 08334
08846 08852 09187 09238 09250 09347 09730
ADRENAL GLANDS
02062 02568 03439 03712* 03717 03816* 03824* 04576
04826 05271 05458 05475 05573* 05594* 05700* 05737*
05821* 06419* 06541 06741* 06746* 07130* 07169 07394
07633* 07676* 07702* 07803 07975 08136 08199 08605
08618 08644 08982 09303 09419 09514 09612 09669
09688 09864

ADRENERGIC AGENTS
00070* 01851 06826*
AFFERENT LOOP SYNDROME,GASTRECTOMY-INDUCED
00326 03142 04201 04204 04224 04299 05150* 06266
07036 07042 08049 08058 08185 08195
AFLATOXIN
SEE ALSO LIVER CARCINOGENESIS
SEE ALSO STOMACH CARCINOGENESIS
02532 04514 04555 05425* 07428
AGE EFFECTS ON ABSORPTION
00025* 00816* 00827 01371* 01721* 01724* 02284* 04731*
05719* 05721* 05722*
AGE EFFECTS ON BILIARY TRACT
04624* 04689* 05651 08660* 08663*
AGE EFFECTS ON BLEEDING
01931*
AGE EFFECTS ON ESOPHAGUS
05119
AGE EFFECTS ON GALLBLADDER
08663*
AGE EFFECTS ON GASTROINTESTINAL TRACT
00233 01028* 01371* 01978 03034 03677 04036* 04691*
08792 08819 09198
AGE EFFECTS ON LARGE INTESTINE
01402* 03252* 03273 03277 05058 05338 05358 07247
08264*
AGE EFFECTS ON LIVER
00647 01584 01839 02635* 02673 03453 03473 03829
03832 03840 03848 03861 03862 03869 04797* 04806*
04820 04821 04847* 05450* 05478 05481 05692* 05694*
06526 06592 06777* 06781* 06806 07137* 07499* 07852*
08970 09007
AGE EFFECTS ON PANCREAS
01506 02442* 03323 03677 04693* 04696 06414
AGE EFFECTS ON RECTUM
05058
AGE EFFECTS ON SMALL INTESTINE
05196 05709* 05722* 08926
AGE EFFECTS ON STOMACH
00061 01203 02084 03106* 06695 06739* 06751* 08176*
09208
AGE FACTORS
04800* 05422* 05672* 05692* 05884* 06082 06383 06526
06603 06926 07155 07561 08098 08514 08590 08731*
08793 08909 09025 09132 09153 09230 09231 09238
09306 09382 09534 09759 09762 09798 09896 09897
09900 09913 09922 09940 09948 09973 09978 09988
AGE FACTORS IN APPENDICITIS
00453* 01028* 01397* 04071 04347 07255 08832 09351
AGE FACTORS IN CANCER
04093 04175* 04348 05131* 05139* 05313* 05338 07007*
07086* 07247 07343* 08066 08070 08071 08078 08089
08320 08764 09501 09562
AGE FACTORS IN CHOLELITHIASIS
05635* 07506
AGE FACTORS IN CIRRHOSIS
02673 04609 05595* 05614 08428* 09729
AGE FACTORS IN HEPATITIS
02607 03523* 03547 03557 03558 03559 03560 03564
04567* 05571* 06556* 06558* 06852* 08524 08599* 08602*
08604* 09681
AGE FACTORS IN MALABSORPTION
05719*
AGE FACTORS IN PANCREATITIS
07299*
AGE FACTORS IN PEPTIC ULCERS
01028* 01312* 01317* 01347 01354 01360 01925* 02200*
04255* 05264 06241* 06248* 06257* 07136* 07150 09295
09311
AGE FACTORS IN ULCERATIVE COLITIS
09421
AGE FACTORS IN ULCERS
02225 04272* 09289
ALBUMIN
SEE ALSO LIVER
SEE ALSO PLASMA PROTEINS
00064* 00636 00900 01261* 01979 03584* 03586* 03859
04927 04948 04952 05074 05383* 05601* 05612* 06315*
06379* 06873* 07699* 07701* 07919 08623 09450
ALCOHOL
SEE ALSO FATTY LIVER
SEE ALSO PANCREATITIS
00315 00427* 00579* 00591 00606* 00631 00686* 00693
02750* 03098* 03113* 03332* 03515 03668* 04127* 04190
04424 04684* 04778* 04848* 05131* 05302 05715* 05740*
06241* 06261* 06703* 07681* 07720* 07771 08038*
ALCOHOL ABSORPTION
01729* 02046* 02582* 02700* 07827
ALCOHOL EFFECTS ON LIVER
00171 00606* 00626 00631 00648 00668 00686* 00693
00738 00739 00914 00947* 01055 01534* 01542* 01552*
01576 01588 01599* 01641* 02470* 02471* 02473* 02536
02539 02577* 02579* 02581* 02585 02586 02587 02589
02590 02627* 02628* 02629* 02630* 02633* 02634* 02751*
03387* 03441 03476 03478 03491 03493 03502 03503
03504 03515 03591* 03607 03619 03836 04434* 04452*
ALCOHOL EFFECTS ON LIVER
CONTINUED
04453* 04523* 04528* 04534 04536 04607* 04798* 04801*
04956 05432* 05447* 05448* 05449* 05457* 05472 05528*
05529* 05531* 05535* 05547* 05550* 05556 05559 05812*
05924 05925 06490 06501 06518 06519 06520 06521
06522 06523 06536* 06546 06585 06589 06591 06810
07016* 07420* 07434 07439 07443* 07451* 07479 07480
07587 07709* 07748* 07749* 07804 07808 08447 08530
08540* 08565 08573 08579 08582 08589 08955 08957
08975 08987 08989 09570 09578 09579 09580 09581
09582 09621 09626 09629 09631 09649 09653 09709
09725
ALCOHOL EFFECTS ON PANCREAS
06774 07695
ALCOHOL EXCRETION
07827
ALCOHOL IN CIRRHOSIS
00686* 00745 01576 02579* 02626* 02652 03486* 03591*
03619 03621 04609 05531* 05613 05614 05615 06579*
06585 06591 08589 09721 09725
ALCOHOL METABOLISM
07827
ALCOHOL,TOXIC EFFECTS OF
09731
ALCOHOLIC PANCREATITIS
00579* 02445* 03332* 04392* 04409* 04778* 05395 05411
05418 05740* 06423* 06426* 06442 06774 07327 08397*
ALCOHOLIC STEATOSIS
00606* 03362* 03502 03503 03836 04517* 05448* 05925
06490 06520 06521 06522 06523 07401 07749* 07808
08540* 08589 08989 09621 09626 09631
ALKALINE PHOSPHATASE
00166 00757* 01255* 01852* 02479* 02824 02858 02916*
03357* 03365* 03531 03681 03685 03962 04033* 04813
04843* 04845* 04879 05142* 05305 05306 05616 05666*
05687* 05919 06466* 06621* 06799* 06852* 06853* 06870*
06923 06928 07369 07448* 07751* 07754 07829 07837*
07840 07941 07962 08103 08135 08441* 08530 08586
09017 09079 09102 09458 09556 09576 09797
ALKALOSIS
01126 01964 08811 08812 08915
ALLERGIES
SEE ALSO IMMUNOLOGY
03019
AMEBIASIS
01445 01564 01995 03055 05004 05354 08253* 08904
09323 09354 09644 09919 09981
AMEBIASIS COMPLICATIONS
08316
AMEBIASIS DIAGNOSIS
01003 01865* 02479* 02560 02891* 03064 03372* 03417
03925* 03938 04930 04971* 04973* 05443* 05498 05905
06340 06851* 06862* 06874* 07351* 07515 07920 07921
08316 08339 08562* 08584 08585 08723* 08724* 08756
08798 08860 08863 09080 09217 09855 09960
AMEBIASIS EFFECTS ON LIVER
00203 00206 01003 02479* 02530 02560 02891* 03372*
03375* 03393 03417 03897* 03917* 04970* 04971* 04973*
05443* 05469 05477 05498 05570* 05905 06458* 06874*
07351* 07395 08456 08562* 08570 08571 08572 08573
08582 08583 08584 08723* 08750 08756 08814 09983
AMEBIASIS EPIDEMIOLOGY
00203 02991* 04930 04973* 05993* 08723* 08724* 08814
08834 09924
AMEBIASIS IMMUNOLOGY
01874* 03925* 06874* 07921
AMEBIASIS IN CHILDREN
08795 08863
AMEBIASIS PATHOLOGY
08814 08869
AMEBIASIS PROPHYLAXIS
05993*
AMEBIASIS TREATMENT
00946* 01019* 01056 01874* 01995 02479* 02891* 02963
03048 03363* 03372* 03417 03897* 03905* 03917* 03928
03932 04862* 04970* 04973* 05004 05443* 05469 05477
05498 05993* 06006* 06074 06076 06085 06097 06100
06103 06340 06458* 07351* 07515 07551 07574 07578
08316 08333 08339 08570 08571 08572 08573 08584
08585 08723* 08724* 08750 08756 08774 08781 08795
08809 08857 08860 08861 08862 08887 08892 09818
09924 09960 09983
AMINO ACID ABSORPTION
SEE ALSO PROTEIN ABSORPTION
00812* 01724* 01742 02247 02293 02294 03229* 03731*
03735 03745 03822* 04714* 04723* 04730* 04829 05286*
05288* 05307 05709* 05715* 05716* 05721* 05723* 06700*
06708* 06711 06808 07202 07625* 07639* 07648 07649
07776 08240 08936 09202
AMINO ACID METABOLISM
00085* 00100 00601* 01129 02755* 02841* 02843 03735
03822* 03824* 03833 03870 03876 03933 04234 04447*
04491 04495 04607* 04676* 04798* 04829 05288* 05362*
05592* 05818* 05828 06166 06401* 06711 06807 07061
07672 07744* 07767 07777 07797 07799 07899 08442*

NO ACID METABOLISM
NTINUED
08557* 08576 08593* 08744* 08903 08963 08981
NO ACID METABOLISM,LIVER
05825 06800* 06808 07698*
NO ACID TRANSPORT
07649 09967 09970
NO ACIDS
07835
ONIA EXCRETION
06205 07019* 07040 09592
ONIA EXCRETION,LIVER
00085* 00100 00597* 00601* 00671 00676 01528* 02509
02630* 03373* 03405 03406 03407 03408 03416 03606
03612 04014 04064 04491 04509 04546 04607* 04608*
04620 04622 04623 04812 04857* 05324 05592* 05828
05881* 06449* 06476 06524 08445 09047 09508 09553
09561
ULLA OF VATER
E ALSO BILIARY TRACT
00573 00930* 01660* 01692 02678 03053 03628* 03950
04082 04102 04233* 04646 04657 04689* 04890 05636*
05638* 05647* 05649* 05658 06410 06418* 06624* 06630*
06644 06661 06872* 07496* 07501* 07528 07538 07878
08672 08678 08698 09772 09782 09803
LASE
E ALSO PANCREAS
00793 00922* 00949* 01648* 01773* 03001* 03683 03806*
03909* 04119 04410* 04693* 04851* 05054 05297 05404
05672* 05740* 05971 06429* 06568 06642 06853* 07296
07301* 07306* 07307* 07329 07446* 07652 07668* 07672
07836* 08409 09096
LASE SECRETION,PANCREAS
00579* 00836* 00860 00930* 01489* 01498 01751* 01753*
01762* 01771* 02407* 02409* 02443* 02729* 02782* 02784*
02786* 02805 02970 03330* 03334* 03343* 03711* 03953
03983 04380* 04389* 04424 04427 04921 05391 05765*
05971 06435 06642 06871* 07278* 07293 07311 07690*
07692* 07693* 07695 07853* 07936 07954 07958 07970
08415 09096 09210
LOIDOSIS
00625 01082 01147 01188 01460 01536* 02042 03028
03035 04057 04097 04432* 05621 06071 07069 07085*
08298 08771 08777 08870 09583 09826
LOPECTIN
07652
STOMOSIS
00363 00672 00675 00735 00736 01045* 01117 01126
01480 01502 01591 03066 03139 03143 05164 06365
07003 07042 07044 07077* 07083* 07114 07231 07300*
07465* 07539 07657* 08007 08013 08017 08067 08074
08097 08117* 08127 08133 08141 08150 08157 08195
08212 08221 08301 08302 08309 08311 08315 08335
08337 08362 08380 08383 08403 08448 08642 08658*
08679 08698 08713 08717* 08777 08852 08854 08858
08901 08908 09027 09101 09144 09152 09158 09169
09189 09200 09236 09253 09255 09274 09296 09370
09374 09434 0943[illegible] 09448 09456 09459 09489 09517
09544 09567 09591 09706 09707 09753 09754 09755
09767 09773 09779 09802 09834 09854 09925 09930
STOMOTIC PEPTIC ULCERS
01005 05213* 06248* 07128* 07160 07164 09274
STOMOTIC ULCERS
08049
MIA
E ALSO FOLIC ACID
E ALSO LIVER DISEASES
E ALSO MALABSORPTION TREATMENT
E ALSO VITAMIN B12
00681 00844* 00845* 00936* 01209 01230 01283 01467*
01619* 01659* 01719* 01728* 01924* 01969 02009* 02470*
02620 03000* 03003 03077* 03107* 03109* 03149* 03240*
03301* 03350 03375* 03554 03590* 04031* 04087 04091
04135 04553 04559* 04590 04950 05167 05368 05589
05635* 05733 06044 06552 06593 07114 07119 07179
07190* 07194* 07198 07201 07351* 07462 07645 07918
08049 08137 08308 08642 08807 08855 08899 08941
09138 09236 09916 09921 09968 09973 09982
MIA IN CIRRHOSIS
00738 01643 01644 02631* 02632* 03589* 03591* 03599*
05629 07464*
MIA IN MALABSORPTION
00430* 00439 01856* 02291 03235* 03240* 05206 05309
05867* 07201 08248 08746* 09176
MIA,DISEASES ASSOCIATED WITH
07266
MIA,GASTRECTOMY-INDUCED
00311* 00312* 01206 02089 02754* 02756* 03097* 03104*
04204 05227* 06266 07177 08036* 08058 08067
MIA,HEMOLYTIC
01244 03133 03299* 05317* 05318* 05373 05875* 07050
07480 08444 08466
MIA,IRON-DEFICIENCY
00308* 02057* 03017 03104* 04088 05127* 05452* 05629
05875* 06332* 07112 07120 07194* 07199 07464* 07645
07646 08036* 08177* 08865 08951 09160 09836
ANEMIA,PERNICIOUS
00308* 00320 00321 01180* 01963 03241* 04193 04850*
05056 05127* 05129* 05148* 05149* 05155 05884* 06162
06176 06673* 06844* 07030* 07031* 07068 07856* 07857*
08076 08090 08242 09098 09176 09180
ANEMIA,SICKLE CELL
06631*
ANESTHESIA
02084 02580* 02808 03040 03394 03740 05667* 05782*
06369 07533 08060 08549* 08587 08829 09075 09288
09643 09847 09922 09925
ANESTHETICS IN LIVER INJURIES
00683* 00688 02578* 02591 03482* 03511 04507 04582
05534* 06532* 06534* 06542 07422* 08549* 08587 09643
ANEURYSMS
00549 01218 01929* 02464 03257* 04099 04497 07926
08532 08873 09517 09606 09928
ANEURYSMS,AORTIC
00522 01098 01107 01984 03043 09098
ANGINA,ABDOMEN
SEE ALSO MESENTERY VASCULAR DISEASES
00973 01248* 08859 09091 09098
ANGIOGRAPHY IN CANCER DIAGNOSIS
03486* 07847*
ANGIOGRAPHY,ABDOMEN
00141 00155 00952* 00973 01929* 02917* 02944 04936
04937 05873* 05920 05964 06895 06910 07286 07918
08864 08911 09061 09091
ANGIOGRAPHY,BILIARY TRACT
06892
ANGIOGRAPHY,GALLBLADDER
02944 09067
ANGIOGRAPHY,GASTROINTESTINAL
00932* 01252* 02315* 05844* 05868* 05873* 06070 06886
06891 06896 06909 07556 08131 09121
ANGIOGRAPHY,LARGE INTESTINE
00420* 00442* 02315* 02353 06887 06888 06889 09420
09427
ANGIOGRAPHY,LIVER
00112* 00116* 00173 00175 00627 01117 01554 02500
02542 03376* 03486* 04701 04900 04938 04953 05516
05848* 05869* 05898 05966 06493 06840* 06842* 06864*
06876* 06884 06893 06894 06959 06965 07550* 07889
07922 07926 08433* 08647 09026 09038 09053 09087
09519 09599 09705 09720 09730 09733
ANGIOGRAPHY,MESENTERY
00069* 00135 00219 02944 02997* 04967 07286 07606
07903 07926 08130 08738* 08850 09420 09457 09517
ANGIOGRAPHY,PANCREAS
00538* 00836* 04893 04967 05904 05911 05949 05953
06409 06840* 06866* 07297 07322 07550* 07847* 07865*
08373* 08382 09052 09057
ANGIOGRAPHY,RECTUM
03257* 06888
ANGIOGRAPHY,SMALL INTESTINE
00420* 01013 03154* 06889 06890 06929 07903 07906
08382
ANGIOGRAPHY,STOMACH
02113
ANGIOMA
02022 02059 02104 08491 08520 08807 08912 09566
ANICTERIC HEPATITIS
00641 00642 02481* 06854* 06869* 07442* 07444* 07452*
07840 08580 08595* 09684 09686
ANOMALIES,ABDOMEN
01223 04056
ANOMALIES,ANUS
00515 00523 00529 01854* 05059 06326* 07245 08340
09458
ANOMALIES,BILIARY TRACT
00759* 00765 01687 01689 01921 03007 03629* 03656
03659 03902* 04448* 04459* 04610 04624* 04644 04660
05595* 06462* 06464* 06624* 06632 06674* 07334* 07335*
07349* 07518 07519 07524 07538 07599* 07841* 07878
07928 08426* 08430* 08431* 08434* 08667* 08683 08689
08919 09603 09742 09767 09779 09789
ANOMALIES,CONGENITAL
01223 02156 02186 03095 04077 04121 04139 04150
04152 04153 04162 04169* 04176* 04180* 04200 04207
04217 04218 04228* 04232* 04243 04280 04349 04359
04367 04394 04463 04509 04515 04610 04624* 04660
04976* 05001 05009 05050 05052 05061 05105 05116
05118 05163 05187* 05193 05210 05324 05331 05336
05492 05509 06019 06056 06081 06126 06232 06335
06362 06372 06505 06551 06632 06886 06989 07059
07068 07084* 07109 07121 07211* 07224 07236 07245
07334* 07335* 07406 07413 07447* 07466* 07488 07503
07524 07538 07558 07599* 07744* 07809 07878 07928
08026 08031 08088 08092 08120* 08231* 08244 08269*
08278 08287 08308 08331 08341 08348 08434* 08460
08462 08494 08526 08601* 08667* 08681 08683 08687
08698 08746* 08762 08782 08830 08835 08901 08914
08915 08916 09103 09137 09142 09146 09152 09158
09219 09224 09245 09336 09358 09375 09380 09388
09451 09496 09531 09559 09563 09565 09575 09603
09607 09617 09767 09777 09779 09862 09869 09874

ANOMALIES,CONGENITAL
CONTINUED
09992
ANOMALIES,DUODENUM
06394
ANOMALIES,ESOPHAGUS
00271 01156 01165 01946 02013* 02017 02019 02027
02043 02114* 02907* 03080 03081 03087 03093 04077
04121 04139 04145 04162 05097 06140 06253* [illegible]
06962 06967 06985 07991* 07992* 08000 08003 08011
08092 09132 09137 09142 09146 09152 09155 [illegible]
ANOMALIES,GALLBLADDER
00779 00783 02659* 04645 07495* 07503 07504 08434*
08080 08661 08663 08667 09779
ANOMALIES,GASTROINTESTINAL TRACT
00157 00232 00998 01088 01094 01187 01217 01221
01950 03266 04055 04077 04121 05001 06019 06032
06056 06669 07902 08782 09992
ANOMALIES,LARGE INTESTINE
00245 00471 00511 01429 01434 01449 01455 01759*
01900 01941 02114* 02303* 02333 02334 03287 04226*
04280 04355 05323 05324 05331 05336 06335 06362
06372 07211* 07219 07224 07236 07246 [illegible] 08026
08092 08249* 08250* 08251* 08287 08319 08331 08348
08901 08916 09141 09146 09219 09345 09358 09366
09380 09388
ANOMALIES,LIVER
00689 01693 02568 03466 03470 03488* 04471 05492
06551 06938 07393 07413 07902 07942 08434* 08494
08526 09544 09548 09563 09603
ANOMALIES,MESENTERY
08120*
ANOMALIES,PANCREAS
00572 00994 02001 02114* 03318 03319 04391* 05009
05154 06232 06239* 06415 06661 07057 07907 08388
08389 08394 09451
ANOMALIES,RECTUM
00511 00523 01900 06362 08331 09985
ANOMALIES,SMALL INTESTINE
00360 01215 01267 01271 01295 01946 01991 02114*
02156 02186 03301* 03319 04228* 04232* 05009 05187*
05210 06221 06232 07084* 07109 07110 07609 08026
08092 08124 08139 08901 08916 09219 09224 09233
09238 09245 09862
ANOMALIES,STOMACH
04200 04205 04207 04211 04212 04217 05099 05138*
05939 06187 07012* 07057 07058 07059 08031 08037
08092 09159
ANORECTAL SURGERY
01382* 01390* 01407* 01408* 01462 08310 08345
ANOREXIA NERVOSA
00212 02280* 05073 05501 06003* 07572
ANTACIDS
00104* 00396* 00875 00878* 01318* 01904 02205* 02206*
02896* 02897* 03190* 03208 03209 03982 04028* 04141
04275 04276 04286 04288 04922 05218* 05268 06095
06113 06114 06211 06237* 06271 06284 07064 07131*
07157 08178* 09250 09251 09278 09319 09816
ANTIBIOTICS
00176 00222 00452* 00681 00938* 00939* 00971 01113
01114 01393* 01733* 01971 02007* 02193 02317* 02331*
02473* 02485* 02667* 02683 02705* 02883* 02950 02974*
02984* 02985* 03048 03076* 03234* 03331* 03653 03725*
03728* 03882* 03928 03951 03990 04035* 04061 04111
04140 04309* 04319 04326 04336* 04339 04345 04382*
04411* 04422 04435* 04554 04584 04586 04587 04627*
04638 04714* 04824 04887 04889 04891 04913 04987*
04990* 05002 05007 05135* 05181* 05184* 05265* 05292
05320* 05334 05369 05500 05589 05598* 05625 05648*
05814* 05863* 05996* 05998* 05999* 06057 06067 06076
06088 06105 06302 06325 06329* 06340 06649 06721*
06780* 06830 06832 06953 06954 07100 07118 07196
07212* 07216* 07255 07303* 07347* 07400 07497* 07527
07529 07535 07560 07563 07639* 07759 07799 07820
08067 08099 08117* 08128 08222 08235 08266* 08311
08358 08369 08429* 08555* 08659* 08664* 08673 08721*
08734* 08739* 08745* 08765 08775 08778 08786 08787
08788 08789 08800 08801 08802 08803 08809 08838
08839 08861 08894 08898 08905 09094 09198 09204
09278 09344 09472 09500 09533 09636 09644 09774
09785 09807 09817 09818 09852 09854 09871 09872
09885 09951
ANTIBIOTICS IN LIVER INJURIES
00683* 01113 01606 03363* 03492 05538* 05564 06537*
08568
ANTIBIOTICS IN MALABSORPTION TREATMENT
03226* 03246* 08248
ANTIBODY,GASTRIC PARIETAL-CELL
00308* 01180* 02943 03109* 03111* 03113* 06176
ANTICHOLINERGIC AGENTS
00390* 00391* 00392* 00412 00832* 00871 00872 00882*
00887* 00953* 01024* 01309* 01313* 01703* 01749* 01759*
02006* 02204* 02795* 02827* 02896* 03190* 03208 03750*
04380* 04422 04874 05030 05239 05369 05744* 05798
06095 06101 06210 06235* 06252* 06259* 06267 06271
06284 06292 07064 07161 07656* 07678* 07684 08267*
ANTICHOLINERGIC AGENTS
CONTINUED
07075 09842
ANTIEMETICS
SEE VOMITING
ANTIMETABOLITES
00702 00909 01643 02506 03845 03849 03871 03904*
04383* 04444* 04527* 04541 04545 04547 04592 05538*
05557 05596* 05813* 06314* 06382* 06784* 07323 07408
07421* 07433 07479 07740* 07820 07870 08542* 08543*
08544* 08576 08722* 08866 09646
ANTISECRETORY AGENTS
[illegible] [illegible] 05761* 06149* 06213 06235* 06255* 06258*
06752* 06754* 06761 06762 06763 06764 06765 06766
07166 07171 07172 07173 07174 07175 07176 07656*
07675* 09251
ANTISPASMODICS
SEE ALSO MOTILITY
00882* 01300* 01318* 02011* 02894* 05175 05243 05644*
06103 06107 06620* 07514 07541* 08222 08402 09839
ANTRECTOMY
[illegible] 01385* 01722* 02780* 02899* 03134 03185* 03186*
03188* 03189* 03192* 03197 03211 03760* 04177* 04261*
04262* 04264* 04292 04296 04297 04748* 05221* 05236
05247 05758* 06259* 06270 06285 06310 07010* 07129*
07166 07177 07885 08203
ANTRUM,PYLORIC
00243 00309* 00572 00786* 00856 00859 00886* 01034*
01035* 01170* 01195 01215 01298* 01303* 01308* 01341
01349 01351 01789 01867* 01915 02109 02110 02208*
02250 02283* 02780* 02785* 02789* 02815 02819 02833
03110* 03218 03695* 03708* 03757* 03779 04169* 04171*
04181* 04186 04207 04699 04748* 04749* 04751* 04773*
04958 05138* 05215* 05695* 05740* 05766 05797 06143*
06202 06244* 06245* 06749* 06756 06849* 06850* 07010*
07658* 07849* 07885 08165* 08168* 09178
ANUS
00506 00515 01053 01390* 08339
ANUS ANOMALIES
00515 00523 00529 01854* 05059 06326* 07245 08340
09358
ANUS BIOPSY
08257* 09387 09390
ANUS CANCER
01407* 01461 04357 05328 05329 07232 07258 07260
08257* 08260* 08345 09390
ANUS CIRCULATION
08345 08920
ANUS DIAGNOSIS
00517 01854* 03906* 04931 07225 08257* 08290 09108
09387 09390
ANUS DISEASES
00517 03906* 04357 05355 06323* 07243 08290 08344
08349 09342
ANUS FISSURES
00515 02364 05059 05325 05326 05330 07225 09362
09373
ANUS FISTULAS
00476 01377* 01382* 04357 05316* 05325 05326 06326*
06328* 08293 09390
ANUS HISTOLOGY
07605
ANUS IN CHILDREN
07225 07245 08290 08291 09108
ANUS MORPHOLOGY
03285 05688*
ANUS MOTILITY
01381* 01799* 01804* 02333 03799 04767* 04788 04789
05316*
ANUS MUCOSA
07605
ANUS NEOPLASMS
02345 09387
ANUS NEOPLASMS,MALIGNANT
00476 01377* 01421 02304* 02342 08297 08345
ANUS OBSTRUCTION
04351 07245
ANUS PATHOLOGY
02364 07251 08343 08344 08345 08349 09387
ANUS RADIOLOGY
SEE ALSO ANUS DIAGNOSIS
03799 04788
ANUS SPHINCTER
01377* 01381* 01386* 01799* 01804* 01854* 02333 03799
04767* 04768* 04788 04789 05312* 05316* 05325 05326
05330 06328* 06373 06689 06882* 07243 07863* 08310
08340 08341 09108 09122 09363 09373 09387
ANUS SURGERY
01386* 01419 02313* 02364 04351 05312* 05316* 05325
05326 05330 06328* 07242 07243 07245 07260 08257*
08291 08292 08293 08294 08345 08351 09358 09361
09362 09372 09373 09418
ANUS TREATMENT
08292 08294

US ULTRASTRUCTURE
07005
US,NERVOUS CONTROL OF
01799* 01824* 04767* 04768*
RTIC ANEURYSMS
00522 01058 01107 01984 03043 09098
RTOGRAPHY
00476
PPENDICITIS
00234 00453* 00522 00928* 01026* 01056 01103 01394*
01397* 01433 01446 01922* 01969 02180 03026 03155
03156 03267 03291 03923* 03937 04347 04365 04896
05300 05641 05326* 05341 05347 05995* 06026 06103
06329* 06340 06374 06575 07217* 07220 07239 07561
07961 08266* 08276 08277 08304 08329 08725* 08770
08784 08785 08829 09348 09380 09395 09406 09411
09478
PPENDICITIS COMPLICATIONS
09351
PPENDICITIS DIAGNOSIS
03944 09414
PPENDICITIS EPIDEMIOLOGY
00453*
PPENDICITIS ETIOLOGY
09351
PPENDICITIS IN CHILDREN
03970 04113 07253 07566 08280 08307 08312 08334
08358 08806 09414 09901
PPENDICITIS TREATMENT
09411 09414
PPENDICITIS,AGE FACTORS IN
00453* 01026* 01397* 04071 04347 07255 08832 09351
PPENDIX
00460 00511 00803 [illegible] 01432 01455 01459 01671
02153 02159 02761* 02977* 03171 05323 05908 06068
06078 06331* 07249 07255 08150 08252* 08280 08281
08313 08314 08329 08331 08334 08339 09380 09388
09406 09410
PPENDIX CANCER
01446 02183 09852
PPENDIX DIAGNOSIS
09348
PPENDIX NEOPLASMS
08285
PPENDIX SURGERY
09347
PPETITE
01960 07572
RGYROPHIL CELLS
02057* 02153 02154 02744 05685* 06678* 06697 06737
07616 08780 08929 09462
RTERIOSCLEROSIS
08840 08853 09788
SCITES
SEE ALSO CIRRHOSIS
00204 00524 00607* 00653 01376 01642* 02517 02561
02629* 02889* 02994* 03584* 03586* 03600* 03613 03615
03617 03618 04034* 04433* 04600* 04612 04912 04939
04984* 05012 05015 05036 05061 05064 05066 05068
05069 05511 05611* 05617 05620 05624 05627 05854*
06418* 06454* 06457* 06679* 07293 07313 07352 07388
07429 07451* 07558 08402 08417 08459 08463 08530
08633* 08833 09235 09236 09488 09521 09538 09548
09661 09878 09929
SCITES IN CIRRHOSIS
00740 04137 04598* 04603* 04606* 04609 04865 05607*
05609* 05612* 05627 06579* 06580* 06585 06611 06613
07344* 07465* 07471* 07472 07474 07475 07478 08428*
08500 08634* 08645 08652 09701 09712 09714 09718
09722 09723 09726 09736 09737 09738
SCITES TREATMENT
07088
THEROSCLEROSIS
02564 06204 06813 08133 08854 08859 09330 09735
TRESIA,BILIARY TRACT
07841* 09617
TRESIA,ESOPHAGUS
02027 02907* 03081 03084 03087 03095 04164 07991*
07995* 09137 09152 09158
TRESIA,LARGE INTESTINE
06335 08287
TRESIA,SMALL INTESTINE
07084* 08835
TROPHIC GASTRITIS
00294* 00298* 00312* 00339 00813* 00834* 00844* 00845*
01027* 01048* 01115 01166* 01177* 01180* 01209 01924*
02980* 03106* 03112* 03114* 03137 03890* 05129* 05133*
05141* 05147* 05232* 06087 06177 06740* 07028* 07030*
07031* 07041 07068 07839* 08073 08076 09098 09171
09176 09180 09181 09210
TROPINE
05784* 05785* 06670* 06702* 06722* 06754*
UTONOMIC NERVOUS SYSTEM
06303 06454* 06506 06733 07005 07039 07049 07224
07305* 07348* 07392 07458 07602 07612 07760 08216
09093 09844

BACTERIA
SEE MICROORGANISMS
BACTERIAL TOXINS
05532*
BALANCE STUDIES,METABOLIC
05846*
BANTIS DISEASE
00734 02643 04504 08648 09081 09711
BENIGN ABDOMEN NEOPLASMS
07559
BENIGN BILIARY TRACT NEOPLASMS
01690 07495* 07977
BENIGN DUODENUM NEOPLASMS
08146
BENIGN ESOPHAGUS NEOPLASMS
00281 00985 01147 01153 01162 02028 02039 04145
04148 04154 04969 05117 06117 06134 06138 06981
07989* 08008 08012 09134
BENIGN GALLBLADDER NEOPLASMS
02671 03646 07495* 08680 08681 08703 08709
BENIGN GASTROINTESTINAL NEOPLASMS
00224 00948* 01077 04042* 04068 06015 08748 08817
08824 08912 09823
BENIGN LARGE INTESTINE NEOPLASMS
00441* 01083 01437 02153 02305* 02343 02361 03254*
04334* 04340 04352 04354 04356 04361 04366 05060
05319* 06330* 06334 06337 06357 07223 07229 07230
07231 07234 07237 07242 08259* 08281 08308 08323
08336 08347 09395
BENIGN LIVER NEOPLASMS
01580 02502 02506 03371* 03939 04476 06464* 06894
07378 08469 08493 08501 08512 08520 09516 09520
09551 09566 09619
BENIGN MESENTERY NEOPLASMS
04030* 06066 08154 09888
BENIGN PANCREAS NEOPLASMS
00564 01507 01508 02415 04386* 05014 08373* 08748
09233
BENIGN RECTUM NEOPLASMS
00443* 00470 04352 06330* 08273 08288 08336 08352
09199
BENIGN SALIVARY GLAND NEOPLASMS
02910* 09831 09931
BENIGN SMALL INTESTINE NEOPLASMS
00165 00346* 00361 00370 00418 01051 01083 01262
01263 01270 01272 01282 01370 02118* 02123* 02139
02154 03149* 03165 04250 04251 05060 05194 05209
05212 06233 06929 07086* 08101 08122 08130 08131
08151 08161 08743* 09823 09905
BENIGN STOMACH NEOPLASMS
00282* 00292* 00325 00332 00333 00341 01174* 01182*
01233 01234 02047* 02059 02104 02234 03126 03130
03134 03135 04191 04193 04199 05151 06165 06169
06194 07022* 08046 08053 08057 08084 08101 08104
08106 09178 09193 09199 09203 09208 09216 09823
09905
BEZOARS
01168* 01171* 01361 01454 03101* 03115* 03124 03138
04175* 05235 05940 07043 08107
BICARBONATE
02437 02817
BICARBONATE ABSORPTION
01412 02753* 04703
BICARBONATE SECRETION,PANCREAS
00930* 01483* 01492* 01751* 01752* 01753* 01759* 01764*
01780 02409* 02797* 02803* 02804 03752* 03753* 04380*
04389* 04750* 04875 05203 05747* 06423* 06772* 06880*
07690* 07692* 07853* 08375*
BILE
00073* 00305* 02105 02165 02170 02467 02667* 02987*
03552 04625* 04630* 05639* 07115 07492* 07634* 07635*
07824 08999 09005 09024 09299
BILE ACIDS AND SALTS
SEE ALSO ABSORPTION
00067 00086* 00098 00107* 00201 00202 00305* 00755*
00796 00851* 00896* 00917 01648* 01672 01795 01803*
01960 02603* 02661* 02664* 02707 02756* 02767 02857
02868 02893* 02923 03227* 03246* 03334* 03369* 03411
03674* 03721* 03790 03801 03809* 03964 04036* 04309*
04445* 04561* 04635* 04729* 04800* 04853* 04918 05208
05494 05632* 05717* 05718* 05779* 05804* 05805* 05822*
05889 06007* 06548 06620* 06623* 06783* 06785* 06799*
07193* 07337* 07502* 07628* 07654 07701* 07791 07792
07794 07810* 07811* 08230* 08306 08426* 08586 08657*
09012 09018
BILE ANALYSIS
01826* 03411 03964 04016 04630* 04635* 04800* 05814*
07754 08657* 08991
BILE COMPOSITION
00756* 01637* 01647* 01648* 01688 01825* 01826* 01828*
02661* 02704* 02840* 02874 03743 03881 03964 04635*
04648 04795* 04800* 05805* 05931 06483 06529 06623*
06638 06770* 06789* 06792* 06799* 06803 07398 07745*
07754 07802 07807 08657* 08664* 08668 08687 08977
08991 09560 09760

BILE DUCT
SEE ALSO BILIARY TRACT
01832* 02717 09763
BILE DUCT,COMMON
SEE ALSO BILIARY TRACT
00015* 00051* 00368 00573 00757* 00758* 00760 00776
00777 00839* 00924* 00962 01539* 01587 01647* 01648*
01651* 01654* 01656* 01657* 01658* 01660* 01670 01682
01687 01690 01691 01692 01792 01852* 01975 02438
02583 02659* 02668* 02696 02704* 03628* 03629* 03630*
03637 03651 03654 03655 03659 04007 04017 04018
04033* 04102 04237 04409* 04410* 04413* 04472 04626*
04631* 04639 04640 04644 04658 04663 04664 04778*
04813 05423* 05436* 05633* 05634* 05636* 05638* 05642*
05646* 05647* 05650* 05651 05659 05662 05862* 06432
06437 06477 06618* 06624* 06629* 06633 06644 06645
06647 06652 06654 06656 06660 06661 06662 06663
06665 06668* 06674* 06720* 06721* 06734* 06879* 06914
07294 07339* 07346* 07356 07357 07358 07366 07385
07426 07449* 07491* 07498* 07500* 07507 07513 07517
07518 07519 07528 07531 07532 07534 07536 07538
07539 07660* 07878 07879 07976 07979 08434* 08467
08515 08656* 08658* 08662* 08665* 08676 08679 08682
08685 08689 08693 08695 08699 08702 08708 08710
08714 08885 09100 09474 09559 09741 09745 09747
09749 09752 09758 09763 09767 09769 09772 09773
09775 09776 09777 09779 09780 09786
BILE EXCRETION
01832* 02122* 02180 02650 03721* 04805* 04873 05796*
06649 06720* 06721* 06799* 07334* 07335* 07533 07628*
07711* 07712* 07715* 07717* 07741* 07774 07802 08306
08443* 08636*
BILE METABOLISM
00098 00168 00790* 00878* 00896* 00964 01795 01826*
02840* 02857 03060 03820* 03881 04652 05479 05805*
06783* 06785* 07376 07791 07792 07793 07794
BILE METABOLITES
00679 01832* 03929 08698
BILE MICROORGANISMS
01648* 01649* 01649* 01662* 02660* 02664* 02667* 03626*
03637 04033* 04111 05639* 05814* 06649 06955 07399
07449* 08609 08656* 09765
BILE PERITONITIS
01044* 03645 03883* 04033* 04662 04984* 04992 06621*
06658 06665 06964 08671 09785
BILE PIGMENTS
SEE ALSO BILIRUBIN
SEE ALSO CHOLESTASIS
00168 00596* 00681 00964 01677 01760* 02662* 03380*
03381* 03743 03820* 03834 04630* 04652 05479 05641*
06466* 07445* 07536 07615 07778 07809 07821 08462
08507 08511 09671 09715
BILE PIGMENTS IN JAUNDICE,SERUM
00596* 00756* 01618* 01677 03380* 03381* 03827 04501
04873 05494 07402 07809 08511
BILE SALT ABSORPTION
02122* 02129* 03150* 03227* 03633* 05717*
BILE SALTS
SEE ALSO BILE ACIDS AND SALTS
00086* 00305* 02865
BILE SECRETION
00023* 00076* 00101 00756* 00797 00839* 00851* 00867
00875 00913 01353 01637* 01688 01760* 01841 01851
02704* 02782* 02815 02858 02874 03360* 03369* 03491
03522* 03639 03641 03642 03757* 03765* 03774 03784
03811* 03881 04006 04007 04539 04670* 04795* 04805*
05072 05648* 05652 05658 05765* 05767 05793* 05804*
05814* 05938 05956 06449* 06483 06508 06529 06620*
06623* 06626* 06634 06734* 06770* 06782* 06789* 06803
06914 06932 07193* 07276* 07356 07367 07398 07514
07523 07745* 07747* 07747* 07762 07969 08387 08426*
08430* 08431* 08441* 08688 08819 08988 09511 09540
09766
BILE SECRETION STUDY TECHNIQUES
04006 04007 07660*
BILE SECRETION,DRUG EFFECTS ON
07823
BILE,DIGESTIVE ACTION OF
00101 02707 03790 07193* 07644*
BILIARY DISEASE DIAGNOSIS
00108* 00111* 00123* 00131* 00138 00169 00172 00598*
00751* 00752* 00753* 00777 00778 00924* 00954* 00961
00962 00977 00981 00996 01503 01566 01649* 01651*
01652* 01654* 01655* 01657* 01659* 01660* 01662* 01664*
01682 01689 01866* 01881 01891 01901 01903 02659*
02665* 02677 02919* 02923 02925 02938 02945 02960
02962 02964 02976* 03332* 03597* 03626* 03629* 03631*
03651 03656 03661 03662 03666 03902* 03926 03950
03979 04001 04007 04016 04017 04018 04019 04410*
04488 04665 04689* 04842* 04843* 04856* 04864* 04871
04879 04886 04890 04918 04965 04966 05636* 05647*
05650* 05661 05662 05664 05850* 05859* 05860* 05862*
05870* 05872* 05880* 05896 05901 05909 05914 05936
05941 05952 05955 05979 06437 06643 06652 06668*
06674* 06789* 06857* 06872* 06878* 06892 06928 06931
06933 06935 06937 06947 06957 06961 06962 07265
BILIARY DISEASE DIAGNOSIS
CONTINUED
07490* 07495* 07503 07515 07522 07525 07538 07754
07837* 07840 07841* 07842* 07878 07879 07884 07886
07898 07904 07914 07931 07932 07938 07943 07957
07964 07973 07974 07976 07977 07978 07979 0798
08171* 08507 08519 08662* 08682 08694 08699 08714
09049 09055 09062 09067 09068 09071 09072 0909
09124 09478 09500 09529 09533 09659 09741 09759
09767 09771 09775 09776 09781 09783 09794 09796
09800 09804
BILIARY DISEASE EPIDEMIOLOGY
00765 03047 06512 09785
BILIARY DISEASE ETIOLOGY
07526 07540 08659* 08665* 08669 09542 09748 09750
BILIARY DISEASE IN CHILDREN
07466*
BILIARY DISEASE TREATMENT
02691 02895* 02919* 03177 03629* 04668 05648* 06620*
06634 06635 07361 07382 07494* 07501* 07515 07522
07523 07529 07530 07533 07535 07538 07539 08058
08426* 08467 08470 08659* 08661* 08670 08673 08689
08699 08700 08702 08708 08778 08803 08820 09495
09498 09776 09783 09785 09934
BILIARY DISEASES
00108* 00123* 00758* 00760 00762 01055 01566 01659*
01662* 01664* 01678 01686 01689 01944 02522 02674
02683 02687 02688 03060 03060 03458 03626* 0366
03666 04080 04237 04431 04488 04984* 05011 05570*
05641* 05789* 05859* 06502 06507 06655 07304* 07349*
07402 07414 07440 07494* 07524 07527 07537 07791
07964 07976 07977 07977 08054 08121 08429* 08464
08465 08522 08530 08609 08645 08656* 08690 087
08710 08712 08713 08715 08777 09478 09524 09623
09715 09756 09760 09769 09770 09784 09789 09793
09794 09795 09797 09799 09800 09802 09828 09842
BILIARY ENZYMES
04843* 05682* 07596* 07754 09797
BILIARY PANCREATITIS
08405
BILIARY TRACT
SEE ALSO CHOLECYSTITIS
SEE ALSO CHOLELITHIASIS
SEE ALSO GALLBLADDER
01532* 01587 02105 02438 02677 02684 04627* 04640
05436* 06462* 06674* 07964 07977 09529 09763
BILIARY TRACT ANGIOGRAPHY
06892
BILIARY TRACT ANOMALIES
00759* 00765 01687 01689 01921 03007 03629* 03656
03659 03902* 04448* 04459* 04610 04624* 04644 04660
05595* 06462* 06464* 06624* 06632 06674* 07334* 07335*
07349* 07518 07519 07524 07538 07599* 07841* 07878
07928 08426* 08430* 08431* 08434* 08667* 08683 08689
08919 09603 09742 09767 09779 09789
BILIARY TRACT ATRESIA
07841* 09617
BILIARY TRACT BIOCHEMISTRY
00916 02861 03411 04675* 04802* 05524 07596* 07719*
07741* 07745* 07774 09660
BILIARY TRACT BIOPSY
04689* 07977
BILIARY TRACT CANCER
01020* 01134 01686 02557 02692 03401 04082 04102
04664 04886 04890 04999 05393 05504 05651 06892
07516 07520 07521 07931 08470 08505 08553* 08678
08706 09542 09577 09707 09743 09749 09753 09758
09770 09796 09798 09801
BILIARY TRACT CIRCULATION
00759* 00916 02676 06892 07532 08999
BILIARY TRACT CIRRHOSIS
00773 01493* 01553 01636* 01637* 01693 02472* 02627*
02628* 02648 02673 02976* 03464 03597* 03631* 04111
04471 04482 04483 04511 04512 04588 04610 04615
04631* 04643 04841* 05473 05602* 05615 05621 05626
06461* 06590 06643 07334* 07337* 07387 07405 07488
08387 08430* 08431* 08462 08463 08640 08646 08698
09512 09591 09730
BILIARY TRACT CYSTS
09777
BILIARY TRACT DEVELOPMENT
06505
BILIARY TRACT DIVERTICULA
03655 07495*
BILIARY TRACT ENDOSCOPY
05860* 05862* 07964
BILIARY TRACT EXCRETION
00117*
BILIARY TRACT FISTULAS
00776 01342 01652* 01657* 01670 01680 01921 01949
02182 02478* 02682 03627* 04006 04007 04661 04871
06618* 06619* 07339* 07507 07508 07511 07513 07537
07791 07793 07800 08202 08530 08669 08700 09249
09659 09744 09771 09774 09785 09786

LIARY TRACT HISTOLOGY
00630* 07453* 07745*
LIARY TRACT ILEUS
00764 02182 04666 04667 08152 08156 09759
LIARY TRACT IMMUNOLOGY
07802
LIARY TRACT IN CHILDREN
00652 01659* 03370* 03629* 03664 03902* 05595* 05644*
06632 07334* 07335* 07524 08507 09524 09617 09793
09795 09800
LIARY TRACT METABOLISM
04675* 04802* 07717* 07719* 07774
LIARY TRACT MORPHOLOGY
03630* 03665 04660 05703 06505 06516 06624* 06661
06663 06668* 06674* 06782* 06878* 07518 07519 07599*
07722* 08434* 08662* 08667* 08676 08919 09489
LIARY TRACT MOTILITY
00076* 01104 01353 01812* 02894* 03412 03639 04085
04086 04668 05648* 05782* 05798 05936 06634 06720*
06721* 06878* 07498* 07514 07660* 08051 08443* 08662*
09062
LIARY TRACT MUCOSA
05703 07596* 07673 07711* 07712* 07715* 07717* 07719*
LIARY TRACT NEOPLASMS
00108* 00777 01664* 02674 02696 04632* 04660 07265
07337* 07878 07978 08506 08702 08704 08708 09743
09747 09752
LIARY TRACT NEOPLASMS,BENIGN
01690 07495* 07977
LIARY TRACT NEOPLASMS,MALIGNANT
00780 00784 01636* 01654* 01658* 01682 01685 01687
01852* 01942 02665* 02692 04657 06030 08675 09527
09542 09772 09796 09801
LIARY TRACT OBSTRUCTION
00051* 00768 00770 00774 00775 01254* 01575 01656*
01657* 01684 01685 01691 01692 01901 02450 02664*
02676 02704* 03597* 03624* 03626* 03629* 03631* 03642
03811* 04043* 04067 04233* 04495 04650 04667 04778*
04813 04837* 04885 05064 05459 05502 05504 05546*
05636* 05638* 05646* 05649* 05650* 05664 05665 05862*
05955 06465* 06466* 06632 06633 06644 06645 07291
07337* 07356 07357 07358 07363 07366 07490* 07504
07524 07526 07536 07539 07879 07914 07938 07947
08152 08156 08387 08423* 08462 08463 08464 08507
08522 08640 08665* 08698 08708 08710 09100 09124
09490 09498 09542 09556 09594 09603 09617 09730
09745 09748 09750 09754 09755 09763 09766 09767
09768 09789 09799
LIARY TRACT PATHOLOGY
01587 03629* 03630* 03662 03665 04102 04632* 05641*
05664 06617* 06630* 07334* 07335* 07526 07528 08429*
08713 09071 09072 09524 09529 09748 09766 09783
LIARY TRACT PERFORATION
00752* 01345 03645 03654 04662 04992 06653 07507
09785
LIARY TRACT PHYSIOLOGY
03662
LIARY TRACT RADIOLOGY
EE ALSO BILIARY DISEASE DIAGNOSIS
00131* 00598* 00751* 00924* 00977 00980 01101 01249*
01503 01652* 01655* 01660* 01664* 01670 01671 01680
01681 01866* 01921 02182 02693 02919* 02938 02962
03634 03635 03656 03661 03926 03950 03979 04017
04019 04643 04657 04839* 04842* 04856* 04861* 04864*
04871 04884 04886 04890 04965 04966 05633* 05636*
05647* 05658 05659 05661 05662 05872* 05880* 05901
05909 05914 05917 05941 05955 06625* 06647 06652
06658 06674* 06853* 06857* 06878* 06879* 06892 06935
06937 06962 07402 07495* 07525 07660* 07841* 07878
07879 07884 07886 07898 07932 07938 07957 07974
07980 08171* 08434* 08662* 08667* 08682 09055 09062
09067 09068 09071 09072 09090 09233 09478 09730
09741 09767 09776
LIARY TRACT STRICTURE
01657* 01691 03631* 04488 05642* 05664 07496* 07500*
08058 08666* 08691 08692 08695 08698 08701 08715
09750 09780 09784
LIARY TRACT SURGERY
00161 00351* 00563 00752* 00756* 00757* 00758* 00759
00760 00771 00775 00776 00777 00780 00962 01022*
01038* 01342 01509* 01510* 01548* 01566 01587 01652*
01654* 01657* 01658* 01659* 01680 01682 01684 01692
01901 01942 01948 01975 02412* 02442* 02664* 02665*
02672 02689 02690 02691 02717 02919* 02976* 03177
03411 03475 03626* 03629* 03631* 03634 03635 03654
04033* 04071 04210 04410* 04413* 04419 04488 04627*
04631* 04639 04646 04657 04658 04663 04664 04665
04975* 05035 05459 05502 05566 05595* 05633* 05636*
05638* 05640* 05642* 05646* 05647* 05650* 05651 05658
05659 05661 05662 05663 05664 05665 05859* 05909
06477 06483 06507 06516 06618* 06624* 06628* 06629*
06630* 06633 06635 06642 06644 06645 06647 06654
06655 06656 06659 06661 06662 06665 06668* 06878*
06914 06964 07302* 07339* 07346* 07366 07389 07390
07414 07491* 07492* 07493* 07496* 07497* 07498* 07500*
07501* 07508 07513 07518 07520 07521 07522 07527

BILIARY TRACT SURGERY
CONTINUED
07528 07531 07532 07534 07537 07538 07539 07540
07552 07582 07762 07878 07879 07938 07964 08152
08156 08405 08434* 08465 08467 08469 08470 08519
08522 08567 08640 08656* 08658* 08660* 08661* 08663*
08666* 08669 08670 08671 08676 08678 08679 08681
08682 08685 08686 08689 08691 08692 08693 08695
08696 08698 08699 08700 08701 08702 08705 08707
08708 08710 08712 08713 08714 08793 09034 09062
09072 09220 09221 09474 09478 09484 09558 09594
09603 09741 09742 09743 09744 09745 09747 09749
09751 09752 09753 09754 09755 09763 09767 09768
09769 09773 09774 09776 09777 09780 09781 09782
09783 09785 09786 09798 09802 09807 09868
BILIARY TRACT TRANSPLANTATION
01792 07500*
BILIARY TRACT TRAUMA
00652 00961 01944 03654 04643 04644 04645 04662
05490 05642* 06629* 06665 07389 08567 08692 09630
09638 09856
BILIARY TRACT ULTRASTRUCTURE
05703 09789
BILIARY TRACT,AGE EFFECTS ON
04624* 04689* 05651 08660* 08663*
BILIARY TRACT,DISEASES ASSOCIATED WITH
00771 02588 02676 08171* 08666*
BILIARY TRACT,DRUG EFFECTS ON
05798 06626* 07530 07661* 07823
BILIARY TRACT,DRUG TREATMENT OF
02919* 04668 05644* 05814* 06626* 06634 07514 07515
07523 07540 07752* 08432* 08673 09765 09934
BILIARY TRACT,FOREIGN BODIES IN
05661
BILIARY TRACT,HORMONAL CONTROL OF
05938 07495*
BILIARY TRACT,NERVOUS CONTROL OF
00086* 00772 00839* 00867 01851 02894* 04626* 05793*
06649 07533
BILIARY TRACT,TOXIC EFFECTS ON
04675* 07514 07664
BILIRUBIN
00102 00145 00166 00596* 00636 00660 00681 00756*
00757* 00790* 00796 00878* 00964 01539* 01543* 01568
01577 01616* 01648* 01677 01832* 02479* 02503 02606
02662* 02666* 02704* 02782* 02837* 02858 02866 02916*
02992* 03365* 03370* 03377* 03380* 03381* 03390 03484*
03532 03575 03594* 03615 03743 03807* 03810* 03811*
03820* 03827 03831 03834 03835 03929 04009 04033*
04439* 04445* 04507 04550 04630* 04652 04678 04902
04927 04964 05479 05546* 05599* 05637* 05814* 05836*
05889 05947 06448* 06484 06510 06537* 06609 06621*
06638 06781* 06865* 06914 07058 07369 07371 07402
07536 07747* 07752* 07800 07809 07837* 07883 07979
08441* 08453 08508 08511 08527 08595* 08601* 08627
09003 09006 09009 09490 09515 09574 09608 09664
09671 09675 09680 09681 09689 09700 09766
BILIRUBIN ABSORPTION
01618* 01824* 03827 05524 05682* 05822* 07778
BILIRUBIN METABOLISM
07742* 07743*
BIOCHEMICAL DIAGNOSIS OF CANCER
01179* 01585 02537 05143* 06621* 07739* 09033
BIOCHEMICAL DIAGNOSIS OF CIRRHOSIS
00107* 00599* 00739 00931* 03594* 03606 03609 05881*
09716
BIOCHEMICAL DIAGNOSIS OF GASTROINTESTINAL DISEASES
00210 00944* 00989 02238 04859* 04980*
BIOCHEMICAL DIAGNOSIS OF HEPATITIS
00107* 00145 00927* 00931* 01894 02601* 03574 03575
03578 04872 05571* 05579 06852* 06854* 08537 08618
09667
BIOCHEMICAL DIAGNOSIS OF JAUNDICE
00145 01009 02916* 03894* 05515 05870* 05881* 05947
06861* 07337* 07837* 08462
BIOCHEMICAL DIAGNOSIS OF LARGE INTESTINE DISEASES
00149
BIOCHEMICAL DIAGNOSIS OF LIVER DISEASES
00159 00596* 00599* 00602* 00616 00617 00619 00623
00626 00660 00690 00937* 01543* 01585 01639* 01896
02474* 02534 02535 02553 02884* 03373* 03467 03500
03954 04020 04806* 05438* 05439* 05866* 05970 05987*
06792* 07899 08523 08537 08611 09047 09086 09089
09508 09576
BIOCHEMICAL DIAGNOSIS OF MALABSORPTION
00062* 03239* 04314 05867* 07194*
BIOCHEMICAL DIAGNOSIS OF PANCREAS DISEASES
00107* 00119* 00544 00545 00547 00592 00836* 00860
00930* 01483* 01485* 01489* 01491* 01514* 01519 02416
03325 03909* 07838* 07970
BIOCHEMICAL DIAGNOSIS OF PARASITIC DISEASES
00180* 04072 04084 08546* 08718*
BIOCHEMICAL DIAGNOSIS OF PEPTIC ULCERS
00144 01129 03918* 08206 09077 09078

BIOCHEMICAL DIAGNOSIS OF SALMONELLOSIS
06870*
BIOCHEMICAL DIAGNOSIS OF SCHISTOSOMIASIS
05612*
BIOCHEMICAL DIAGNOSIS OF SMALL INTESTINE DISEASES
04307*
BIOCHEMICAL DIAGNOSIS OF STOMACH DISEASES
01129 02905* 02980*
BIOCHEMICAL DIAGNOSIS OF ULCERATIVF COLITIS
06380*
BIOCHEMISTRY,BILIARY TRACT
00916 02861 03411 04675* 04802* 05524 07596* 07719*
07741* 07745* 07774 09660
BIOCHEMISTRY,GALLBLADDER
01824* 02613 02695 04776* 05682* 07489* 07489*
BIOCHEMISTRY,GASTROINTESTINAL
00003* 00005* 00018 00210 00944* 01104 01713 01793*
02003 02161 02273 02655 02761* 02764 02812 02965
03244* 03683 03703* 04685* 04691* 05697* 05778 05865*
06037 06038 06095 06735* 06737 07631* 07673 07818
07821 07834 09019 09023
BIOCHEMISTRY,LARGE INTESTINE
01405* 01406* 02745 02812 03254* 03669* 03677 03778
03780 03781 04023* 04672* 04685* 04707 05362* 05380
06675* 06678* 07205* 07205* 07636* 07637* 07638* 07667*
07820 08258* 08263*
BIOCHEMISTRY,LIVER
00037 00082* 00083* 00084* 00085* 00088* 00091* 00094
00095 00097 00099 00100 00102 00145 00597* 00601*
00613 00614 00615 00617 00647 00657 00658 00676
00682* 00683* 00684* 00685* 00715 00730* 00732* 00742
00891* 00892* 00893* 00894* 00898 00899 00900 00901
00904 00907 00909 00910 00912 00913 00914 00916
00918 01008 01235 01528* 01541* 01542* 01547* 01563
01567 01581 01589 01611* 01632 01637* 01646* 01823*
01827* 01829* 01830* 01833* 01835 01836 01837 01838
01839 01840 01842 01843 01847 01848 01970 02475*
02477* 02490 02515 02523 02536 02537 02566 02576*
02577* 02630* 02737 02837* 02838* 02839* 02840* 02841*
02843 02846 02847 02848 02849 02850 02852 02853
02855 02856 02858 02859 02862 02863 02864 02865
02868 02869 02870 02871 02872 02875 02876 02878
02879 02880 02881 02882 02951 03358* 03370* 03380*
03390 03392 03397 03405 03406 03407 03415 03416
03427 03429 03430 03431 03432 03433 03434 03435
03448 03449 03450 03451 03452 03454 03467 03479*
03482* 03483* 03488* 03490 03492 03496 03501 03507
03512 03514 03515 03524* 03537 03550 03575 03575
03578 03592* 03606 03612 03629* 03682 03685 03698*
03806* 03807* 03808* 03812* 03816* 03817* 03818* 03819*
03821* 03822* 03824* 03825* 03826 03828 03829 03830
03831 03833 03834 03835 03837 03838 03839 03840
03843 03844 03845 03846 03848 03851 03852 03853
03854 03855 03856 03857 03858 03861 03862 03863
03864 03865 03866 03868 03869 03872 03875 03876
03879 03880 04064 04320 04439* 04446* 04451* 04469
04474 04491 04506 04509 04515 04518* 04519* 04520*
04521* 04522* 04545 04546 04547 04548 04552 04591
04601* 04604* 04607* 04608* 04620 04622 04623 04670*
04676* 04690* 04792* 04793* 04794* 04796* 04797* 04798*
04801* 04802* 04804* 04807* 04809* 04810 04811 04812
04814 04816 04817 04818 04819 04820 04821 04822
04824 04825 04826 04827 04828 04829 04830 04831
04832 04833 04834 04835 04836 04847* 04853* 04857*
04932 05049 05433* 05438* 05439* 05442* 05460 05481
05484 05505 05512 05524 05535* 05537* 05539* 05542*
05549* 05558 05561 05563 05567 05568 05574* 05592*
05689* 05693* 05694* 05701* 05804* 05807* 05812* 05817*
05818* 05819* 05820* 05821* 05823* 05824 05825 05827
05828 05830 05834 05866* 05934 06228 06460* 06466*
06510 06519 06531* 06539 06540 06685 06697 06769*
06777* 06778* 06779* 06780* 06781* 06783* 06785* 06786*
06787* 06788* 06789* 06791* 06792* 06793* 06794* 06795*
06797* 06797* 06800* 06801 06804 06806 06807 06809
06811 06812 06813 06814 06815 06818 06819 06827*
07350* 07428 07467* 07596* 07619 07697* 07698* 07700*
07701* 07702* 07703* 07704* 07707* 07708* 07709* 07710*
07711* 07712* 07713* 07714* 07715* 07716* 07717* 07718*
07720* 07721* 07725* 07726* 07727* 07728* 07732* 07734*
07735* 07737* 07738* 07739* 07746* 07749* 07753 07757
07758 07760 07761 07763 07766 07768 07769 07770
07772 07773 07775 07776 07777 07782 07784 07786
07787 07788 07789 07791 07792 07793 07794 07796
07797 07798 07801 07804 07806 07807 07829 07864*
07934 07946 07962 07963 07975 08441* 08449 08451
08495 08506 08509 08513 08515 08516 08523 08535
08540* 08543* 08544* 08545* 08546* 08558* 08565 08574
08577 08587 08626 08636* 08643 08652 08657* 08921
08922 08930 08933 08956 08959 08960 08961 08962
08963 08964 08965 08966 08967 08968 08969 08970
08972 08973 08974 08975 08978 08979 08980 08981
08982 08983 08984 08985 08986 08988 08992 08994
08996 08997 08998 09000 09001 09002 09003 09004
09006 09008 09010 09011 09013 09014 09015 09016
09047 09086 09105 09508 09553 09555 09556 09557
09563 09581 09597 09598 09601 09625 09637 09641
BIOCHEMISTRY,LIVER
CONTINUED
09648 09649 09651 09653 09654 09656 09673 09675
09676 09677 09698 09719 09972
BIOCHEMISTRY,PANCREAS
00044* 00067 00223 00556 00590 00804 00805 00836*
00860 00864 00879* 00895* 00967 01021* 01116 01486*
01492* 01494 01500 01509* 01513* 01514* 01516* 01522
01710* 01730* 01771* 01933* 02416 02417 02430 02449
02453 02454 02729* 02787* 02810* 02825* 02828 02846
02970 03325 03338* 03339* 03340* 03351 03354 03677
03683 03685 03686 03689 03690 03691 03692 03699*
03704* 03711* 03739 03766* 03783 03787 03788 03789
03790 03909* 03965 03969 03971 03974 04382* 04412*
04424 04431 04669* 04675* 04687* 04693* 04734 05203
05382* 05708 05737* 05833 05850* 05932 06400* 06424*
06425* 06770* 06774 06827* 06880* 07276* 07696 07740*
07937 07954 07958 08391 08415 08924 08925 08954
09073 09446
BIOCHEMISTRY,SALIVARY GLAND
00055 00793 00794 00801 00802 01110 03683 03684
03783 04119 04385* 05381* 07666* 07672 07683 07936
BIOCHEMISTRY,SMALL INTESTINE
00011* 00024* 00063* 00179 00432* 00892* 00922* 00923*
01255* 01371* 01373* 01374* 01718 01731* 01793* 02157
02246 02391* 02728 02730* 02731* 02734* 02736* 02765
02812 02824 02826* 02827* 02902* 02913* 03002* 03110*
03228* 03233* 03239* 03243* 03245* 03500 03673* 03677
03681 03682 03685 03686 03687 03703* 03703* 03718
03727* 03735 03737 03930 03966 03968 04227* 04229*
04253 04305* 04306* 04313 04676* 04685* 04686* 04688*
04695* 04704 04709 04713* 04714* 04737 04868 05160
05181* 05295 05386* 05666* 05681* 05687* 05714* 05715*
05727 05853* 06260* 06691 06700* 06700* 06708* 06735*
06827* 07083* 07085* 07203 07593* 07594* 07595* 07597*
07620 07621 07626* 07629* 07634* 07667* 07810* 07811*
07816* 07829 07915 07976 08225* 08226* 08233* 08237
08263* 08918 08926 09020 09022 09292 09322 09332
09333 09429 09898
BIOCHEMISTRY,STOMACH
00001* 00013* 00040* 00041* 00047* 00053 00056 00061
00064* 00133 00134 00289* 00298* 00378* 00398* 00789*
00838* 00853 00880 01179* 01181* 01235 01309* 01700*
01701* 01761* 01769* 01781 01794* 02064 02741 02783*
02947 02980* 03110* 03694* 03708* 03715 03732* 03769*
03778 03779 04179* 04215 04685* 04702 04745* 04764
05141* 05143* 05147* 05275 05673* 05683* 05686* 05695*
05696* 05702 05741* 05742* 05743* 05750* 05751* 05754*
05762* 05768 05769 05770 05771 06148* 06155* 06208
06307 06677* 06690 06737 06750* 06755 06769* 06833
07048 07061 07440 07603 07604 07667* 07687 07689
07835 08044* 08947 09205 09292 09302
BIOPOTENTIALS,GASTROINTESTINAL
00021* 00944* 02723 02775 02776
BIOPSY IN CANCER DIAGNOSIS
01219 02052* 03485* 03889* 04196 06178 06398* 06859*
06936 07258 08015 08070 08325 08326 09143 09501
09753
BIOPSY IN CHILDREN
09092
BIOPSY IN MALARSORPTION DIAGNOSIS
02281* 04314 08247
BIOPSY IN ULCERATIVE COLITIS DIAGNOSIS
01865* 04375* 06396 09423
BIOPSY,ANUS
08257* 09387 09390
BIOPSY,BILIARY TRACT
04689* 07977
BIOPSY,ESOPHAGUS
00146 00284 01888 05123 05929 06116 06118 06946
06971* 07875 09129 09140
BIOPSY,GALLBLADDER
01674
BIOPSY,GASTROINTESTINAL
00018 00130* 00788* 00943* 00982 00989 01027* 02283
02929 04685* 09092
BIOPSY,LARGE INTESTINE
00422* 01865* 01880 02935 04330* 04929 07236 08246
08263* 08330 08359* 08365 09363 09889 09920
BIOPSY,LIVER
00112* 00146 00156 00603* 00623 00646 00668 00697
00700 00729* 00738 00782 00937* 00941* 00947* 00981
00987 00991 01015 01536* 01537* 01538* 01542* 01543
01552* 01579 01592 01597 01603 01608 01612* 01675
01860* 01880 02499 02500 02540 02562 02570 02635
02649 02890* 02900* 02916* 02920* 02932 03357* 03358
03461 03464 03485* 03503 03518* 03520* 03595* 03706
03883* 03902* 03951 04005 04008 04470 04494 04587
04593 04656 04853* 05144* 05241 05440* 05456* 05497
05506 05516 05582 05612* 05693* 05694* 05892 05937
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06496 06583* 06600 06786* 06787* 06788* 06814 06947
06948 07021* 07187* 07332* 07334* 07335* 07338* 07342
07377 07420* 07427 07445* 07490* 07841* 07934 07941
07957 07971 08088 08427* 08430* 08431* 08447 08454
08474 08486 08491 08500 08502 08507 08523 08525

PSY,LIVER
NTINUED
08528 08538 08569 08580 08583 09041 09116 09236
09538 09555 09556 09591 09603 09610 09618 09662
09697
PSY,PANCREAS
04390* 04852* 05241 05968 08384
PSY,RECTUM
00422* 01398* 01865* 01880 01900 02390* 02732* 03255*
03257* 03265* 05688* 07944 08249* 08263* 08298 08338
08341 08364 08365 08751 09365
PSY,SMALL INTESTINE
00371 00436 00811 00922* 00923* 00925* 00982 01250*
01266 01269 02248 02988* 02989* 03152* 03228* 03232*
03233* 03891* 03986 04002 04231* 04234 04246 04271*
04311 04313 04314 04315 04686* 04868 05182* 05303
05851* 05886 06220* 07187* 07189* 07192* 07202 07611
07861* 07881 08063 08144 08229* 08233* 08234* 08243
08246 08263* 08918 09084 09110 09225 09234 09726
09889 09920
PSY,STOMACH
00132* 00146 00304* 00339 00789* 00834* 01034* 01166*
01167* 01177* 01209 01216 01232 01234 01242 01888
02050* 02052* 02912* 02928 02971 03106* 03889* 03890*
03940 03945 04184 04882 04958 05133* 05232* 05894
05900 06151* 06178 06188 06212 06673* 06859* 06860*
06936 06946 06951 07025* 07028* 07030* 07839* 07875
07896 08052 08068 08073 08211 08888 09179 09180
09184
EDING
E ALSO ESOPHAGUS VARICES
00182* 00411 00649 01512* 01574 01897 01934* 01944
01955 01959 01962 01964 01965 01973 01984 01989
01994 02021 02036 02048* 02123* 02213* 02214* 02216*
02218* 02232 02318* 02468 02484* 02564 02982* 02992*
03030 03104* 03160 03186* 03255* 03589* 03740 04030*
04038* 04198 04264* 04373* 04994 05733 05806* 06151*
07114 07938 08021 08024 08036* 08101 08109 08161
08199 08363 08412 08420 08438* 08439* 08532 08590
08645 08650 08740* 08899 08900 08909 08910 09117
09138 09139 09183 09231 09244 09269 09277 09529
09557 09574 09602 09628 09728 09786 09886 09897
09916 09935
EDING DIAGNOSIS
00135 00216 00956 00971 01016 01030* 01032* 01057
01089 01100 01272 01467* 01598* 01897 01927* 01929*
01931* 01955 02198* 02205* 02235 02961 02992* 03043
03044 03046 03077* 03108* 03256* 03906* 03949 04063
04073 04083 04222 04248 04273 04475 04840* 04985*
04994 04997 05011 05013 05038 05041 05042 05043
05055 05391 05897 06012 06022 06044 06086 06096
06657 06843* 06889 06891 06929 06941 06992 07131*
07318 07389 07390 07871 07889 07898 07918 07919
07968 07994* 08106 08130 08161 08295 08729* 08807
08813 09048 09060 09098 09168 09529 09850 09902
09905 09906 09908 09909 09910 09912 09928 09982
EDING DIAGNOSIS,ENDOSCOPY IN
00130* 01089 02961 03108* 03149* 05052 07873 07968
08018 09836
EDING DIAGNOSIS,RADIOLOGY IN
00952* 01089 02961 03149* 04177* 05052 05868* 05873*
05897 07968 08106 08131 08161 08873 09836 09904
EDING ETIOLOGY
00112* 00181* 00182* 00197* 00213 00215 00217 00218
00367 00549 00991 01016 01030* 01036* 01089 01108
01111 01154 01182* 01218 01238 01270 01271 01283
01307* 01333 01435 01453 01496 01571 01598* 01600*
01667 01679 01683 01860* 01868* 01926* 01927* 01928*
01929* 01931* 01952 01961 02009* 02010* 02048* 02139
02216* 02298* 02418 02452 02628* 02640 02890* 02994*
02998* 03003 03043 03044 03046 03073* 03077* 03108*
03113* 03134 03149* 03206 03256* 03336* 03558 03563
03636 03883* 03884* 03949 04025* 04038* 04042* 04063
04067 04073 04094 04099 04100 04118 04129* 04158
04168* 04202 04206 04212 04218 04220 04238 04244
04248 04250 04254 04273 04285 04295 04300 04340
04342 04366 04433* 04475 04504 04537 04613 04621
04641 04659 04840* 04978* 04983* 04985* 04994 04997
05011 05012 05013 05036 05038 05042 05043 05052
05055 05088* 05100 05109 05178* 05234 05265 05319*
05350 05390 05391 05493 05550* 05597* 05625 05655
05868* 05897 05903 05981* 05994* 06005* 06008* 06012
06022 06044 06064 06086 06124 06241* 06243* 06250*
06262* 06291 06326* 06332* 06342 06438 06527 06559*
06582* 06587 06597 06610 06612 06615 06637 06657
06941 06992 07011* 07013* 07043 07044 07067 07069
07092 07113 07131* 07136* 07141 07183* 07227 07266
07316 07318 07368 07389 07390 07406 07412 07464*
07465* 07483 07562 07871 07873 07922 07994* 08018
08033* 08040* 08042* 08106 08123 08137 08151 08161
08295 08308 08370 08448 08643 08729* 08757 08761
08771 08773 08791 08807 08816 08817 08836 08840
08845 08855 08864 08873 08899 08900 08912 09139
09156 09174 09275 09295 09296 09310 09461 09477
09479 09565 09573 09623 09638 09655 09724 09730
09812 09836 09850 09902 09903 09904 09905 09906

BLEEDING ETIOLOGY
CONTINUED
09907 09908 09909 09910 09912 09928
BLEEDING IN CHILDREN
00216 00932* 01271 01363 01926* 03481* 03558 04113
04212 05052 06332* 08308 08729* 08900 09528 09575
09655 09683 09969
BLEEDING PEPTIC ULCERS
00182* 00395* 00416 01016 01032* 01297* 01312* 01321*
01328 01360 01956 01958 01961 02045* 02194* 02195*
02198* 02205* 02213* 02217* 02237 02239 02263 02998*
03004 03044 03182* 03202 03203 03206 03408 03949
04063 04073 04212 04255* 04260* 04261* 04262* 04266*
04270* 04273 04277 04285 04292 04295 04298 05221*
05249 05256 05265 05267 06002* 06177 06241* 06243*
06246* 06254* 06257* 06274 06283 07133* 07396 07968
08193 08424* 08448 08779 08813 08816 08832 08836
08842 08845 09048 09252 09275 09285 09295 09296
09479 09905 09907 09908 09909 09912 09927 09935
BLEEDING TREATMENT
00112* 00215 00267 01056 01087 01130 01258* 01296*
01927* 01955 02010* 03043 03044 03046 03073* 03202
03206 03529* 03884* 03949 04038* 04063 04128* 04174*
04273 04285 04298 04433* 04475 04926 04994 04997
05013 05038 05042 05043 05146* 05265 05603* 05625
06012 06022 06099 06182 06241* 06274 06567 06610
06657 06973* 06980 07009* 07062 07131* 07389 07563
07871 07998 08027 08286 08315 08457 08471 08761
08813 08836 08855 09139 09156 09252 09296 09449
09552 09613 09708 09724 09828 09850 09903 09911
09912 09925 09969
BLEEDING,AGE EFFECTS ON
01931*
BLEEDING,COOLING TREATMENT OF
00356 00989 01089 02010* 04174* 05043 05994* 06002*
06022 06770* 07009* 07470* 08018 08027 08813 09139
09911
BLEEDING,DRUG-INDUCED
06064 06086
BLEEDING,GASTROINTESTINAL
00135 00181* 00215 00216 00217 00218 00267 00356
00361 00932* 01060 01130 01238 03102* 03173 07972
08173* 09964
BLEEDING,SURGICAL TREATMENT OF
00217 00264* 00395* 00607* 00656 01032* 01089 01218
01272 01363 01600* 01642* 01926* 01956 02045* 02199*
02205* 02217* 02263 02318* 03009 03044 03046 03102*
03149* 03206 03355* 03949 04177* 04212 04220 04262*
04269* 04273 04285 04292 04298 04342 04371* 04490
04598* 04599* 04606* 04997 05011 05038 05042 05043
05100 05221* 05231* 05249 05256 05265 05267 05981*
06022 06064 06096 06124 06254* 06274 06283 06578*
06579* 06583* 06587 06596 06597 06601 06970* 06973*
06995 07390 07406 07429 07465* 07468* 07470* 07473
07483 07486 07562 07871 07984* 07994* 07996* 08109
08173* 08193 08482 08632* 08813 08816 08842 08845
08873 09139 09156 09168 09285 09288 09296 09304
09497 09539 09613 09703 09927
BLIND LOOP SYNDROME
08145
BLIND LOOP-INDUCED SECONDARY MALABSORPTION
00345* 02664* 03981 04124 04242 04729* 06219* 06844*
07188* 08228*
BLOOD COAGULATION
00411 00531* 00905 01079 01297* 01958 01965 02198*
02992* 03014 03479* 03663 03884* 04270* 04374* 05077
06129 06177 06524 06530 06815 07336* 07355 07664
08855 08900 09890
BLOOD COAGULATION IN CIRRHOSIS
03611 04504 04614 04885 06612 06615 08639 08643
09715
BLOOD COAGULATION IN LIVER DISEASES
00599* 00644 00659 03535 04504 04537 04885 05427*
05493 06559* 07350* 08477 08639 09529 09554 09573
09683
BLOOD COAGULATION IN ULCER DISEASE
02229 03203 05234
BLOOD GROUP GENETIC RELATIONSHIPS
00389* 01317* 01617* 02060 05149* 05616 08244 09017
09734
BLOOD GROUP SECRETOR STATUS
00389* 04270* 06748* 08177* 09017
BLOOD GROUPS
00003* 00316 02229 03113* 05131* 05569* 05610* 06456*
07015* 07369 08177* 08624 09111 09600 09903
BONE DISEASES
05156 05281 05430* 06646 08425* 09576
BONE DISEASES,GASTRECTOMY-INDUCED
04171* 04187 04204 04219 04221 04278 04940 05070
05071 05072 08041* 08103 08885
BORBORYGMUS
01976 04074
BOTULISM
02024

BRADYKININ
00002* 00080 05754* 06723* 06768 07663 08400*
BROMELAINS
03785* 05382*
BUDD-CHIARI SYNDROME
00607* 00610 00645 01538* 01558 01885 01930* 05848*
06489 06842* 08454 08530 09544 09545 09585 09733

CALCIUM
03746* 03763* 03814* 04171* 04187 04221 04278 05028
05218* 05233* 05289* 05430* 05645* 05743* 07328 07381
07477 08928 08969 09019 09022 09458
CALCIUM ABSORPTION
00030 00031 00032 00393* 00396* 00893* 00894* 00978
01037* 01192 01315* 01726* 01739 01842 02738 02752*
02802* 03234* 03500 03679 04219 04309* 04310* 04717*
04719* 04940 04941 04951 05070 05071 05072 05305
05306 05720* 05846* 07289 07309* 07631* 07632* 07642*
07655 08103 08110 08937 08942 08988 09106 09465
CALCIUM EXCRETION
01245
CALCIUM METABOLISM
08885 09237
CALCIUM SECRETION,PANCREAS
00547 00589 01521 [illegible] 06429*
CALCULI,INTESTINAL
00366
CALCULI,PANCREAS
02435 03324 06407 08388 09079
CANCER
01118 02100
CANCER CHEMOTHERAPY
00227 00322 01020* 01212 01447 02055* 02056* 02073
02307* 02310* 02950 02975* 03079* 03376* 03474 03904*
05067 05998* 06091 06095 06156* 06199 06322 06370
06398* 07286 08070 08083 08722* 08742* 09502 09577
09809 09851
CANCER DIAGNOSIS
00114* 00300* 00306* 00318 00331 00334 00346* 00678
00837* 00929* 00930* 00935* 00937* 00948* 00965 00975
00983 00992 01096 01118 01169* 01185 01186 01216
01232 01233 01288 01396* 01488* 01498 01501 01563
01578 01585 01852* 01861* 01863* 01867* 01885 01890
01895 01941* 01942 02041 02047* 02097 02131 02304*
02306* 02372 02373 02390* 02426 02487 02525 02548
02665* 02885* 02886* 02890* 02901* 02912* 02947 02958
02960 03075* 03256* 03270 03296* 03309* 03357* 03485*
03595* 03625* 03887* 03893* 03896* 03901* 03916* 03918*
03921* 03938 03939 03941 03992 03994 03995 03996
03998 04004 04005 04008 04146 04183 04184 04185
04186 04191 04196 04215 04223 04231* 04244 04251
04327* 04328* 04357 04381* 04390* 04400 04405 04455*
04464 04691* 04837* 04844* 04852* 04866 04867 04873
04882 04890 04891 04893 04901 04906 04917 04943
04944 04945 04946 04953 04955 04961 04962 05039
05103 05107 05168 05387* 05391 05396 05467 05472
05640* 05835* 05849* 05857* 05863* 05882* 05884* 05892
05896 05911 05912 05918 05920 05921 05923 05929
05942 05943 05953 05964 05969 06021 06055 06104
06141 06212 06358 06359 06409 06493 06621* 06837*
06838* 06843* 06850* 06860* 06864* 06875* 06876* 06880*
06882* 06888 06889 06891 06893 06899 06900 06904
06919 06920 06921 06923 06927 06928 06939 06943
06949 06959 06963 06979 07018* 07019* 07020* 07053
07076 07221 07226 07260 07279* 07846* 07850* 07862*
07869 07875 07877 07888 07893 07900 07907 07908
07917 07931 07935 07950 07953 07965 08066 08082
08096 08106 08125 08155 08258* 08262* 08297 08320
08322 08355 08368 08382 08383 08392 08460 08530
08675 09031 09033 09040 09049 09064 09065 09081
09094 09125 09173 09190 09193 09392 09435 09437
09440 09443 09457 09463 09493 09499 09749 09758
09770 09796 09813
CANCER DIAGNOSIS,ANGIOGRAPHY IN
03486* 07847*
CANCER DIAGNOSIS,BIOPSY IN
01219 02052* 03485* 03889* 04196 06178 06398* 06859*
06936 07258 08015 08070 08325 08326 09143 09501
09753
CANCER DIAGNOSIS,ENDOSCOPY IN
00132* 01137* 02912* 03889* 06859* 06913 06936 07258
08015 08070 08326 09101 09144 09149 09364 09371
CANCER DIAGNOSIS,LYMPHOGRAPHY IN
04933 05906 08096
CANCER DIAGNOSIS,RADIOLOGY IN
00106* 00132* 00296* 00920* 00963 00969 00999 01053
01178* 01183* 02050* 02052* 02369 02373 02557 02927
02966 03075* 03099* 03105* 03269 03485* 03486* 03486*
03889* 04362 04890 04900 04933 04937 05839* 05874*
05906 06222 06842* 06910 06936 07066 07120 07258
07286 07847* 07880 07901 08015 08070 08305 08326
08382 09051 09056 09085 09101 09113 09364 09371
09377
CANCER EPIDEMIOLOGY
00292* 01169* 02060 02077 02306* 02312* 03024 03026
03098* 03276 03296* 03309* 04053 04093 04146 04175*
04185 04188 04251 04327* 04352 04455* 04464 04485
05103 05104 05130* 05131* 05132* 05139* 05212 05356
05387* 05425* 05467 06055 06142* 06161* 06222 06456*
06604 06990 07007* 07015* 07018* 07032 07041 07258
07338* 07342* 07343* 07372 07547* 08017 08020 08038*
08071 08078 08086 08089 08368 08505 08706 08764
08886 09190 09371 09813
CANCER ETIOLOGY
00292* 00296* 00344 00532* 00534 01169* 01446 01479
01687 02312* 02642 03098* 03107* 03131 04050* 04146
04157 04175* 04188 04361 04455* 04464 04510 04610
05083* 05103 05130* 05131* 05139* 05222* 05425* 05437*
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06198 06209 06358 06361 06604 06639 06987 07008*
07018* 07022* 07023* 07126* 07146 07147 07221 07258
07547* 07587 07740* 08020 08028 08038* 08086 08089
08090 08102 08167* 08252* 08288 08300 08505 08544*
08550* 08553* 08675 08719* 08764 08821 08886 09149
09188 09293 09536 09537 09542 09717
CANCER IMMUNOLOGY
00908 01581 03678 03896* 03901* 04050* 05849* 07055
08553* 09353 09921
CANCER IN CHILDREN
00293* 01222 01578 04610 05964 07035 07234 08460
09501 09609
CANCER METABOLISM
03463 03875 03876 03877 03878 05865* 06788* 07019*
08903
CANCER PATHOLOGY
00296* 00300* 00948* 01179* 01196 01378* 02041 02061
02368 02369 03075* 04146 04185 04464 05103 05142*
05437* 06179 06183 06191 06218* 07008* 07066 07073
07086* 07343* 07999 08039* 08047 08086 08096 08111
08318 08322 08383 08711 08719* 08876 09143 09144
09149 09190 09353 09357 09435 09437 09454 09501
09516 09717 09749 09753 09832
CANCER PROPHYLAXIS
08020 08090 08147 08336 08337 08368 08388 08505
08706
CANCER SURGERY
00260 00346* 00446* 00551 00624 00632 00929* 01017*
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01162 01170* 01183* 01224 01225 01227 01233 01253*
01279 01281 01380* 01396* 01448 01488* 01501 01654*
01942 02008* 02039 02044* 02055* 02056* 02058* 02095
02304* 02306* 02309* 02310* 02329* 02340 02367 02369
02371 02407* 02495 02665* 03075* 03117* 03262* 03276
03292 03333* 03625* 04124 04126* 04140 04146 04151
04156 04194 04198 04201 04203 04208 04244 04246
04327* 04333* 04348 04355 04390* 04975* 04991 04999
05081* 05089 05107 05130* 05132* 05153 05158 05189
05212 05222* 05310* 05313* 05327 05328 05329 05339
05340 05356 05384* 05387* 05396 05640* 05651 06055
06061 06077 06122 06131 06135 06178 06184 06186
06199 06202 06344 06355 06364 06367 06370 06403
06410 06447* 06478 06635 06882* 06936 06963 06972*
06990 06996 06997 07003 07007* 07026* 07029* 07034
07035 07052 07054 07065 07066 07120 07146 07207*
07208* 07209* 07232 07240 07247 07257 07260 07291
07520 08015 08017 08037* 08039* 08064 08070 08071
08074 08086 08109 08111 08125 08155 08257* 08260*
08261* 08262* 08265* 08274 08275 08296 08302 08309
08310 08314 08320 08322 08325 08326 08355 08383
08433* 08510 08514 08517 08529 08535 08675 08717*
08876 08877 09101 09131 09144 09173 09190 09353
09364 09371 09377 09384 09392 09437 09440 09443
09457 09463 09493 09516 09562 09569 09743 09749
09753 09796 09803 09831 09892
CANCER SURVIVAL RATES
01936* 02058* 02097 02482* 03072* 03075* 03281 04332*
04333* 05081* 05087* 05106 05139* 05313* 05384* 05387*
05640* 06108* 06398* 06464* 06972* 07007* 07026* 07029*
07052 07086* 07120 07207* 07209* 07338* 08037* 08039*
08064 08071 08086 08090 08260* 08261* 08262* 08265*
08296 08310 08314 08320 08325 09173 09353 09364
09770
CANCER TREATMENT
00227 00260 00346* 00447* 00466 00929* 00935* 00945*
01020* 01029* 01033* 01096 01128 01136* 01160 01170*
01186 01212 01219 01224 01225 01233 01279 01380*
01396* 01409* 01447 01448 01501 01557 01654* 01687
01941* 01942 02008* 02044* 02055* 02056* 02073 02095
02097 02304* 02306* 02307* 02309* 02310* 02329* 02360
02548 02665* 02950 02975* 03072* 03075* 03079* 03082
03259* 03262* 03270 03276 03292 03293 03333* 03376*
03474 03625* 03904* 04126* 04155 04185 04251 04333*
04400 04991 04999 05023 05081* 05088* 05089 05132*
05158 05222* 05313* 05327 05339 05384* 05387* 05396
05998* 06109* 06156* 06344 06364 06398* 06403 06478
07066 07209* 07257 07258 07286 07989* 08016 08017
08037* 08039* 08064 08071 08072 08083 08086 08257*
08260* 08261* 08262* 08297 08309 08310 08314 08320
08326 08383 08433* 08514 08517 08529 08679 08877

NCER TREATMENT
ONTINUED
09101 09128 09131 09170 09173 09190 09353 09364
09371 09377 09384 09392 09432 09437 09502 09569
09577 09590 09753 09770 09796 09803 09832 09838
09892
NCER TREATMENT,RADIATION THERAPY IN
00262* 01053 01128 01137* 01269 01380* 01427 01461
01488* 03079* 03259*
NCER TREATMENT,RADIOTHERAPY IN
01993 02008* 02035 02056* 02297 02309* 04065 04143
04146 04246 04333* 04866 05081* 05106 05107 05328
05329 06010 06047 06108* 06119 06128 06129 06135
06199 06336 06370 06398* 06936 06972* 06996 07565
08015 08070 08083 08302 08322 08325 08498 08722*
08876 09144 09159 09376 09831
NCER,ABDOMEN
04946 07559
NCER,AGE FACTORS IN
04093 04175* 04348 05131* 05139* 05313* 05338 07007*
07086* 07247 07343* 08066 08070 08071 08078 08089
08320 08764 09501 09562
NCER,ANUS
01407* 01461 04357 05328 05329 07232 07258 07260
08257* 08260* 08345 09390
NCER,APPENDIX
01446 02183 09892
NCER,BILIARY TRACT
01020* 01134 01686 02557 02692 03401 04082 04102
04664 04886 04890 04999 05393 05504 05651 06892
07516 07520 07521 07931 08470 08505 08553* 08678
08706 09542 09577 09707 09743 09749 09753 09758
09770 09796 09798 09801
NCER,BIOCHEMICAL DIAGNOSIS OF
01179* 01585 02537 05143* 06621* 07739* 09033
NCER,CLINICAL STUDIES OF
00259 00290* 00344 00945* 01240 02058* 02077 02085
02309* 02310*
NCER,DISEASES ASSOCIATED WITH
00331 00625 01188 01189 01370 02298* 02396* 02497
02551 03047 03400 03918* 04361 04475 05074 05075
06104 06198 06204 06361 06527 07021* 07050 07287
07343* 07394 07521 07545* 07585 08004 08090 08167*
08220* 08318 08374* 08428* 08505 08675 08706 08877
09190 09360 09432 09457
NCER,DUODENUM
06202 06222 06227 06410 07120 08125 08678
NCER,ESOPHAGUS
01020* 01150 01153 01165 02025 02039 02041 03072*
03075* 03082 03109* 04053 04126* 04132 04140 04143
04146 04151 04154 04155 04156 04157 04999 05081*
05083* 05087* 05088* 05089 05103 05104 05106 05107
05117 05119 05124 05698* 05865* 05929 06077 06108*
06109* 06119 06121 06122 06128 06129 06135 06141
06838* 06972* 06987 06990 06996 06997 07003 07587
07875 07989* 07999 08004 08013 08016 08017 08020
08028 08028 08246 08487 08821 09051 09094 09113
09128 09131 09135 09143 09147 09149 09159 09832
NCER,EXPERIMENTAL STUDIES OF
01581 01582 01583 01585 01586 02077
NCER,GALLBLADDER
01531* 01650* 01654* 01674 01679 01942 02975* 02986*
03382* 03625* 04659 06621* 06622* 06627* 06639 07528
07931 08675 08680 08706 08711 09527 09743 09770
09772
NCER,GASTROINTESTINAL
02056* 02155 02950 03010 03022 03024 04053 04073
04093 04124 05015 05067 05865* 06001* 06021 06077
06091 06359 06899 07155 07547* 07569 07585 07901
08719* 08764 08821 08876 08903 09094 09813 09838
09892
NCER,LARGE INTESTINE
00106* 00114* 00259 00448* 00454* 00460 00464 00465
00534 00948* 00963 01017* 01020* 01033* 01047* 01128
01380* 01383* 01396* 01403* 01426 01427 01447 01457
01936* 01941* 01969 02290 02298* 02306* 02307* 02309*
02310* 02312* 02329* 02332* 02336 02340 02369 02373
02379 02387* 02390* 02394* 02395* 02901* 02975* 03053
03254* 03256* 03270 03272 03276 03292 03296* 03297*
03941 03997 03998 04053 04082 04327* 04328* 04332*
04333* 04334* 04348 04351 04352 04355 04357 04361
04362 04381* 04867 04999 05310* 05311* 05313* 05338
05339 05346 05356 05865* 05943 06049 06061 06330*
06357 06361 06363 06367 06882* 06949 07207* 07208*
07209* 07213* 07221 07226 07232 07234 07237 07238
07247 07258 07273 07557 08220* 08252* 08253* 08258*
08260* 08261* 08262* 08265* 08271 08274 08296 08300
08302 08305 08309 08310 08314 08318 08320 08337
08355 08368 08722* 08742* 09101 09353 09357 09377
09384 09391 09415 09577 09832 09889 09892 09897
09922
NCER,LIVER
00113* 00114* 00609 00624 00937* 01033* 01531* 01533*
01537* 01563 01578 01581 01583 01585 01586 01593
01837 01852* 01861* 01862* 01863* 01887 02487 02497
02524 02525 02537 02542 02548 02551 02557 02565
CANCER,LIVER
CONTINUED
02628* 02634* [illegible] 02878 02884* 02890* 02916* 02927
02958 02975* 03357* 03365* 03376* 03383* 03391 03400
03414 03418 03452 03463 03485* 03486* 03595* [illegible]
03875 03876 03877 03878 03879 03901* 03904* 03938
03939 03954 04005 04008 04013 04050* 04053 04450*
04455* 04459* 04464 04466 04471 04475 04485 04514
04610 04886 04900 04901 04937 04943 04946 05425*
05425* 05427* 05441* 05467 05468 05472 05484 05519
05617 05849* 05874* [illegible] 06456* 06464* 06478 [illegible]
06493 06527 06604 06788* 06837* 06842* 06893 07338*
07342* 07343* [illegible] 07372 [illegible] [illegible] 07476 07585
07707* 07877 07950 [illegible] 08427* 08428* 08433* [illegible]
08498 08500 08505 08510 08514 08517 08529 [illegible]
08535 08550* 08553* 08582 08644 [illegible] 08725* 08742*
08876 09033 09047 09049 [illegible] [illegible] [illegible] 09493
09499 09502 09527 09536 09537 09562 09569 [illegible]
09577 09588 09589 09590 [illegible] 09609 09614 [illegible]
09707 09717
CANCER,METASTATIC
00114* 00609 00624 00929* 00937* [illegible] 01033* 01170*
01235 01282 01396* 01533* 01583 01861* 01862* 01863*
01928* 02041 02061 02096 02136 02155 02340 02342
02482* 02524 02890* 02916* 02958 [illegible] 03133 03309*
03365* 03382* 03414 03901* 03942 04013 04022* 04048*
04146 04151 04196 04206 04362 04485 04945 05018
05044 05047 05391 05698* 05921 05969 06156* 06164
06171 06204 06234 06347 [illegible] 06363 06464* 06480
06493 06617* 06621* 06788* 06837* 06864* 06963 06996
07007* 07029* 07050 07067 07092 07209* 07338* 07342*
07343* 07559 07869 07879 07950 [illegible] [illegible] [illegible]
08111 08257* 08261* 08302 08320 08384 08392 08428*
08487 08498 08505 08514 08720* 08730* 08862 08876
08877 09033 09144 09173 09353 09364 09435 09437
09458 09502 09516 09577 09590 09717
CANCER,PANCREAS
00551 00560 00860 00930* 00988 01033* 01112 01134
01488* 01489* 01491* 01498 01658* 01873* 01889 01890
01942 02412* 02414* 02419 02420 02426 02781* 02927
02975* 03309* 03319* 03320 03321 03326 03333* 03894*
03896* 03904* 03916* [illegible] 03992 03994 03995 04102
04122 04208 04381* 04386* 04387* 04390* 04395 04396
04400 04405 04851* 04852* 04866 04893 04917 04937
04944 04946 04961 04962 04999 05017 05039 05053
05075 05186* 05384* 05387* 05391 05393 05396 05651
05855* 05911 05953 [illegible] 06398* 06409 06410 06432
06635 06880* 07279* 07286 07287 07291 07297 07585
07838* 07847* 07880 07907 08045* 08374* 08382 08383
08388 08392 08742* 08764 08862 09049 09432 09435
09437 09440 09443 09451 09454 09457 09467 09577
09769
CANCER,RECTUM
00447* 00466 00935* 01017* 01020* 01033* 01380* 01383*
01407* 01426 01427 01448 01936* 02304* 02306* 02309*
02310* 02342 02360 02367 02371 02373 02975* 02977*
03256* 03259* 03276 03293 04328* 04332* 04333* 04351
04352 04355 04361 05313* 05327 05329 05338 05339
05340 05356 05865* 06330* 06350 06358 06359 06361
06367 06370 06882* 06888 06949 07206* 07207* 07213*
07232 07257 07258 08220* 08260* 08265* 08274 08275
08288 08296 08302 08310 08320 08322 08325 08326
08345 08368 08722* 08862 09101 09364 09371 09376
09377 09392 09415 09577 09892 09982
CANCER,SMALL INTESTINE
00259 01128 01253* 01281 01282 01501 01943 02118*
02131 02155 02191 02290 02977* 03225 04053 04185
04244 04246 04699 04890 05017 05212 05894 06222
06223 06227 06234 07081* 07086* 07557 08147 08149
08155 08220* 08220* 08392 08743* 09832 09843 09875
09889 09892
CANCER,STOMACH
00290* 00292* 00296* 00298* 00300* 00306* 00316 00318
00322 00331 00333 00344 00609 00837* 00929* 00935*
00948* 00963 00975 00999 01020* 01028* 01033* 01035*
01047* 01090 01129 01167* 01169* 01170* 01175* 01178*
01179* 01182* 01183* 01186 01188 01189 01190 01212
01215 01216 01219 01222 01224 01226 01227 01232
01235 01239 01240 01241 01243 01244 01895 01936*
01941* 02044* 02047* 02049* 02050* 02053* 02055* 02058*
02060 02061 02073 02077 [illegible] 02092 02095 02096
02097 02234 02407* 03105* 03887* 03889* 03907* 03921*
03996 04004 04053 04063 04155 04175* 04183 04184
04185 04186 04188 04191 04193 04194 04196 04198
04203 04206 04208 04215 04223 04275 04299 04691*
04699 04882 04891 04997 [illegible] 05081* 05130* 05131*
05132* 05134* 05135* 05139* 05142* 05143* 05158 05161
05168 05222* 05835* 05863* 05865* 05884* 05894 05912
05923 05929 06049 06061 06143* 06148* 06150* 06156*
06161* 06164 06168 06170 06171 06178 06179 06183
06184 06191 06198 06199 06202 06204 06212 06748*
06755 06788* 06838* 06843* 06859* 06904 06936 07007*
07008* 07014* 07015* 07018* 07019* 07020* 07021* 07023*
07026* 07029* 07032 07034 07035 07041 07048 07050
07051 07052 07053 07054 07055 07056 07061 07065
07066 07071 07072 07073 07076 07146 07147 07155

CANCER,STOMACH
CONTINUED
07181 07286 07329 07545* 07587 07850* 07869 07900
07916 08037* 08038* 08039* 08042* 08044* 08047 08049
08055 08058 08064 08066 08070 08071 08076 08078
08082 08083 08086 08089 08090 08091 08093 08096
08101 08102 08109 08111 08246 08392 08620 08669
08722* 08725* 08742* 08764 08836 08913 09031 09048
09051 09056 09064 09065 09085 09094 09172 09173
09188 09190 09193 09194 09197 09206 09212 09213
09293 09577 09921 09927
CARBOHYDRATE ABSORPTION
00011* 00025* 00129* 00425* 00428* 00433* 00765* 00814*
00817* 00820 00827 00926* 01260* 01371* 01721* 01722*
01724* 01725* 01741 01825* 01870* 01923* 02051* 02283*
02284* 02291 02293 02391* 02712 02748* 02758* 02759*
02763* 02765 [illegible] 02913* 03110* 03116* 03228* 03244*
03245* 03373* 03673* 03720* 03727* 03728* 03729* 03745
03913* 04044* [illegible] 04091 04092 04170* 04227* 04240
04312 04312 04313 04320 04325 04680 04713* 04715*
04721* 04736 04737 04738 04739 04863* 04959 04988*
05293 05361* 05386* 05480 05711* 05714* 05719* 05722*
05723* 05724* 05732 05780 06228 06691 06699* 06701*
06702* 06703* 06706* 06707* 06714 06717 06810 06836*
06851* 06861* 07112 07123 07194* 07197 07202 07203
07391 07440* 07577 07629* 07630* 07650 07652 08043*
08225* 08231* 08234* 08241 08244 08495 08508 09083
09175 09218 09325 09333 09336 09718
CARBOHYDRATE DIGESTION
00933* 01657* 02203* 03677 03687 03803 04044* 04079
04092 04253 05672* 05780 06695* 07577 07650 07652
07668* 08043*
CARBOHYDRATE INTOLERANCE
05985* 05986* 07187*
CARBOHYDRATE MALABSORPTION
00923*
CARBOHYDRATE METABOLISM
00432* 01828* 01831* 01913 01939* 02129* 02712 03100*
03116* 03613 03668* 03673* 03675* 03745 04079 04170*
04384 04386* 04393 04397 04434* 04608* 04680 04737
04801* 04826 04829 04878 04988* 05407 05410 05592*
05593* 05625* 05654 05780 06004* 06717 06810 06826*
07132* 07191* 07277* 07371 07629* 07668* 07781 08043*
08947 08980 09333 09444 09668
CARBOHYDRATE METABOLISM,LIVER
00083* 00091* 00094 00102 00612 00622 00732* 00737
00743 00947* 01589 01823* 01831* 01836 02518 02699*
02844 02847 02848 02849 02853 02854 02859 02871
02881 02951 03397 03430 03606 03819* 03824* 03829
03837 03839 03840 03850 03853 03856 03860 03861
03862 03863 03878 04320 04518* 04528* 04545 04604*
04791* 04792* 04807* 04821 04822 05033 05438* 05439*
05505 05529* 05561 05563 05689* 05811* 05812* 05821*
05823* 05826 05831 05832 05950 06228 06480 06543
06550 06551 06608 06778* 06786* 06787* 06788* 07187*
07355 07430 07709* 07713* 07714* 07732* 07747* 07761
07773 07785 07796 07801 08543* 08545* 08560* 08563
08597* 08657* 08933 08971 08982 09531
CARBOHYDRATES
00047* 00153 02666 02812 05738* 05749* 07624* 07704*
07783 07894
CARBOXYPEPTIDASE SECRETION,PANCREAS
00836* 00860 03689 03690 03691 03692 04389*
CARCINOGENESIS
00185* 00330 00464 03314* 06161* 09363
CARCINOGENESIS,LARGE INTESTINE
01437 02356 05357 05359 07229
CARCINOGENESIS,LIVER
00907 01556 01581 01582 01584 01586 02498 02505
02532 02561 02642 03391 03452 03453 03846 03873
03874 03875 04469 04505 04510 04514 04531 04831
05425* 05437* 05468 05544* 05554 06534* 06604 07342*
07380 07421* 07430 07437 07438 07441 07587 07739*
07740* 07741* 08506 08542* 08543* 08544* 08550* 08552*
08553* 08555* 08556* 09536 09537 09588 09589 09614
09656 09660
CARCINOGENESIS,STOMACH
01173* 01233 01243 01702* 01943 02082 02088 02108
02258 04122 06142* 06209 07008* 07014* 07018* 07022*
07023* 07126* 08080 09194
CARCINOGENS
01702* 04122 04505 06142* 06209 07437 07438 08886
09002 09660 09921
CARCINOID SYNDROME
SEE ALSO CARCINOIDS
SEE ALSO SEROTONIN
00089* 00370 00541* 00570 01071 02057* 02368 02977*
03006 03015 03067 03121 03227* 04117 04246 05017
05023 05314* 06078 06368 07258 08266* 08285 08498
08877 09446 09828 09832 09892
CARCINOIDS
SEE ALSO CARCINOID SYNDROME
00089* 00370 01051 01070 01072 01073 01074 01075
01076 01077 01253* 01262 01263 01282 01420 02057*
02118* 02154 02696 02977* 07011* 08137 08147 08281
08498 08780 08856
CARCINOMA
00224 00262* 00290* 00316 00332 00333 00460 00465
00474 00476 00530 00650 00778 00780 01170* 01190
01196 01253* 01335 01378* 01391* 01392* 01414 01421
01537* 01685 01687 02008* 02097 02131 02155 02191
02304* 02306* 02342 02371 02379 02685 02692 03072*
03079* 03133 03175 03254* 03262* 03292 03314* 03896*
04102 04362 04657 04678 05083* 05087* 05103 05865*
06027 06049 06055 06141 06148* 06161* 06222 06398*
06627* 06639 06990 07018* 07026* 07048 07061 07066
07338* 07372 07394 07569 07931 07950 07953 07999
08017 08058 08076 08080 08082 08102 08111 08167*
08243 08309 08355 08392 08428* 08517 08529 08675
08711 08719* 08764 09047 09051 09056 09065 09081
09193 09206 09212 09371 09376 09377 09390 09391
09419 09432 09440 09443 09467 09527 09542 09574
09770 09772 09796 09801 09932 09982
CARCINOSARCOMA
00279 08015
CARDIOSPASM
SEE ALSO ACHALASIA
06974* 07600
CARDIOVASCULAR SYSTEM
SEE ALSO PORTAL HYPERTENSION
00698 00699 01132 01175* 01668 02435 02523 02564
02574 03011 03100* 03257* 03603 04272* 04294 04860*
05111 05989* 06261* 06375 06386 06452* 06454* 06472
06564 06581* 06629* 06666* 06679* 06771* 06866* 06886
07075 07254 07279* 07287 07340* 07344* 07360 07379
07396 07412 07550* 07553 07831 07879 07897 07912
07926 07944 07969 08062 08063 08118* 08162 08163
08259* 08440* 08473 08482 08484 08492 08532 08534
08567 08645 08674 08690 08719* 08728* 08760 08771
08792 08793 08805 08831 08837 08850 08853 08870
08887 08911 08923 08995 09027 09061 09132 09141
09232 09262 09457 09523 09535 09546 09567 09568
09682 09706 09707 09720 09727 09735 09786 09857
09867 09902 09904 09935
CAROTENE ABSORPTION
SEE ALSO VITAMIN A ABSORPTION
09082
CARRIER STATE,SALMONELLOSIS
04045* 05019 06856* 08775 08786 09953
CARRIER STATE,TYPHOID FEVER
06856* 07905 08803 09848
CATHARTICS
SEE LAXATIVES
CECUM
SEE ALSO LARGE INTESTINE
00224 00451* 00498 00499 00512 00530 00816* 01050
01391* 01392* 01396* 01412 01414 01446 01463 01742
02298* 02300* 02388* 03067 03161 03272 03291 04065
04342 04366 04672* 04987* 05025 05197 05323 05908
06026 06027 06028 06067 06318 06337 06354 07216*
07252 07628* 08262 08324 09391 09394 09395
CELIAC DISEASE
00354* 00433* 01255* 01373* 02274 02280* 02293 02324*
02911* 03051 03231* 03235* 03243* 03248 03989 04002
04230* 04311 04312 04316 04317 04318 05284* 05287*
05290* 05291* 05293 05299 06863* 07189* 07197 08147
08158 08247 08248 08898 09074 09082 09083 09320
09324 09325 09326 09328 09334 09855
CELLULAR INFILTRATION
01208 06810 07565 08071 09480
CENTRAL NERVOUS SYSTEM
SEE ALSO NEUROENDOCRINE CONTROL
00038 00559 00676 01149 01175* 01189 01759* 01970
02448 02473* 02799* 02809* 02979* 03198 03592* 03602
03754* 03758* 03792* 03833 04015 04129* 04321 04381*
04491 04605* 04620 04622 05145* 05404 05426* 05489
05514 05547* 05618 05656 05678 05679 05732 06084
06157* 06206 06243* 06261* 06445 06475 06476 06607
06649 06741* 07017* 07039 07170 07344* 07392 07458
07466* 07572 07662* 07950 08080 08179 08203 08270
08411 08453 08455 08461 08479 08496 08499 08500
08614 08638* 08728* 08870 09264 09639 09640 09658
09710 09812 09842 09844 09917 09985 09989
CERULOPLASMIN
01611* 01646* 07467* 07770 07893 09534
CHAGAS DISEASE
03063
CHEMICAL COMPOSITION,LIVER
01547* 03501 03822* 05456*
CHEMICAL COMPOSITION,PANCREAS
04669*
CHEMICAL COMPOSITION,SALIVARY GLAND
05381*
CHEMICAL COMPOSITION,SMALL INTESTINE
02734* 04733
CHEMICAL COMPOSITION,STOMACH
00040* 00041* 00064* 00134 03694* 03769* 05702 05762*
06307
CHEMICAL TOXINS
SEE ALSO TOXIC EFFECTS ON ABDOMEN

ICAL TOXINS
TINUED
ALSO TOXIC EFFECTS ON BILIARY TRACT
ALSO TOXIC EFFECTS ON ESOPHAGUS
ALSO TOXIC EFFECTS ON GASTROINTESTINAL TRACT
ALSO TOXIC EFFECTS ON LARGE INTESTINE
ALSO TOXIC EFFECTS ON LIVER
ALSO TOXIC EFFECTS ON PANCREAS
ALSO TOXIC EFFECTS ON RECTUM
ALSO TOXIC EFFECTS ON SALIVARY GLANDS
ALSO TOXIC EFFECTS ON SMALL INTESTINE
ALSO TOXIC EFFECTS ON STOMACH
0010 00014* 00022* 00176 00287 00289* 00628 00629
0683* 00685* 00692 00693 00694 01257* 01469* 01501
1633* 01734* 02069 02545 02580* 02594 02595 02596
2747 03479* 03501 03513 03514 03516 03874 04633*
5731* 05442* 05533* 05534* 05541* 05542* 05544* 05549*
5553* 05555 05820* 06205 06536* 06549 07424 07431
7432 07514 07587 07706* 07716* 08134 08135 08560*
8563 08956 08975 08976 08987 09536 09537 09543
9650 09662
CTHERAPY,CANCER
0227 00322 01020* 01212 01447 02055* 02056* 02073
2307* 02310* 02950 02975* 03079* 03376* 03474 03904*
5067 05998* 06091 06095 06156* 06199 06322 06370
5398* 07266 08070 08083 08722* 08742* 09502 09577
9809 09851
DREN,ABDOMEN IN
1078 01659* 04058 08249* 08822
DREN,ABDOMEN TRAUMA IN
1490* 01602 02996*
DREN,ABSORPTION IN
4000
DREN,ACUTE ABDOMEN IN
0998 01040* 01120 02128* 03050 04113 04654 04982*
DREN,AMEBIASIS IN
8795 08863
DREN,ANUS IN
7225 07245 08290 08291 09108
DREN,APPENDICITIS IN
3970 04113 07253 07566 08280 08307 08312 08334
3358 08806 09414 09901
DREN,BILIARY DISEASE IN
7466*
DREN,BILIARY TRACT IN
0652 01659* 03370* 03629* 03664 03902* 05595* 05644*
5632 07334* 07335* 07524 08507 09524 09617 09793
9795 09800
DREN,BIOPSY IN
9092
DREN,BLEEDING IN
0216 00932* 01271 01363 01926* 03481* 03558 04113
4212 05052 06332* 08308 08729* 08900 09528 09575
9655 09683 09969
DREN,CANCER IN
0293* 01222 01578 04610 05964 07035 07234 08460
9501 09609
DREN,CHOLELITHIASIS IN
1659* 04654 06653 09740
DREN,CIRRHOSIS IN
2631* 02632* 02635* 02657 03049 03462 04563* 04564*
4589 04610 04885 05446* 05595* 05608* 06616 06855*
7334* 07347* 07397 07466* 07469* 07484 07488 08639
8642 09528
DREN,DIARRHEA IN
ALSO CELIAC DISEASE
0225 00938* 01037* 01093 01095 01868* 01919 01938*
1981 02984* 03041 03042 03960 03990 04023* 04035*
4041* 04047* 04070 04092 04104 05008 05293 05985*
5986* 06017 06020 06048 06092 06102 07200 07543*
8227* 08231* 08234* 08240 08245 08247 08248 08249*
8726* 08727* 08731* 08796 08898 08915 09074 09326
9328 09450 09819 09840
DREN,DIGESTION IN
0153 06019 08881
DREN,DYSENTERY IN
1919 01938* 01971 03882* 03987 05888 08788 08796
9962
DREN,ENTERITIS IN
8737*
DREN,ENTEROCOLITIS IN
0938* 01106 01398* 04027* 04059 04367 05838* 05888
5992* 06048 07548* 07549* 08726* 08727* 08728* 08775
8785 08786 09937 09945 09993
DREN,ESOPHAGUS IN
1145 01146 01156 02012* 02017 02027 02907* 03069*
3087 03095 04121 04138 04162 04164 04771* 06112
6137 06970* 06982 06989 07600 07982* 07991* 07995*
8023 09152 09165 09167
DREN,GALLBLADDER IN
1097 03664 04637 04654
DREN,GASTRITIS IN
1106 06048

CHILDREN,GASTROENTERITIS IN
05996* 06088 06092 07543* 07544* 08728* 08884 09989
CHILDREN,GASTROINTESTINAL DISEASES IN
00250 01938* 02378 03049 03288 03948 04028* 04070
04089 04090 04113 04364 05008 05010 05016 05997*
06051 06858* 08784 08848
CHILDREN,GASTROINTESTINAL TRACT IN
00939* 00998 01094 01919 01950 02002 02004 02029
02421 04027* 04055 04077 04121 04691* 04976* 05001
05050 05847* 06019 06032 06047 06056 06106 06107
07111 07566 08731* 08733* 08751 08761 08806 08811
08812 08823 08825 08828 08830 08881 09070 09899
09957 09964 09969
CHILDREN,HEPATITIS IN
03528* 03571 03572 03579 03581 03902* 04563* 04564*
04567* 04572 04574 04591 05446* 05493 06462* 06855*
07347* 07841* 08455 08462 08503 08524 08628 09100
09563 09615 09667 09683 09684
CHILDREN,HERNIA IN
00286 01040* 01907 02128* 04152 04153 05105 05116
05118 06081 06126 07940 08019 08031 08914 09103
09142
CHILDREN,JAUNDICE IN
06479 06484 06488 07058 07524 08227* 08426* 08478
08567 09524 09617
CHILDREN,LARGE INTESTINE IN
00472 00511 01434 01455 02303* 02305* 02313* 02348
02366 04228* 04349 04359 04364 05059 05317* 05318*
05324 05331 05336 06081 06335 06349 06362 07211*
07224 07234 07246 07744* 08249* 08278 08287 08331
08335 08348 08350 08901 09108 09342 09414 09417
CHILDREN,LIVER DISEASES IN
08426* 09538 09615 09670
CHILDREN,LIVER ENZYMES IN
00638 03370* 04847*
CHILDREN,LIVER IN
00088* 00612 00652 00660 01540* 01579 01860* 02505
03370* 03371* 03461 03481* 04013 04058 04435* 04459*
04515 04530 04572 04821 05446* 05456* 05479 05608*
05893 06450* 06471 06488 06552 06938 07334* 07335*
07413 07746* 08455 08491 08497 08503 08733* 08830
09116 09496 09503 09524 09540 09562 09563 09575
09605 09609 09655
CHILDREN,MALABSORPTION IN
00354* 00426* 02286* 02296 04000 04312 04322 05293
05299 05733 06669* 07112 07200 08112* 08227* 08231*
08240 08242 08245 08426* 09337 09339
CHILDREN,MESENTERY IN
08120*
CHILDREN,PANCREAS IN
00558 01490* 02410* 02411* 02421 03311* 03312* 03318
03319 04384* 04389* 04394 05009 06004* 06415 07091
07276* 07292 08378 08385 08393 09433 09473 09483
CHILDREN,PANCREATITIS IN
00575* 03049 03337* 03341* 03342* 04429 04982* 05409
06422* 06427* 08399* 08418 09477 09480 09483 09485
09488
CHILDREN,PARASITIC DISEASES IN
03063 05982* 06072 06332* 06422* 08766 08865 08882
08890 08897 09503
CHILDREN,PEPTIC ULCERS IN
00382* 00387* 01328 01361 01363 02225 03049 04281
06250* 06268 06272 07159 07165 07882 08194 08203
08214 08217 08729* 09301
CHILDREN,PORTAL HYPERTENSION IN
06970* 08473 08729* 09496 09538 09539
CHILDREN,RECTUM IN
00511 01900 04364 05350 05688* 06362 08932 09398
CHILDREN,SALIVARY GLANDS IN
04385*
CHILDREN,SALMONELLOSIS IN
00938* 01052 03990 04026* 04035* 04045* 05019 05991*
07544* 08726* 08736* 08749 08775 08786 09230 09814
09953 09994
CHILDREN,SHIGELLOSIS IN
07544* 08726*
CHILDREN,SMALL INTESTINE IN
01267 01295 01373* 01923* 02114* 02122* 02141 03157
03176 03179 03318 04228* 04234 04243 05009 05178*
05187* 05190 05193 05851* 06081 06232 06669* 07091
07097 07105 07106 07109 07112 07113 07744* 08112*
08115* 08144 08149 08234* 08776 08835 08848 08901
09111 09238 09240 09819 09986
CHILDREN,STOMACH IN
00286 00786* 01097 01210 01213 01222 03120 03127
03129 03139 04180* 04200 04211 04212 04771* 05170
05174 05175 05939 06143* 06175 06187 06695 06739*
06751* 07012* 07035 07058 07059 07070 07074 07600
08031 08048 08104 08203 08214 09272 09301
CHILDREN,TYPHOID FEVER IN
08736* 08787 08875
CHILDREN,ULCERATIVE COLITIS IN
03049 03305 04371* 04379 05371 06388 09422

CHILDREN,ULCERS IN
02219* 03216 04982*
CHILDREN,VIRAL HEPATITIS IN
01613* 01615* 01617* 01622 02601* 02604* 02619 02631*
03049 03527* 03531 03539 03547 03548 03554 03558
03564 03573 03574 03575 03576 03577 03578 03582
04566* 04579 04583 04589 05576* 05577 05584 06556*
06560 06562 06563 06575 06576 06577 06852* 06854*
06869* 07279* 07442* 07444* 07448* 07452* 07459 07462
07484 07493 08463 08524 08593* 08594* 08597* 08598*
08599* 08600* 08602* 08604* 08605 08606 08607 08609
08610 08616 [illegible] [illegible] 08630 09540 09664 09670
09676 09677 09679 [illegible] 09695 09697 09700
CHILDREN,VISITING IN
07902
CHLORIDE ABSORPTION
01412 02753* 02758* [illegible] [illegible] 04674* 06743* 08227*
CHLORIDE METABOLISM
02817 06208 07747*
CHLORIDE SECRETION,PANCREAS
02797* [illegible]
CHOLANGIOGRAPHY
00108* 00138 00159 [illegible] 00751* 00752* 00754* 00777
00778 00779 00924* [illegible] [illegible] 00962 00977 00981
00996 [illegible] 01651* 01652* 01655* 01657* 01660* 01662*
01681 01682 01686* 01891 [illegible] 01903 02677 02925
02945 02962 02964 02976* 03626* 03631* 03655 03885*
03926 [illegible] [illegible] 04001 04017 04018 04237 04410*
04415 04484 04551 04625* 04660 04665 04842* 04856*
04861* 04864* 04965 04966 05633* 05636* 05650* 05662
05872* 05880* 05901 [illegible] 05941 05955 06437 06621*
06647 06652 06674* 06853* 06878* 06937 06947 06962
07265 07503 07525 07526 [illegible] 07841* 07878 07879
07886 [illegible] 07932 07938 07957 07973 07974 07978
07979 [illegible] [illegible] [illegible] 08507 08519 08662* 08667*
09052 09055 [illegible] 09067 09071 09090 09500 09542
09659 09730 09741 09753 09758 09771 09782
CHOLANGITIS
04488 06387 07445* 08429* 08470 08656* 08666* 08670
08690 08701 08705 [illegible] 09500 09802
CHOLECYSTECTOMY
SEE ALSO GALLBLADDER SURGERY
00756* 00757* 00758* [illegible] 01097 01653* 01655* 01658*
01812* 02668* 02675 02680 02919* 02976* 03332* 03624*
03626* 03627* 03628* 03630* 03633* 03637 03643 04017
04409* 04410* 04413* 04632* 04638 04640 04644 04647
04651 04663 04664 04665 04666 05035 05423* 05633*
05634* 05635* 05637* 05638* 05647* 05649* 05650* 05651
05653 05654 05655 05657 05663 [illegible] 06432 06483
06532* 06599 06631* 06634 06636 06640 06641 06644
06647 06650 06652 06654 06662 06663 06665 06879*
07357 07426 07491* 07496* 07497* 07498* 07510 07511
07522 07532 07534 07537 07538 07657* 07878 08058
08384 08530 08655* 08657* 08660* 08663* 08672 08676
08681 08686 08692 08702 08703 08705 08707 08777
08919 09221 [illegible] 09618 09740 09746 09757 09762
09770 09778 09781 [illegible] 09787 09788 09790 09791
CHOLECYSTITIS
00762 00781 [illegible] 01112 01647* 01648* 01649* 01650*
01651* 01652* 01658* 01667 01668 01671 01672 01683
02460 02668* 02669* 02691 02884* 02976* 03047 03356*
03475 03626* 03627* 03630* 03636 03649 03658 03663
04017 04018 04299 04627* 04637 04638 04642 04654
04662 04663 04664 04972* 04992 05475 05634* 05637*
05638* 05645* 05653 05655 05656 05665 06069 06103
06507 06529 06619* 06621* 06628* 06630* 06636 06640
06643 06657 06664 06858* 06872* 06955 07000 07489*
07490* 07493* 07499* 07511 07522 07523 07526 07529
07535 07561 07904 07957 07973 08657* 08659* 08660*
08663* 08670 08671 08673 08674 08680 08688 08690
08693 08694 08697 08706 08712 08714 08725* 08777
08837 09081 09297 09500 09756 09760 09762 09765
09771 09773 09788 09790 09791 09793 09794 09798
09800 09804 09807 09867 09905
CHOLECYSTITIS DIAGNOSIS
06879*
CHOLECYSTITIS ETIOLOGY
07528
CHOLECYSTITIS TREATMENT
07303* 07361 07362 07528
CHOLECYSTITIS,DISEASES ASSOCIATED WITH
08166*
CHOLECYSTOGRAPHY
00954* 01881 02925 02944 04861* 04884 05842* 05917
05922 06628* 06879* 06933 07265 07824 07938 07974
08468 08668 08687 08943 09034 09037 09055 09192
09791
CHOLECYSTOKININ
00877 00878* 01699* 04085 04557* 04753 04776* 05782*
05786* 05796* 05938 06957 08045*
CHOLELITHIASIS
00187* 00750* 00751* 00753* 00757* 00761 00763 00772
01022* 01433 01587 01655* 01657* 01661* 01662* 01663*
01666* 01668 01674 01675 01676 01692 02236 02468
02678 02686 02693 02695 02884* 02981* 03332* 03356*
03647 03653 03658 04085 04264* 04265* 04390* 04392*

CHOLELITHIASIS
CONTINUED
04415 04420 04430 04466 04484 04627* 04628* 04629*
04632* 04635* 04660 05423* 05640* 05863* 06426* 06428*
06532* 06619* 06622* 06630* 06668* 07311 07337* 07453*
07490* 07496* 07507 07518 07540 07836* 07973 07978
08375* 08429* 08486 08522 08655* 08668 08680 08686
08687 08688 08706 08709 08710 08712 08715 08725*
08803 08991 09072 09244 09474 09542 09763 09770
09772 09776 09790 09791 09800 09805 09866
CHOLELITHIASIS COMPLICATIONS
09746 09807
CHOLELITHIASIS DIAGNOSIS
00753* 00781 00924* 00930* 01647* 01648* 01852* 01861*
01863* 01881 01901 02960 03624* 03632* 03651 03893*
03894* 03962 03964 04012 04016 04017 04018 04655
04667 04842* 04851* 04873 04888 05636* 05665 05860*
05862* 06625* 06631* 06643 06651 06653 06879* 06880*
06892 06923 07265 07402 07491* 07517 07524 07525
07899 07904 07914 08519 08682 08690 09034 09495
09740 09741 09798
CHOLELITHIASIS EPIDEMIOLOGY
01055 02010* 02011* 02201* 03047 03638 03648 04634*
04636 04647 04651 04655 05635* 05641* 06512 06640
07512 08166*
CHOLELITHIASIS ETIOLOGY
00098 00753* 00755* 00763 00772 01653* 01663* 01672
01673 01688 02659* 02660* 02661* 02662* 02663* 02670
03117* 03638 03640 03644 04237 04493 04634* 04636
04647 04648 05152 05153 05632* 05639* 05645* 05652
06463* 06623* 06631* 06638 07489* 07502* 07512 07525
07526 08664* 08665* 08677 08696 09740 09748 09750
09766
CHOLELITHIASIS IN CHILDREN
01659* 04654 06653 09740
CHOLELITHIASIS PATHOLOGY
00591 01650* 04649 04655 05643* 05645* 06463* 06623*
06651 07512 07526 07580 09495 09806
CHOLELITHIASIS SURGERY
00753* 00775 01028* 01651* 01658* 02668* 02670 03627*
03628* 03638 04639 04647 04667 05634* 05636* 05638*
05647* 05649* 05651 05657 05659 05665 06628* 06633
06635 06650 06652 06656 06661 06662 06663 07491*
07511 07522 07525 07531 07534 08519 08661* 08672
08685 08693 08695 08696 08699 08702 09220 09221
09495 09741 09752 09757 09763 09769 09780
CHOLELITHIASIS TREATMENT
04658 04664 05647* 05659 06631* 07493* 07506 07517
07523 07530 07538 08058 08660* 08663* 08676 08679
08682 08699 09740 09745 09757
CHOLELITHIASIS,AGE FACTORS IN
05635* 07506
CHOLELITHIASIS,DISEASES ASSOCIATED WITH
00299* 00764 00954* 01249* 01514* 01575 01652* 01654*
01667 01670 02140 02201* 02450 02664* 02670 02981*
03336* 03475 03627* 03647 04235 04472 04636 04641
04655 04656 04659 04661 04662 04666 04667 05014
05473 05635* 06432 06463* 06627* 06631* 06639 06658
07102 07304* 07325 07327 07346* 07499* 07501* 07505
07506 07509 07521 07526 07537 07580 07843* 08166*
08388 08656* 08660* 08663* 08669 08674 08675 08682
08683 08684 08690 08700 08712 09220 09221 09449
09478 09759 09761 09788 09798 09804
CHOLERA
00022* 01734* 02694 02908* 02999* 03913* 03946 04035*
04863* 04981* 05185* 06214* 07575 09975
CHOLERETIC AGENTS
06626* 06799* 09540
CHOLESTASIS
00628 00670 00753* 00768 00770 00774 00782 00851*
01539* 01555 01568 01577 01650* 01656* 01666* 01678
01852* 01863* 02472* 02492 02511 02588 02603* 02604*
02627* 02628* 02648 02704* 02884* 03117* 03356* 03597*
03629* 03661 03662 03665 03666 03914* 03975 04009
04012 04299 04450* 04462 04463 04465 04471 04472
04484 04486 04488 04493 04496 04507 04515 04527*
04531 04538 04543 04587 04619 04633* 04634* 04663
04813 04886 04904 05152 05153 05365 05423* 05473
05487 05546* 05645* 05837* 05979 06451* 06461* 06465*
06466* 06481 06525 06530 06544 06923 06928 06931
06932 06939 07337* 07346* 07348* 07376 07381 07382
07385 07386 07399 07417 07418* 07425 07426 07440
07445* 07449* 07453* 07454* 07499* 07516 07517 07518
07520 07523 07701* 07754 07840 08051 08432* 08436*
08464 08474 08507 08515 08516 08522 08564 08586
08588 08640 08649 09492 09498 09558 09748 09750
09766
CHOLESTEROL
SEE ALSO HORMONAL CONTROL OF GALLBLADDER
SEE ALSO LIVER METABOLISM
00208 00595* 00753* 00755* 01562 01653* 01663* 01672
02661* 02662* 02673 02679 02681 02704* 02711 02873
03648 03826 03881 04294 04458* 04629* 04635* 04648
04649 04651 04800* 04859* 05254 05632* 05641* 05652
05814* 06003* 06007* 06623* 06638 06648 06802 07502*
07654 07792 07793 08515 08668 08684 08686 08687

STEROL
TINUED
697 08991 09012 09766 09828
STEROL ABSORPTION
2757* 03741 03817* 03886* 04722* 04728* 06698* 06813
815 07439 07492* 07811* 08133 08306 08688 08854
STEROL METABOLISM
827* 07791 07811* 08688
STEROL METABOLISM,LIVER
037 00208 00595* 00665 00687 00755* 00911 01637*
063* 02528 02601* 02838* 02860 02868 03721* 03809*
3817* 03826 03860 04508 04682 04803* 04816 04824
825 05432* 05805* 05808* 05815* 06536* 06783* 06813
7494* 07673 07725* 07756 07764 07780 07785 07821
617 08980 09805
STEROL SYNTHESIS
687 02860 02868 04803* 04824 04825 04827 04828
813 07083* 07673 07810* 07816* 07821 08980
STEROL,SERUM
7083* 07810*
NERGIC AGENTS
0070* 00186* 03711* 06146* 07682*
IC HEPATITIS
0020 00634 00635 00639 00641 00642 00659 00697*
0702 00709 00710 00931* 01543* 01571 01612* 01619*
2597* 02600* 02613 02618 03356* 03358* 03378* 03383*
3389 03424 03503 03555 03621 03976 04478 04511
553 04568* 04570* 04571 04588 04591 04592 04593
5433* 05434* 05444* 05519 05570* 05580 05584 05602*
469* 06470 06473 06491 06494 06497 06500 06501
503 06528 06570 06571 06577 06787* 06855* 06868*
6901 06947 07134* 07354 07362 07365 07370 07391
7404 07408 07411 07416 07450* 07451* 07453* 07778
7889 07891 07913 07914 08424* 08442* 08449 08463
3483 08485 08502 08513 08523 08530 08541* 08603*
3611 08613 09120 09633 09663 09686 09692 09696
9867
IC PANCREATITIS
0574* 00581 00587 01483* 01484* 01486* 01491* 01513*
1515* 01524 01526 02449 02449 02452 02461 02463
2468 03316* 03330* 03333* 03628* 03766* 03950 03952
3953 03981 04390* 04396 04409* 04413* 04414 04415
416 04418 04420 04426 04878 05179* 05241 05398
5400* 05402* 05407 05408 05413 05416 05418 05419
5420 05421 05654 05854* 05880* 05971 05977 06414
5421* 06425* 06426* 06428* 06432 06433 06435 06438
6439 06440 06442 06871* 06872* 06880* 07036 07285
7286 07299* 07300* 07302* 07303* 07304* 07305* 07309*
7313 07314 07318 07321 07325 07326 07328 07838*
7958 08375* 08381 08397* 08398* 08403 08406 08411
3413 08421 09470 09472 09475 09476 09482 09485
9486 09756 09845
NIC PANCREATITIS DIAGNOSIS
3608
NIC VIRAL HEPATITIS
2604* 04443* 04444* 05571* 05585 05587 06558* 07454*
9573 09681 09687
OTRYPSIN SECRETION,PANCREAS
0058 00119* 00804 00805 00836* 00860 01765* 01771*
2804 04389* 04412* 07838* 07937
ULATION IN CIRRHOSIS
0733 00744 00747 01635* 01845 02484* 02638* 03599*
3607 03622 04597* 04846* 05611* 06578* 06581* 07481
3459 08654 09547 09705 09708 09735
ULATION IN HEPATITIS
0020 02511 05611* 06459* 09574 09682 09683
ULATION IN PANCREATITIS
2413* 02444* 04760 06421* 06992
ULATION IN PEPTIC ULCERS
0396*
ULATION IN ULCERATIVE COLITIS
0535 05373 07266
ULATION STUDY TECHNIQUES
1239 01590 01591 04899 04935 05667* 05668* 06482
ULATION STUDY TECHNIQUES,GASTROINTESTINAL
0798 00799 00897 00903 01977 01984 01989 01990
1994 02036 02112 02121* 02315* 02353 02510 02514
3903* 09036
ULATION,ABDOMEN
9027
ULATION,ANUS
8345 08920
ULATION,BILIARY TRACT
0759* 00916 02676 06892 07532 08999
ULATION,ESOPHAGUS
1934* 05084* 05164 06724* 09133 09708
ULATION,GALLBLADDER
1669 02768 04642 09790
ULATION,GASTROINTESTINAL
0213 00221 00522 00799 00830 00973 01132 01252*
1927* 02130* 02188 02317* 02319* 02514 02523 02698*
2861 02997* 03011 03251* 04067 04069 04118 04353
4460* 04681 04738 04935 04995 05040 05063 05066
5067 05304 05667* 05670* 05844* 06023 06070 06666*
6702* 06817 06835 06886 06891 06896 06899 07550*
7553 07554 07555 07556 07831 07887 08728* 08740*
CIRCULATION,GASTROINTESTINAL
CONTINUED
08792 08817 08831 08850 08853 09091
CIRCULATION,LARGE INTESTINE
00798 02298* 02319* 02327* 03263* 04353 04373* 05029
05315* 05317* 05337 05355 05358 05360* 06386 06887
07254 07557 08301 09133
CIRCULATION,LIVER
00090* 00264* 00607* 00647 00897 00902 00903 00916
00919 01358 01536* 01538* 01549* 01550* 01551* 01565
01574 01600* 01845 01877* 02476* 02480* 02510 02514
02517 02531 02542 02558 02574 02575 02645 02721
02821 02861 03360* 03365* 03367* 03403 03412 03444
03445 03460 03465 03481* 03486* 03591* 03593* 03599*
03903* 03936 03939 04010 04367 04435* 04448* 04449*
04454* 04460* 04465 04466 04467 04472 04477 04481
04487 04490 04497 04498 04526* 04584 04586 04597*
04603* 04611 04621 04623 04694* 04700 04701 04705
04791* 04799* 04846* 04848* 04899 04926 04935 04938
04953 04954 05003 05063 05423* 05424* 05427* 05429*
05445* 05451* 05455* 05458 05470 05481 05482 05488
05490 05503 05514 05516 05518 05519 05520 05521
05528* 05536* 05540* 05556 05559 05562 05611* 05619
05620 05671* 05844* 05848* 05890 05898 05899 05906
05916 06454* 06457* 06472 06474 06475 06476 06478
06482 06489 06504 06514 06518 06539 06545 06571
06592 06594 06596 06599 06601 06605 06654 06666*
06679* 06682 06683 06798* 06814 06864* 06884 06885
06893 06894 06948 06960 06965 07331* 07332* 07333*
07344* 07352 07356 07357 07358 07359 07360 07366
07375 07384 07388 07403 07412 07423* 07429 07470*
07472 07473 07474 07475 07481 07550* 07598* 07615
07706* 07722* 07724* 07730* 07748* 07762 07763 07822
07828 07844* 07877 07889 07897 07908 07949 07955
07969 08423* 08429* 08437* 08443* 08452 08454 08459
08471 08472 08473 08480 08484 08488 08489 08492
08496 08504 08510 08530 08532 08534 08535 08536
08551* 08567 08632* 08647 08719* 08923 09026 09028
09053 09059 09494 09497 09505 09507 09512 09513
09514 09517 09521 09523 09530 09535 09538 09539
09548 09550 09552 09565 09574 09577 09592 09596
09599 09606 09702 09703 09720 09730 09733 09790
09828
CIRCULATION,MESENTERY
00791 00792 00818* 00976 01276 01277 02640 02973*
02997* 03148* 03167 03171 03936 04069 04252 04353
05562 05670* 05844* 06666* 06681 06834 06888 07024*
07115 07264* 07550* 07555 07606 07922 08130 08717*
08738* 08867 09091 09828 09857
CIRCULATION,PANCREAS
00538* 01505 02413* 04392* 04692* 04760 04762 05063
05383* 05388 05401* 06070 06420* 06681 06684 06771*
07279* 07280* 07296 07324 07550* 07865*
CIRCULATION,PORTAL
01528* 02721 02906* 03360* 03403 03413 03481* 04456*
04467 04526* 04799* 04846* 04848* 04953 05451* 05511
05520 05848* 05869* 05881* 05899 05966 06421* 06450*
06454* 06457* 06459* 06474 06482 06503 06504 06518
06578* 06592 06594 06705* 06864* 06948 06992 07331*
07340* 07347* 07353 07375 07384 07396 07403 07405
07406 07407 07412 07481 07486 07487 07499* 07722*
07762 07822 07860* 07922 07955 08458 08535 08536
08633* 08635* 09038 09059 09507 09519 09523 09552
09613 09658 09705 09708 09828
CIRCULATION,RECTUM
01704* 03257* 04353 06341 06888 08345 08920
CIRCULATION,SALIVARY GLAND
03759*
CIRCULATION,SMALL INTESTINE
00007* 00008* 00359 00830 01248* 01270 01375 01518
01878* 02121* 02137 02179 02192 02698* 02702* 02776
02888* 03148* 03167 03670* 03671* 03680 03710* 03908*
04240 04245 04252 04695* 04782 04783 04849* 05029
05190 05196 05201 06890 06897 06898 06977 07024*
07084* 07088 07089 07090 07204 07557 07592* 07648
08118* 08717* 09036 09232 09236
CIRCULATION,STOMACH
00004* 00808 00861 01175* 01704* 01711* 01747* 01762*
01952 02045* 02111 02112 02709 02779* 02798* 02800*
02892* 03693 04099 04192 04218 05136* 05161 05223*
05667* 05668* 05679 07126* 07127* 07130* 07167 07900
07912 07997 08033*
CIRRHOSIS
SEE ALSO ASCITES
SEE ALSO ESOPHAGUS VARICES
SEE ALSO LIVER COMA
SEE ALSO PORTAL HYPERTENSION
00370 00648 00749 00870 00926* 01530* 01568 01612*
02512 02513 02525 02539 02648 02650 02651 02655
02884* 02997* 03356* 03357* 03383* 03408 03424 03435
03451 03458 03485* 03486* 03549 03600* 03604 03605
03624* 03903* 04063 04593 04602* 04617 04886 05000
05433* 05434* 05440* 05453* 05481 05491 05511 05519
05520 05540* 05602* 05631 06220* 06428* 06557* 07085*
07349* 07778 07957 08463 08476 08486 08500 08505
08513 08530 08553* 08583 08650 08651 08653 08862

CIRRHOSIS
 CONTINUED
   08995 09048 09499 09538 09546 09554 09573 09576
   09603 09613 09647 09657 09686 09713 09731 09732
   09788
CIRRHOSIS COMPLICATIONS
   08638*
CIRRHOSIS DIAGNOSIS
   00107* 00110* 00113* 00677 00678 00941* 00987 00992
   01533* 01543* 01637* 01639* 01852* 01860* 01861* 01862*
   01863* 01865 01917 01951 02472* 02474* 02487 02626*
   02629* 02635* 02647 02653 02890* 02922 03358* 03364*
   03521* 03534 03555* 03597* 03601* 03608 03609 03610
   03611 03612 03615 03616 03618 03893* 03894* 03934
   03938 03942 03954 03962 03963 03976 04005 04008
   04011 04012 [illegible] 04478 04596* 04609 04611 04637*
   04841* 04844* 04845* 04885 04902 04934 04953 04954
   04955 04963 04964 05472 05599* 05622 05625 05836*
   05848* 05865* 05891 05892 05916 05919 05925 05927
   06485 06510 06584 06585 06609 06840* 06855* 06893
   [illegible] [illegible] 06923 06932 06939 06947 06959 06966
   07354 07391 07464* 07837* 07842* 07864* 07877 07889
   07890 07893 07899 07904 07913 07942 07965 08427*
   08525 08653 09047 09081 09120 09125 09500 [illegible]
   09720 09721
CIRRHOSIS DIAGNOSIS,RADIOLOGY IN
   01887 01889 02889* 03611* 06600
CIRRHOSIS EPIDEMIOLOGY
   01055 02010* 02633* 02639 02641 03598* 03619 04588
   04589 04609 05422* 05462 05610* 05614 05615 06261*
   06512 07482 08428* 08651 09721 09734
CIRRHOSIS ETIOLOGY
   00205 00613* 00651 00672 00697* 00738 01536* 01552*
   01575 01576 01588 01615* 01634* 01641* 02471* 02579*
   02627* 02628* 02630* 02631* 02632* 02633* 02634* 02639
   02641 02653 02654 03389 03462 03620 04117 [illegible]
   04479 04480 04515 04531 04563* 04564* 04589 04596
   04609 04610 04615 04616 04643 05064 05446* 05448*
   05449* 05452* 05473 05501 05531* 05532* [illegible] [illegible]
   05595* 05596* 05600* 05608* 05614 05623 06033 06261*
   06452* 06469* 06477 06490 06494 06518 06522 [illegible]
   06579* 06584 06589 06590 06591 06603 07397 07423*
   07432 07434 07438 07451* 07464* 07474 07482 07484
   08502 08613 08651 09547 09635 09696 09704 09721
   09729 09730 09734
CIRRHOSIS IMMUNOLOGY
   04021 06498 06593 06602 07354 07415 07450* 07479
   07891
CIRRHOSIS IN CHILDREN
   02631* 02632* 02635* 02657 03049 03462 04563* 04564*
   04589 04610 04885 05446* 05595* 05608* 06616 06855*
   07334* 07347* 07397 07466* 07469* 07484 07488 08639
   08642 09528
CIRRHOSIS PATHOLOGY
   00682* 00686* 00741 00744 00748 01635* 01636* 01637*
   01644 02471* 02626* 02631* 02634* 02635* 02642 02654
   03374* 03387* 03414 03503 03593* 03594* 03596* 03598*
   03599* 03607 03616 03620 04482 04483 04529* 04596*
   04597* 04601* 04609 04610 04611 04612 04614 05488
   05492 05548* 05600* 05607* 05611* 05615 05622 05623
   06461* 06515 06522 06533* 06581* 06584 06585 06591
   06592 06600 06614 07354 07360 07469* 07474 07475
   07476 07477 07482 08449 08631* 08643 08651 08654
   09541 09711 09714 09716 09717 09722
CIRRHOSIS TREATMENT
   00729* 00730* 00735 00736 00748 00773 01531* 01596
   01634* 01636* 01643 01857* 02483* 02484* 02627* 02631*
   02638* 02639 02652 02653 03415 03416 03418 03529*
   03600* 03614 03616 03617 04014 04015 04468 04494
   04517* 04595* 04598* 04600* 04605* 04606* 04608* 04609
   04612 04615 04616 04618 05461 05489 05513 05598*
   05605* 05613 05619 05620 05624 05625 05627 05628
   05630 05869* 06453* 06491 06495 06517 06580* 06582*
   06585 06586 06602 06606 06613 07340* 07341* 07347*
   07404 07466* 07470* 07471* 07472 07478 07479 07481
   07485 07730* 07984* 08432* 08472 08483 08485 08541*
   08632* 08641 08652 08654 08820 09060 09500 09547
   09561 09588 09701 09710 09712 09722 09723 09729
   09730 09736 09738
CIRRHOSIS,AGE FACTORS IN
   02673 04609 05595* 05614 08428* 09729
CIRRHOSIS,ALCOHOL IN
   00686* 00745 01576 02579* 02626* 02652 03486* 03591*
   03619 03621 04609 05531* 05613 05614 05615 06579*
   06585 06591 08589 09721 09725
CIRRHOSIS,ANEMIA IN
   00738 01643 01644 02631* 02632* 03589* 03591* 03599*
   05629 07464*
CIRRHOSIS,ASCITES IN
   00740 04137 04598* 04603* 04606* 04609 04865 05607*
   05609* 05612* 05627 06579* 06580* 06585 06611 06613
   07344* 07465* 07471* 07472 07474 07475 07478 08428*
   08500 08634* 08645 08652 09701 09712 09714 09718
   09722 09723 09726 09736 09737 09738
CIRRHOSIS,BILIARY TRACT
   00773 01493* 01553 01636* 01637* 01693 02472* 02627*
   02628* 02648 02673 02976* 03464 03597* 03631* 04111
   04471 04482 04483 04511 04512 04588 04610 04615
   04631* 04643 04841* 05473 05602* 05615 05621 05626
   06461* 06590 06643 07334* 07337* 07387 07405 07488
   08387 08430* 08431* 08462 08463 08640 08646 08698
   09512 09591 09730
CIRRHOSIS,BIOCHEMICAL DIAGNOSIS OF
   00107* 00599* 00739 00931* 03594* 03606 03609 05881*
   09716
CIRRHOSIS,BLOOD COAGULATION IN
   03611 04504 04614 04885 06612 06615 08639 08643
   09715
CIRRHOSIS,CIRCULATION IN
   00733 00744 00747 01635* 01845 02484* 02638* 03599*
   03607 03622 04597* 04846* 05611* 06578* 06581* 07481
   08459 08654 09547 09705 09708 09735
CIRRHOSIS,CLINICAL STUDIES OF
   00733 00737 00742 00743 00744 00746 00748 01590
   01636* 01906 02473* 02628* 02638* 08653
CIRRHOSIS,DISEASES ASSOCIATED WITH
   00600* 00699 00734 00741 00743 00747 01034* 01035*
   01036* 01537* 01571 01642* 02473* 02640 02642 02645
   02646 02658 02994* 03404 03475 03504 03603 03607
   03617 04015 04034* 04442* 04445* 04455* 04464 04475
   04492 04504 04604* 04609 04610 04612 04613 04614
   04618 04620 04621 04622 04623 04916 05068 05069
   [illegible] 05260 05452* 05496 05521 05522 05592* 05593*
   05595* 05596* 05597* 05603* 05609* 05618 05621 05623
   05625 05626 05628 05629 05887 06475 06508 06527
   06528 06579* 06583* 06588 06593 06596 06597 06598
   06601 06603 06604 06605 06607 06608 06610 06611
   06612 06614 06615 06616 06848* 06948 06968* 06973*
   [illegible] [illegible] 07343* 07344* 07406 07407 07416 07423*
   07451* 07464* 07465* 07466* 07470* 07473 07476 07480
   07483 07485 07486 07487 07488 07558 07858* 08422*
   08428* 08521 08635* 08640 08644 08645 08646 08647
   08648 09497 09547 09611 09703 09706 09707 09708
   09711 09714 09718 09724 09726 09727 09728 09730
CIRRHOSIS,ELECTROLYTE METABOLISM IN
   [illegible] [illegible] 04600* 05604* 05613 05881* 06580* 07471*
   07478 08634*
CIRRHOSIS,EXPERIMENTAL STUDIES OF
   00729* 00730* 00731* 00732* 01633* 01720* 02579* 02656
   07433 09589 09704
CIRRHOSIS,HORMONAL CONTROL IN
   [illegible] 02615* 03424
CIRRHOSIS,IMMUNOLOGY IN
   00739 02647 03534 03608 03610 03621 03963 04443*
   04482 04483 04511 04512 04609 04963 05606* 05621
   07913
CIRRHOSIS,METABOLIC CHANGES IN
   00096 00732* 00745 00773 02490 02629* 02636* 02751*
   03613 03481 [illegible] 04434* 04495 04604* 04607* 04619
   04719* 05444* 05480 05592* 05593* 05594* 05599* 05601*
   05604* 05609* 05616 05881* 06595 06787* 07337* 07467*
   07722* 07761 07862* 08475 08483 08485 08631* 08636*
   08637* 08649 08991 09491 09709 09716 09722 09725
   09867
CIRRHOSIS,MICROORGANISMS IN
   00741 08646
CIRRHOSIS,WATER METABOLISM IN
   02512 02658 03617 05609* 05613 08634*
COBALT METABOLISM
   06791* 09861
COLITIS
 SEE ALSO ENTERITIS
 SEE ALSO ENTEROCOLITIS
 SEE ALSO GASTROENTERITIS
 SEE ALSO REGIONAL ENTERITIS
   00513 00936* 00946* 01415 01416 01441 01932* 02308*
   02338 02353 02361 02365 02375 02377 02894* 02957
   03019 03251* 03275 03300* 04970* 05334 05358 05379
   06316* 07222 09416 09995
COLITIS COMPLICATIONS,ULCERATIVE
   00186* 00535 01466* 01467* 01471* 02390* 02395* 03295*
   03296* 03297* 03300* 03301* 03303 04060 04370* 04376
   04378 05357 05364 05365 05366 05368 05373 05375
   05376 05379 06326* 06391 08254* 08288 08359* 08368
   08369 09426 09429
COLITIS DIAGNOSIS,BIOPSY IN ULCERATIVE
   01865* 04375* 06396 09423
COLITIS DIAGNOSIS,RADIOLOGY IN ULCERATIVE
   00532* 01013 01464* 01472* 02382* 02383* 03888* 04370*
   04375* 09423
COLITIS DIAGNOSIS,ULCERATIVE
   00149 00532* 00536 01453 01472* 01474 02381* 02387*
   02391* 03295* 03304 03305 03997 03999 04356 04929
   05366 05367 05374 05376 05378 06042 06378* 06383
   06384 06388 06394 06395 06880* 06887 06921 07263*
   07265 07267 07270 07895 08359* 08361 08363 08366
   09083 09421 09422 09423 09424 09426 09429

ITIS EPIDEMIOLOGY,ULCERATIVE
01455 01461 01925* 02385* [illegible] 05371 05375 [illegible]
06383 06388 08224 09424
ITIS ETIOLOGY,ULCERATIVE
00533 00537 01169* 01367* [illegible] 01468* 01470* 01477
02381* 02384* 02386* 02389* [illegible] 03307 04369* 04379
05360 05360* 05363* 05370 05371 05374 [illegible] 05378
06011 06360 06376* 06377* 06379* 06381* 06384 06386
06390 06394 06395 [illegible] [illegible] [illegible] 07261* 07262*
07264* [illegible] 07271 [illegible] [illegible] 08364 08367 09423
09424 09425 09426 09427
ITIS IMMUNOLOGY,ULCERATIVE
06376* 06377* 06381* [illegible] [illegible] 06394 [illegible] [illegible]
07261* 07263* 07264* 09335
ITIS IN CHILDREN,ULCERATIVE
[illegible] 03305 04371* [illegible] 05371 [illegible] 09422
ITIS PATHOLOGY,ULCERATIVE
00456 00532* 00534 [illegible] 01471* 01473* 01474 01478
[illegible] [illegible] [illegible] [illegible] [illegible] 02394* 02406 03301*
03304 03307 03888* [illegible] [illegible] 05361* 05362* 05367
05373 05376 06011 06326* 06366 06376* 06378* 06389
06390 06392 06396 06882* 07267 07269 [illegible] 07557
07895 08263* 08359* 08361 08363 08364 08365 08366
08368 09422 09423 09425 09427 09428 [illegible]
ITIS SURGERY,ULCERATIVE
01379* 01468* 01471* 01472* 01475 01480 01482 02387*
02396* 02397 02399 [illegible] 03297* 04370* 04371* 04375*
04379 05343 05374 05377 05379 [illegible] 06385 06388
06393 06395 07267 07273 08254* 08361 08362 08371
09421
ITIS TREATMENT,ULCERATIVE
00536 01024* 01367* 01464* 01468* 01469* 01476 01477
01925* 02276* 02381* 02386* 02392* 02393* 02395* 02396*
02397 02399 02401 02405 02894* 02976* 03294* 03299*
03305 03306 03308 04370* 04371* 04372* 04373* 04375*
04376 04377 04379 05364 05369 05372 05374 05376
05377 05378 05379 05380 06314* 06360 06377* 06382*
06384 06385 06388 06391 06392 06393 06395 07272
07273 08362 08367 08371 08820 09344 09354 [illegible]
09424 09426 09427 09880
ITIS,AGE FACTORS IN ULCERATIVE
09421
ITIS,BIOCHEMICAL DIAGNOSIS OF ULCERATIVE
06380*
ITIS,CIRCULATION IN ULCERATIVE
00535 05373 07266
ITIS,CLINICAL STUDIES OF ULCERATIVE
00531* 00532* 00536 01469* 01481 02396*
ITIS,DISEASES ASSOCIATED WITH ULCERATIVE
00531* 00535 01466* 01481 01482 01925* 02385* 02390*
02391* 02392* 02393* 02396* 02976* 03295* 03296* 03297*
03300* 03302 04373* 04374* 05361* 05365 05366 05375
06387 06391 07238 07265 08224 08359* 09354 [illegible]
09430
ITIS,DRUG TREATMENT OF ULCERATIVE
00946* 01464* 01469* 01471* 02386* 02397 03298* 04370*
04371* 04372* 04376 04377 04379 05369 05372 05374
05380 06382*
ITIS,GRANULOMATOUS
E CROHNS DISEASE OF COLON
ITIS,IMMUNOLOGY IN ULCERATIVE
00533 00537 01406* 01465* 01470* 01471* 01871* 02389*
02398 02405 04379 05363* 05370 09425
ITIS,METABOLIC CHANGES IN ULCERATIVE
00531* 02365 02391* 05368 09429
ITIS,MICROORGANISMS IN ULCERATIVE
01367* 01466* 02391* 02398 05363* 06377* 06381* 07262*
09423
ITIS,ULCERATIVE
00497 00526 00773 01104 01115 01440 01470* 01479
01871* 02281* 02290 02339 02400 02402 02403 02404
02901* 03253* 03265* 03300* 03305 03306 03307 03308
04111 04361 05342 05342 06021 07183* 07266 07268
08246 08250* 08289 08313 08339 08758 08909 09082
09982
LAGEN DISEASES
05045 06087
ON
E ALSO LARGE INTESTINE
02778 02894* 02895* 02901* 02935 03008 03253* 03257*
03258* 03270 03272 03287 03290 09404 09409
ON DIVERTICULA
03274
ON,CROHNS DISEASE OF
00442* 00456 00521 00526 01368 01436 01458 01460
01466* 01470* 01474 01479 01969 02275* 02276* 02299*
02301* 02329* 02330* 02339 02341 02366 02385* 02387*
ON,IRRITABLE
01402* 01805* 01865* 02893* 02894* 03016 03038* 05073
05354 06031 06101 06348 06882* 08328 09381 09393
09396 09397 09950
OSTOMY
00527 03018 06355 08265* 08272 08291 08292 08311
COMA,HEPATIC
SEE [illegible]
COMA,LIVER
00206 00597* 00608* 00676 01537* 01596 01621 01622
01636* 01638* 01642* 02473* 02486* 02508 02509 02517
02535 02552 02554 02571 02599* 02604* 02665* 03386
[illegible] [illegible] 03408 03410 03416 03472 03475 03520*
03523* 03539 03602 03612 03616 04015 04064 04198
04437* 04491 04530 04546 04560* 04598* 04599* 04605*
[illegible] 04620 04622 04845* 04857* 05489 05534* 05592*
05602* [illegible] 05605* 05607* 05617 05618 05619 06449*
06453* 06475 06476 06488 06491 06509 06515 06524
06565 [illegible] 06605 06607 06613 07344* 07347* 07355
07485 07837* 07840 08438* 08441* 08455 08461 08495
08638* 08645 09505 09522 09530 09532 09553 09561
09568 09574 09604 09674 09710 09719
[illegible]
SE[illegible] BILIARY TRACT
00015* 00051* 00368 [illegible] 00757* [illegible] 00760 00776
00777 00839* 00924* 00962 01539* 01587 01647* 01648*
01651* 01654* 01656* 01657* 01658* 01660* 01670 [illegible]
01687 01690 01691 01692 01792 01852* 01975 02438
02583 02659* 02668* 02696 02704* 03628* 03629* 03630*
03637 03651 03654 03655 03659 04007 04017 04018
04033* 04102 04237 04409* 04410* 04413* 04472 04626*
04631* 04639 04640 04644 04658 04663 04664 04778*
04813 05423* 05436* 05633* 05634* 05636* 05638* 05642*
05646* 05647* 05650* 05651 05659 05662 05862* 06432
[illegible] 06477 06618* 06624* 06629* 06633 06644 06645
06647 06652 06654 06656 06660 06661 06662 06663
06665 06668* 06674* 06720* 06721* 06734* 06879* 06914
07294 07339* 07346* 07356 07357 07358 07366 07385
07426 07449* 07491* 07498* 07500* 07507 07513 07517
07518 07519 07528 07531 07532 07534 07536 07538
07539 07660* 07878 07879 07976 07979 08434* 08467
[illegible] 08656* 08658* 08662* 08665* 08676 08679 08682
08685 08689 08693 08695 08699 08702 08708 [illegible]
08714 08885 09100 09474 09559 09741 09745 09747
09749 09752 09758 09763 09767 [illegible] 09772 09773
09775 09776 09777 09779 09780 09786
CONGENITAL ABNORMALITIES
01223 02156 02186 03095 04077 04121 04139 04150
04152 04153 04162 04169* 04176* 04180* 04200 [illegible]
04217 04218 04228* 04232* 04243 04280 04349 04359
04367 04394 04463 04509 04515 04610 04624* [illegible]
04976* 05001 05009 05050 05052 05061 05105 05116
05118 05163 05187* 05193 05210 05324 05331 05335
05492 05509 06019 06056 06081 06126 06232 06335
06362 06372 06505 06551 06632 06886 06989 07059
07068 07084* 07109 07121 07211* 07224 07236 07245
07334* 07335* 07406 07413 07447* 07466* 07488 07503
07524 07538 07558 07599* 07744* 07809 07878 [illegible]
08026 08031 08088 08092 08120* 08231* [illegible] 08269*
08278 08287 08308 08331 08341 08348 08434* 08460
08462 08494 08526 08601* 08667* 08681 08683 08687
08698 08746* 08762 08782 08830 08835 08901 08914
08915 08916 09103 09137 09142 09146 09152 09158
09219 09224 09245 09336 09358 09375 09380 09388
09451 09496 09531 09559 09563 09565 09575 09603
09607 09617 09767 09777 09779 09862 09869 09874
09992
CONSTIPATION
08342
CONTRACEPTIVE DRUGS IN LIVER INJURIES
01558 01850 06525 06544 07376 07377 07418* 07427
07439 07725* 08558* 08564 08586 09545
COOLING TREATMENT OF BLEEDING
[illegible] 00989 01089 02010* 04174* 05043 05994* 06002*
[illegible] [illegible] 07009* 07470* 08018 08027 08813 09139
09911
COPPER ABSORPTION
00773 03736 08942 09534
COPPER METABOLISM
07467*
COPPER METABOLISM,LIVER
01541* 03597* 04447* 04516* 04915 05426* 05456* 06451*
06515 07397 07770 07893 08425* 09572 09611
CORTICOSTEROIDS
00045* 00304* 00404 00436 00575* 00679 01211 01227
01296* 01301* 01314* 01367* 01395* 01615* 01731* 01838
01925* 02007* 02301* 02305* 02386* 02477* 02597* 02600*
02605* 02631* 03076* 03120 03176 03448 03496 03538
03540 03541 03553 03565 03631* 03740 03808* 03821*
03824* 03845 03873 03884* 04257* 04284 04303 04370*
04371* 04377 04399 04411* 04422 04463 04514 04527*
04570* 04576 04584 04586 04595* 04618 04619 04793*
04797* 04819 04820 04823 04941 05140* 05271 05334
05369 05372 05403 05450* 05475 05480 05487 05573*
05576* 05594* 05630 05668* 05700* 05737* 05808* 05810*
05830 06059 06064 06247* 06255* 06314* 06322 06382*
06392 06427* 06453* 06476 06543 06564 06565 06567
06582* 06613 06672* 06746* 06747* 06766 06800* 06867*
07047 07148 07184 07212* 07272 07273 07307* 07320
07397 07425 07429 07713* 07737* 07751* 07801 07837*
07975 08088 08113* 08136 08158 08189 08190 08199
08222 08235 08321 08409 08424* 08427* 08475 08524

CORTICOSTEROIDS
CONTINUED
08569 08598* 08605 08606 08619 08727* 08733* 08761
08791 08970 08978 08982 09180 09297 09301 09303
09344 09548 09636 09637 09647 09668 09671 09680
09699 09816 09826 09852 09894
CRIGLER-NAJJAR SYNDROME
03390 09620
CROHNS DISEASE
03227* 03279 03290 03304 04301* 04304 04330* 04374*
04867 05277 05278 05281 05282 05283 05284* 05333
05342 05343 05367 05963 06011 06042 06133 06315*
06316* 06317 06318 06319 06320 06321 06326* 06356
06360 06366 06385 06887 06890 07090 07181* 07182*
07183* 07184 07185 07186 07212* 07215* 07251 07252
07263* 07267 07269 07271 07557 07895 07903 07933
08218* 08219* 08220* 08221 08222 08223 08224 08232*
08254* 08263* 08313 08327 08370 09084 09320 09321
09355 09356 09359 09360 09379 09426 09835 09843
09866
CROHNS DISEASE OF COLON
00442* 00456 00521 00526 01368 01436 01458 01460
01466* 01470* 01474 01479 01969 02275* 02276* 02299*
02301* 02329* 02330* 02339 02341 02366 02385* 02387*
CROHNS DISEASE OF STOMACH
02080 02107
CRYOSURGERY
04216 04438* 06160* 06424* 07075 07422* 08072 08810
CURLINGS ULCER
03182* 04100 04972* 07131* 08173*
CUSHINGS SYNDROME
00541*
CYSTADENOCARCINOMA
01508 02439 08383 08384 08388
CYSTADENOMA
00564 01508 02439 08383 08384 08388
CYSTIC FIBROSIS
SEE ALSO MUCOVISCIDOSIS
03329
CYSTS,BILIARY TRACT
09777
CYSTS,GASTROINTESTINAL
09959
CYSTS,LARGE INTESTINE
04338 08330
CYSTS,LIVER
00655 01527* 01531* 01533* 01548* 01554 01556 01561
01861* 01918 01947 02478* 02498 02519 02541 02936
03422 03466 03938 03939 03954 04466 04503 04943
05486 05492 05499 05893 06499 06511 06619* 06842*
06893 06894 06938 07339* 07395 07409 07410 07417
08434* 08464 08468 08490 08494 08512 08530 09087
09503 09509 09510 09542 09559 09586 09619 09622
09659 09990
CYSTS,MESENTERY
06066 08818 08843
CYSTS,PANCREAS
00524 00561 01496 01504 01510* 01947 02418 02468
03311* 03317 03329 03341* 03350 03975 04386* 04387*
04391* 04419 04420 05390 05395 05402* 06411 06418*
07283 07284 07285 07297 07507 07847* 08379 08380
08381 08385 09431 09439 09441 09442 09448 09449
09459 09460 09461 09470
CYSTS,RECTUM
00514 00518
CYSTS,SMALL INTESTINE
08776
CYSTS,STOMACH
08069
CYTOLOGY
06678* 07670 09049
CYTOLOGY,EXFOLIATIVE
03916* 04215 05835* 05923 06212 06859* 06936 07869
07875 09308
CYTOTOXICITY
01470*

DEFECATION
01805* 01854* 03799 04343 04770* 04788 04789 05058
05059 05062 05316* 05349 06327* 06719* 07214* 08340
08847
DEGLUTITION
00078* 04784 05082* 05097 06732 06967* 07902
DIABETES MELLITUS
00249 00669 00703 01266 01675 02434 02465 03321
03351 03755* 03823* 03839 03842 04252 04394 04434*
04494 04528* 04604* 04656 04791* 04919 05294 05400*
05407 05412 05448* 05623 05654 05660 05714* 05737*
06013 06204 06324 06416 06468* 06494 06573 06588
06608 06880* 07191* 07302* 07328 07451* 07509 07510
07790 08375* 08413 08422* 08424* 08475 08528 08620
08645 08853 09041 09432 09444 09458 09475 09480
09532 09657 09718 09788 09845 09958
DIAPHRAGM
SEE ALSO DIAPHRAGM HERNIA
SEE ALSO HIATUS HERNIA
01143 04153 06999* 07952 09419
DIAPHRAGM HERNIA
00288 00462 01164 03900* 04060 04125* 04150 04152
04153 05091 05096 05105 05116 05118 05125 05171
06120 06125 06125 06126 06139 06942 06991 06994
06999 07987* 08001 08002 08024 08026 08029 08759
08914 09103 09154 09157 09827 09869 09874 09895
DIAPHRAGM RUPTURE
04120 04125* 05096 06050 06999 08002 08759 09874
09895
DIARRHEA
00126* 00195* 00201 00202 00223 00251 00256 00291*
00349* 00354* 00429* 00433* 00570 00925* 00946* 00953*
00971 01064 01066 01067 01068 01092 01112 01113
01255* 01260* 01282 01306* 01373* 01375 01442 01734*
01802* 01808* 01919 01938* 01980 01983 01988 02132
02138 02171 02236 02279* 02280* 02288 02293 02320*
02829* 02893* 02908* 02911* 02972* 02984* 03002* 03003
03004 03029 03051 03188* 03228* 03231* 03232* 03235*
03236* 03243* 03244* 03248 03263* 03720* 03793* 03897*
03913* 03974 03989 03991 04002 04035* 04037* 04040*
04049* 04051* 04057 04094 04096 04103 04106 04111
04112 04114 04230* 04236 04237 04261* 04264* 04283
04311 04312 04313 04314 04316 04317 04318 04328*
04336* 04357 04373* 04376 04775* 04787 04863* 04924
04981* 05002 05004 05017 05073 05075 05076 05184*
05185* 05208 05240 05257 05258 05273 05284* 05287*
05290* 05303 05309 05369 05376 05731 05841* 05846
06071 06165 06173 06263* 06264* 06324 06353 06851*
06863* 06873* 07062 07078* 07089 07114 07129* 07145
07183* 07189* 07191* 07192* 07194* 07197 07198 07199
07262* 07859* 08049 08063 08158 08177* 08201 08215
08216 08230* 08241 08248 08256* 08328 08399* 08443*
08718* 08730* 08732* 08739* 08756 08768 08780 08798
08799 08807 08851 08863 08877 08879 08883 08898
08902 09025 09082 09083 09182 09191 09230 09320
09324 09425 09458 09460 09657 09773 09809 09843
09859 09933 09947 09950 09953 09954 09958 09963
09968 09977 09995
DIARRHEA IN CHILDREN
SEE ALSO CELIAC DISEASE
00225 00938* 01037* 01093 01095 01868* 01919 01938*
01981 02984* 03041 03042 03960 03990 04023* 04035*
04041* 04047* 04070 04092 04104 05008 05293 05985*
05986* 06017 06020 06048 06092 06102 07200 07543*
08227* 08231* 08234* 08240 08245 08247 08248 08249*
08726* 08727* 08731* 08796 08898 08915 09074 09326
09328 09450 09819 09840
DIETARY DEFICIENCIES
00427* 01728* 01938* 01939* 02501 02585 02586 02587
02841* 03152* 03245* 03843 04062 04081 04168* 04229*
04523* 04669* 04683* 04715* 04723* 05131* 05452* 05532*
05992* 06802 06824 07079* 07192* 07198 07276* 07547*
07696 07729* 07761 07768 07775 07812* 08119* 08234*
08741* 09997
DIETARY DEFICIENCIES IN LIVER INJURIES
00729* 00730* 01544* 01599* 01641* 02579* 02627* 02630*
02639 02641 03374* 04515 05501 05600* 06033 06549
06778* 06808 06810 07330* 07780 08050 08557*
DIETARY FACTORS
00753* 01710* 01765* 02582* 02585 02586 02587 02700
02710 02724 02760* 02843 02872 02873 02898* 03101
03361* 03412 03497 03640 03677 03679 03816* 03817
03824* 03825* 03826 03828 03829 03832 03839 03850
03857 03858 03860 03862 03873 03877 04036* 04047
04062 04079 04080 04081 04088 04166* 04175* 04201
04278 04318 04339 04473 04508 04515 04535 04615
04634* 04648 04688* 04719* 04724* 04725* 04726* 04727
04744* 04800* 04815 04916 04990* 05008 05070 05361
05437* 05529* 05600* 05632* 05639* 05643* 05666* 05677
05678 05737* 05738* 05749* 05815* 05818* 05834 05997
06004* 06005* 06019 06040 06041 06069 06093 06142
06196 06216* 06220* 06236* 06261* 06267 06401* 06490
06509 06541 06623* 06709 06710 06712 06713 06714
06717 06735* 06735* 06794* 06795* 06802 06808 06820
06827* 06881* 07022* 07023* 07028* 07093 07106 07143
07188* 07195 07288 07380 07420* 07502* 07577 07631
07632* 07640* 07642* 07646 07647 07671 07686 07698
07704* 07707* 07710* 07716* 07726* 07764 07772 07773
07775 07779 07783 07785 07789 07810* 07823 07902
08038* 08089 08112* 08119* 08133 08226* 08234* 08377
08485 08543* 08548* 08555* 08557* 08576 08641 08735
08741* 08784 08797 08819 08820 08881 08886 08927
08957 08972 08989 08992 08993 09017 09264 09305
09339 09452 09487 09572 09580 09581 09590 09595
09811 09920 09924 09973
DIETARY MALABSORPTION TREATMENT
01372* 01373* 01376 02286* 02911* 03231* 04321 05296
05308 08239 08245 09326 09328
DIETETIC THERAPY
00396* 00425* 00429* 00978 00984 01026* 01250* 01638
01857* 01926* 02197* 02205* 02262 02286* 02395* 03097
03330* 03538 03556 03614 04015 04074 04273 04281

TETIC THERAPY
NTINUED
04288 04314 04379 04494 04509 04517* 04584 04586
04595* 04605* 04648 04939 05236 05284* 05286* 05308
05461 05463 05558* 05613 05632* 05986* 05997* 06007*
06211 06216* 06236* 06276 06476 06491 06567 06576
06613 06641 07094 07194* 07195 07494* 07502* 07523
07535 07563 07823 08222 08245 08607 08820 08891
09260 09266 09278 09320 09326 09328 09816 09880
09997
ESTION
E ALSO BILE
E ALSO PANCREAS
E ALSO SMALL INTESTINE
00210 00230 00258 00879* 01914 01966 02075 02125*
02987* 03039 03721* 04074 04230* 04714* 07193* 07834
09017 09023 09825 09887
ESTION IN CHILDREN
00153 06019 08881
ESTION,CARBOHYDRATE
00933* 01697* 02203* 03677 03687 03803 04044* 04079
04092 04253 05672* 05780 06699* 07577 07650 07652
07668* 08043*
ESTION,LIPID
00034 00066 00890 00895* 00898 01731* 01736* 01738*
02392* 02603* 02707 02822 03246* 03633* 03802 03886*
04080 05676 05779* 05796* 07290 07643* 07651 07654
07688 07783
ESTION,PROTEIN
00033 00062* 00064* 00193* 01765* 02473* 02603* 02755*
02807 02813 02825* 03019 03233* 03769* 03785* 03786*
03789 03930 04062 04230* 04714* 05676 05738* 06769
06833 07577 07914 08208 09967 09970
ESTIVE ACTION OF BILE
00101 02707 03790 07193* 07644*
ACCHARIDASE ACTIVITY
00179 02393* 02812 02980* 03110* 03112* 03397 03677
04044* 07650
ACCHARIDASE DEFICIENCY
00065 00126* 00179 00435* 01374* 02281* 03238* 04044*
04079 04092 04325 04880 05386* 06851* 07202 07203
08234* 08241 08244 09323
ACCHARIDASES,SMALL INTESTINE
00011* 00065 01255* 02281* 02391* 02392* 02393* 02731*
03238* 03245* 04092 04227* 04229* 04236 04253 04312
04325 04688* 04709 04739 04880 05181* 05293 05295
05386* 06669* 06699* 06707* 06851* 07202 07203 07621
08231* 08237
SEASES ASSOCIATED WITH ANEMIA
07266
SEASES ASSOCIATED WITH BILIARY TRACT
00771 02588 02676 08171* 08666*
SEASES ASSOCIATED WITH CANCER
00331 00625 01188 01189 01370 02298* 02396* 02497
02551 03047 03400 03918* 04361 04475 05074 05075
06104 06198 06204 06361 06527 07021* 07050 07287
07343* 07394 07521 07545* 07585 08004 08090 08167*
08220* 08318 08374* 08428* 08505 08675 08706 08877
09190 09360 09432 09457
SEASES ASSOCIATED WITH CHOLECYSTITIS
08166*
SEASES ASSOCIATED WITH CHOLELITHIASIS
00299* 00764 00954* 01249* 01514* 01575 01652* 01654*
01667 01670 02140 02201* 02450 02664* 02670 02981*
03336* 03475 03627* 03647 04235 04472 04636 04641
04655 04656 04659 04661 04662 04666 04667 05014
05473 05635* 06432 06463* 06627* 06631* 06639 06658
07102 07304* 07325 07327 07346* 07499* 07501* 07505
07506 07509 07521 07526 07537 07580 07843* 08166*
08388 08656* 08660* 08663* 08669 08674 08675 08682
08683 08684 08690 08700 08712 09220 09221 09449
09478 09759 09761 09788 09798 09804
SEASES ASSOCIATED WITH CIRRHOSIS
00600* 00699 00734 00741 00743 00747 01034* 01035*
01036* 01537* 01571 01642* 02473* 02640 02642 02645
02646 02658 02994* 03404 03475 03504 03603 03607
03617 04015 04034* 04442* 04445* 04455* 04464 04475
04492 04504 04604* 04609 04610 04612 04613 04614
04616 04620 04621 04622 04623 04916 05068 05069
05109 05260 05452* 05496 05521 05522 05592* 05593*
05595* 05596* 05597* 05603* 05609* 05618 05621 05623
05625 05626 05628 05629 05887 06475 06508 06527
06528 06579* 06583* 06588 06593 06596 06597 06598
06601 06603 06604 06605 06607 06608 06610 06611
06612 06614 06615 06616 06848* 06948 06968* 06973*
07016* 07136* 07343* 07344* 07406 07407 07416 07423*
07451* 07464* 07465* 07466* 07470* 07473 07476 07480
07483 07485 07486 07487 07488 07558 07858* 08422*
08428* 08521 08635* 08640 08644 08645 08646 08647
08648 09497 09547 09611 09703 09706 09707 09708
09711 09714 09718 09724 09726 09727 09728 09730
SEASES ASSOCIATED WITH DIVERTICULITIS
08283
DISEASES ASSOCIATED WITH ENTEROCOLITIS
09360
DISEASES ASSOCIATED WITH ESOPHAGUS
01149 03062 03088 03095
DISEASES ASSOCIATED WITH GALLBLADDER
01661* 08712
DISEASES ASSOCIATED WITH GASTRECTOMY
02091 02663* 03117* 05145* 05152 05153
DISEASES ASSOCIATED WITH GASTRITIS
01034* 01035* 01924* 01954 03890* 05144* 05418 07046
07274* 07839* 08076 08166* 09183 09197 09210
DISEASES ASSOCIATED WITH GASTROINTESTINAL TRACT
00213 00235 00237 00242 00258 01063 01065 01966
07956 08911 09933 09996
DISEASES ASSOCIATED WITH HEPATITIS
00699 00703 00719 01619* 02511 02600* 02602* 02603*
02645 03477 03570 04553 04558* 04575 04593 05260
05493 05522 06475 06508 06528 07016* 07134* 07344*
07416 08449 08463 08465 08521 08522 09614 09629
09672 09683 09789
DISEASES ASSOCIATED WITH JAUNDICE
00600* 00698 00916 02488 03380* 03381* 03893* 04445*
05393 05435* 07058 07348* 07349* 07866* 07890 08095
08477 08478 08511 08521 09576 09675
DISEASES ASSOCIATED WITH LARGE INTESTINE
00062* 00501 00502 00503 00504 00505 00509 00510
03061 09890
DISEASES ASSOCIATED WITH LIVER
00610 00616 00618 00627 00630 00645 00661 00664
00668 00669 00704 00716 00728 00770 00774 01610
01678 02486* 02559 02612 03458 03468 03587* 07969
08534 09552 09644
DISEASES ASSOCIATED WITH MALABSORPTION
00354* 00429* 01177* 02281* 04029* 04316 04321 04402
04403 04404 05289* 05293 05305 05306 05864* 05867*
06324 07655 08103 08243
DISEASES ASSOCIATED WITH MESENTERY
01291
DISEASES ASSOCIATED WITH PANCREAS
00062* 00385* 00540* 00543 00569 00719 00771 01121
01692 01939* 02465 02917* 03060 03068 03351
DISEASES ASSOCIATED WITH PANCREATITIS
00580 00586 01111 01499 01516* 01517 01518 01521
01661* 02434 02448 02458 03060 03332* 03342* 03344
03345 03672* 03908* 04391* 04392* 04396 04416 04423
04426 04429 04558* 05054 05179* 05288* 05383* 05400*
05404 05407 05408 05412 05414 05867* 06407 06411
06416 06418* 06419* 06421* 06424* 06429* 06432 06438
06992 07290 07299* 07302* 07309* 07310 07312 07313
07317 07318 07324 07558 07836* 08374* 08376 08379
08381 08388 08396* 08397* 08398* 08402 08408 08410
08411 08414 08418 08420 09442 09459
DISEASES ASSOCIATED WITH PARASITIC DISEASES
01068 01564 01924* 04493 05004 06332* 06422* 07339*
07570 07992* 08250* 08253* 08339 08521 08671 08833
08882 08889 09163 09354 09503 09541 09622 09635
09878
DISEASES ASSOCIATED WITH PEPTIC ULCERS
00284 00385* 01034* 01168* 01242 02201* 02235 02253
02998* 03047 04280 05131* 05144* 05221* 05241 06195
06242* 06256* 06286 06290 06508 06757 07134* 07136*
07137* 07152 07154 07155 07162 07179 07180 07274*
07304* 07327 07545* 07580 07689 08166* 08167* 08171*
08179 08196 08199 09728
DISEASES ASSOCIATED WITH PORTAL HYPERTENSION
01117 01952 02473* 02640 02644 02645 03046 04449*
05612* 06474 06549 06588 06980 06995 07318 07858*
07998 08380 08448 08635* 09059 09060 09523 09528
09552 09613 09707
DISEASES ASSOCIATED WITH RECTUM
00506 01053 09341
DISEASES ASSOCIATED WITH SALIVARY GLANDS
00185* 03344 04385*
DISEASES ASSOCIATED WITH SCHISTOSOMIASIS
09708
DISEASES ASSOCIATED WITH SMALL INTESTINE
00347* 00437 03061 03062 04029* 08159 09890
DISEASES ASSOCIATED WITH STOMACH
00330 00340 00341 00342 00343 01998 03062
DISEASES ASSOCIATED WITH ULCERATIVE COLITIS
00531* 00535 01466* 01481 01482 01925* 02385* 02390*
02391* 02392* 02393* 02396* 02976* 03295* 03296* 03297*
03300* 03302 04373* 04374* 05361* 05365 05366 05375
06387 06391 07238 07265 08224 08359* 09354 09422
09430
DISEASES ASSOCIATED WITH VIRAL HEPATITIS
00720 02615 02620 02625 03402 03527* 03554 03580
03590* 04559* 04580 04590 04594 05488 05570* 05573*
05578 05584 05587 05589 06430 06553* 06558* 06559*
06564 06566 06570 06573 06575 07446* 07449* 07451*
07453* 07454* 07458 07460 07462 07484 08609 08613
08614 08618 09675 09682 09684
DISEASES IN CHILDREN,GASTROINTESTINAL
00250 01938* 02378 03049 03288 03948 04028* 04070
04089 04090 04113 04364 05008 05010 05016 05997*
06051 06858* 08784 08848

DISEASES IN CHILDREN,LIVER
08426* 09538 09615 09670
DISEASES IN CHILDREN,PARASITIC
03063 05982* 06072 06332* 06422* 08766 08865 08882
08890 08897 09503
DISORDERS,STOMACH SECRETION
00040* 00041* 00118* 00188* 00189* 00290* 00291* 00294*
00312* 00320 00383* 00845* 00869 00935* 00970 00974
01034* 01166* 01361 01485* 01872* 02125* 03000* 03109*
03113* 03723* 03749* 03768* 03887* 03945 03974 03978
04031* 04049* 04193 04213 04258* 04989* 05031 05053
05098 05131* 05266 05418 05435* 05626 05764* 05852*
05867* 05879* 05884* 05928 06000* 06014 06018 06145*
06150* 06166 06208 06210 06240* 06252* 06598 06742*
06748* 06766 06858* 06934 06956 06958 07014* 07019*
07027* 07039 07040 07041 07068 07134* 07152 07154
07160 07176 07361 07362 07581 07582 07583 07584
07839* 07846* 07856* 08044* 08168* 08176* 08458 08768
08769 08779 08780 08798 08824 08826 09097 09210
09298 09313 09396 09809 09824 09865
DIURETIC AGENTS
06580* 07471* 08927 09699 09701 09712 09723 09736
09738
DIVERTICULA
01424
DIVERTICULA,BILIARY TRACT
03655 07495*
DIVERTICULA,COLON
03274
DIVERTICULA,DUODENUM
00187* 01254* 01258* 02148 03157 03164 04237 05039
05192 07104 07110 08121 08138 08151 08434* 08762
09223
DIVERTICULA,ESOPHAGUS
00283 01034* 01135* 01152 03009 03080 03086 04149
05082* 05126 06974* 08000 08004 08012 09837
DIVERTICULA,GALLBLADDER
07495* 08680 08687 09742
DIVERTICULA,GASTROINTESTINAL
00370 00484 01798* 01801* 01969 02322* 02354 04121
06689 08773 08806 09837 09866 09891
DIVERTICULA,LARGE INTESTINE
00187* 01402* 02314* 02318* 02322* 03067 03277 03283
03997 04335* 04341 04360 04368 05352 06339 06342
06345 06921 07100 07227 07233 07270 08153 08284
08303 08354 09385 09402 09913
DIVERTICULA,RECTUM
00459 02314*
DIVERTICULA,SMALL INTESTINE
00367 00368 00966 01034* 01274 01285 01286 01288
02115* 02143 02150 02289 03149* 05039 05159 05178*
05191 05192 06230 07100 07104 07110 07695 08121
08124 08131 08161 08176* 09223 09242
DIVERTICULA,STOMACH
02079 04176* 05157 05159 06185 06207 07249 08762
08773 09186
DIVERTICULITIS
00235 02354 09891 09922
DIVERTICULITIS DIAGNOSIS
00235 00451* 02901* 03161 03167 03286 03937 04105
04360 04368 04870 05353 07100 08773 09891
DIVERTICULITIS TREATMENT
00451* 02370 03161 03261* 03264* 04337* 04341 04342
04358 04360 04870 05353 06105 06339 06345 07100
08282 08773 09891
DIVERTICULITIS-INDUCED SECONDARY MALABSORPTION
02289
DIVERTICULITIS,DISEASES ASSOCIATED WITH
08283
DIVERTICULITIS,LARGE INTESTINE
00449* 00451* 00465 00946* 01414 01459 01805* 01882
02010* 02299* 02311* 02314* 02322* 02332* 02349 02370
02901* 03161 03261* 03264* 03286 04337* 04341 04342
04344 04358 04360 04867 05352 05353 06339 06342
06345 06354 06887 07238 08282 08283 08284 08332
09367 09369 09370 09402 09403 09891
DIVERTICULITIS,SMALL INTESTINE
02349 03167 09231
DIVERTICULUM,MECKELS
01265 02146 03169 03170 03171 04870 04976* 06890
07109 08124 08131 08147 08153 08266* 08729* 08743*
08823 08909 09231 09837
DRUG ABSORPTION
00808 00824 03738 03750* 04442* 04779* 05725* 05998*
06159* 06702* 08333 08905 08943 09005 09024 09914
DRUG EFFECTS ON BILE SECRETION
07823
DRUG EFFECTS ON BILIARY TRACT
05798 06626* 07530 07661* 07823
DRUG EFFECTS ON ESOPHAGUS
05785*
DRUG EFFECTS ON GALLBLADDER
04884 05956 08664*
DRUG EFFECTS ON GASTROINTESTINAL TRACT
00158 00167 00516 00518 01062 01820 01822 01937*
01938* 01995 02048* 02223 02992* 03014 03108* 03113*
03232* 03258* 03275 03725* 03884* 03980 04091 04094
04305* 04306* 04345 04382* 04600* 04779* 04786 04983*
05005 05181* 05784* 05789* 05885* 05981* 06080 06084
06090 06149* 06241* 06249* 06582* 06672* 06678* 06906
06912 07135* 07254 07656* 07658* 07664 07815* 07823
07981 08040* 08783 08791 08874 08879 08896 08927
08929 09123 09198 09297 09393 09400 09401 09504
09825 09839 09844 09851 09870 09887 09894 09899
09910
DRUG EFFECTS ON LARGE INTESTINE
04775* 07216* 07663 07820 08721* 08847
DRUG EFFECTS ON LIVER
00633 02552 02591 03359* 03394 03481* 03492 03496
03497 03498 03505 03585* 04439* 04444* 04471 04486
04494 04524* 04527* 04531 04694* 04697 04803* 04809*
04817 04818 04848* 05533* 05765* 05808* 05817* 05820*
05869* 06537* 06538* 06540 06626* 06769* 06780* 06799*
06805 06812 06818 06821 06822 06823 06853* 07401
07425 07426 07438 07702* 07705* 07713* 07723* 07728*
07737* 07748* 07751* 07757 07758 07760 07764 07771
07778 07782 07797 07798 07799 07807 07808 07864*
08450 08489 08522 08549* 08560* 08574 08586 08587
08591 08594* 08641 08652 08967 08970 08973 08978
08987 08994 08996 09000 09004 09504 09508 09540
09543 09624 09636 09648 09651 09668 09671 09712
09765 09918
DRUG EFFECTS ON MOTILITY
03796* 03797* 03800 04777* 04779* 04780* 04786 04884
05648* 05782* 05784* 05785* 05786* 05789* 05790* 05792*
05797 05798 05802 05803 05885* 05933 05956 05967
06080 06107 06620* 06702* 06719* 06721* 06726* 06731
06906 06912 07656* 07657* 07658* 07659* 07661* 07662*
09050 09054 09393 09816 09950
DRUG EFFECTS ON PANCREAS
02827* 03711* 04380* 04383* 04399 04407 05765* 06416
06775 07690* 08395*
DRUG EFFECTS ON PANCREAS SECRETION
00863 02782*
DRUG EFFECTS ON SALIVARY GLANDS
07672
DRUG EFFECTS ON SMALL INTESTINE
04978* 05710* 05777 07639* 08118* 08157 08158 09021
09022
DRUG EFFECTS ON STOMACH
04168* 04874 04978* 05668* 05686* 05744* 06758 07013*
07038 07603 07689 08040* 09195 09214 09282
DRUG EFFECTS ON STOMACH SECRETION
00040* 00041* 00043* 00046* 00328 00383* 00384* 00397*
00819* 00831* 00832* 00842* 00846* 00847* 00853 00867
00871 00872 00888 00953* 00986 01172* 01310* 01313*
01314* 01702* 01703* 01748* 01749* 01767* 01768* 01770*
01775* 01778* 01782 01791 02090 02216* 02788* 02789*
02792* 02796* 02799* 02808 02821 03000* 03122 03180*
03190* 03193* 03695* 03747* 03762* 03769* 03778 03796*
04141 04256* 04259* 04756 04764 05140* 05146* 05215*
05218* 05239 05268 05748* 05755* 05763* 06210 06235*
06252* 06258* 06259* 06287 06744* 06752* 06754* 06769*
07675* 07676* 07678* 07682* 07685 07687 08059 08178*
08200 09246 09307
DRUG METABOLISM
06825*
DRUG METABOLISM,LIVER
00088* 01559 02492 02863 02877 03482* 03483* 03880
04486 04524* 04525* 04550 04670* 04796* 04802* 04804*
04814 04818 04830 05589 05817* 05827 06541 06549
06795* 06811 06812 06818 06823 07711* 07712* 07715*
07716* 07741* 07758 07759 08444 08636* 08967 08973
08979 08995 08999 09000 09001 09024 09598
DRUG TREATMENT OF BILIARY TRACT
02919* 04668 05644* 05814* 06626* 06634 07514 07515
07523 07540 07752* 08432* 08673 09765 09934
DRUG TREATMENT OF ESOPHAGUS
05099 06113 06114
DRUG TREATMENT OF ESOPHAGUS SPASMS
00269 04141
DRUG TREATMENT OF GALLBLADDER
07535
DRUG TREATMENT OF GASTROINTESTINAL TRACT
00122* 00127* 00177 00384* 00946* 00953* 00986 00997
01024* 01093 01095 01219 01331 01364 01367* 01495
01522 01808* 01853* 01905 01909 01938* 01971 01972
01976 02098 02181 02252 02287 02302* 02305* 02397
02893* 02894* 02895* 02896* 02897* 02963 02984* 02987
03048 03076* 03284 03298* 03372* 03617 03734* 03882
03897* 03905* 03917* 03928 03932 03948 03956 03974
03988 04028* 04061 04075 04106 04595* 04865 04883
04923 04989* 04998 04998 05175 05334 05598* 05803
05993* 05999* 06006* 06007* 06012 06039 06059 06073
06074 06075 06083 06085 06088 06089 06091 06092
06094 06095 06098 06099 06100 06101 06102 06103
06105 06106 06107 06156* 06210 06213 06314* 06953
07064 07181* 07184 07382 07541* 07549* 07551 07560
07563 07564 07574 07578 07588 07590 07591 07744

TREATMENT OF GASTROINTESTINAL TRACT
INUED
981 08178* 08198 08216 08222 08267* 08286 08356
722* 08723* 08724* 08739* 08742* 08745* 08750 08754
756 08765 08766 08774 08775 08778 08781 08783
795 08799 08801 08802 08819 08827 08837 08844
847 08857 08860 08861 08862 08865 08866 08882
883 08887 08889 08890 08892 08910 09343 09354
393 09504 09816 09818 09825 09840 09842 09843
854 09861 09870 09873 09877 09887 09896 09905
910 09914 09918 09924 09934 09936 09947 09948
950 09978 09984 09995
TREATMENT OF HEPATITIS
692
TREATMENT OF LARGE INTESTINE
272 08321 08333
TREATMENT OF LIVER
361* 03362* 03382* 03405 03406 03407 03408 03539
540 03541 03543 03544 03614 03897* 03917* 03951
956 04447* 04489 04506 04568* 04584 04586 04592
925 05461 05469 05477 05576* 05613 06100 06247*
470 06491 06495 06506 06511 06517 06560 06562
567 06576 06582* 06613 06965 07351* 07367 07404
408 07471* 07478 07479 07752* 07808 08426* 08446
456 08570 08571 08572 08573 08585 08606 08641
652 08723* 08754 08756 09504 09508 09516 09519
540 09636 09637 09671 09680 09699 09719 09736
765
TREATMENT OF MOTILITY
797* 01804* 01867* 08267*
TREATMENT OF PANCREAS
455 02460 02466 04418 05416 06446 07307* 07315
319 07320 07321 07323 08376 08406 08407 09474
484
TREATMENT OF PEPTIC ULCERS
190* 03195 03198 08198
TREATMENT OF STOMACH
047
TREATMENT OF ULCERATIVE COLITIS
946* 01464* 01469* 01471* 02386* 02397 03298* 04370*
371* 04372* 04376 04377 04379 05369 05372 05374
380 06382*
TREATMENT OF ULCERS
758* 02195* 02202* 02204* 02205* 02235 02237 02243
252 02268 02272 03200 03208 03982 04276 04286
288 04290 05239 05243 05269 05275 05744* 06149*
235* 06237* 06247* 06249* 06255* 06258* 06267 06271
284 06292 06294 06763 06766 07020* 07038 07064
125* 07142 07143 07144 07156 07157 07160 07161
163 07170 07171 07172 07173 07174 07175 07176
191 08879 09250 09251 09260 09270 09273 09278
282 09298 09307 09308 09319 09894
-INDUCED BLEEDING
064 06086
-INDUCED LIVER INJURIES
683* 03481* 03719 03834 04094 04486 04533 04537
538 04542 04543 04545 04547 04548 04549 04550
554 05476 05526* 05534* 05539* 05544* 05547* 05554
557 05596* 05813* 06247* 06460* 06534* 06537* 06540
822 06823 06868* 07364 07425 07438 07441 07740*
771 08522 08539* 08555* 08574 08586 08588 09561
624 09646
-INDUCED PANCREATITIS
575* 05403
-INDUCED SECONDARY MALABSORPTION
113 02090 02138 03232* 03234* 03237* 03980 04091
305* 04306* 05285* 09329
-INDUCED ULCERS
304* 00401 01442 01925* 01982 02196* 02207* 02235
273 02722 03151* 03172 03200 03768* 04172* 04257*
267* 04279 05215* 05219* 06159* 06247* 06250* 06255*
262* 06298 06582* 06758 06766 06769* 07127* 07148
179 07397 08059 08172* 08179 08189 08190 08791
246 09297 09301 09894 09910
S IN LIVER INJURIES,CONTRACEPTIVE
558 01850 06525 06544 07376 07377 07418* 07427
439 07725* 08558* 08564 08586 09545
N-JOHNSON SYNDROME
665* 03369* 08478 09492 09584
S,PANCREAS
152 00172 00368 00549 00554 00566 00573 00776
343 01490* 01717 02438 02810* 04102 04409* 04431
658 04760 04858* 05401* 06426* 06431 06776 06935
324 07325 07326 07695 07884 07886 08379 08421
648 08873 09431 09455 09462
ING SYNDROME
184* 08174*
ING SYNDROME,GASTRECTOMY-INDUCED
186* 00302* 00303* 00314 00787 00791 01100 01211
322 01331 02011* 02062 02106 03009 03100* 03142
188* 03189* 03194* 04189 04204 04283 04296 04299
198 05885* 06147* 06152* 06157* 06173 06231 06266
277 07036 07060 07062 07129* 07132* 07177 08035*
049 08058 08062 08182 08185 09093 09175 09196
271 09300 09314

DUODENAL DISEASES
00228 00243 00355 00389* 00418 01013 01281 01293
01295
DUODENITIS
01259* 03064 03986 04271* 04280 04778* 05224* 08176*
DUODENUM
SEE ALSO SMALL INTESTINE
00224 00252 00351* 00368 00374 00524 00550 00573
00787* 00816* 00922* 00966 00994 01038* 01051 01080
01187 01247* 01253* 01264 01268 01273 01277 01278
01287 01288 01302* 01304* 01307* 01324 01337 01676
01723* 01739 01745 01878* 01975 02114* 02115* 02138
02145 02181 02191 02733* 02751* 02756* 02777 02786*
02981* 02983* 03008 03030 03037 03066 03175 03222
03224 03318 03328 03682 03725* 03726* 03756* 03764*
03777 03803 03805 03898* 03935 03966 03968 04185
04238 04243 04664 04741* 04751* 04766* 04785 04878
04890 04897 04986* 05186* 05191 05676 05716* 05748*
05789* 05791* 05794* 05795* 06221 06231 06232 06417*
06670* 06687 06720* 06729 06733 07043 07531 07658*
07660* 07691* 08126 08127 08147 08226* 08946 09117
09228 09247
DUODENUM ANOMALIES
08394
DUODENUM CANCER
06202 06222 06227 06410 07120 08125 08678
DUODENUM DEVELOPMENT
01708* 04698 08138
DUODENUM DIAGNOSIS
05930 06843* 08121 08171* 09317 09936
DUODENUM DISEASES
SEE ALSO ULCERS
00307* 02131 02139 02141 02206* 02960 03153* 03168
04233* 04244 06224 06234 08101 08151 08154 08420
08762
DUODENUM DIVERTICULA
00187* 01254* 01258* 02148 03157 03164 04237 05039
05192 07104 07110 08121 08138 08151 08434* 08762
09223
DUODENUM ENDOSCOPY
06843* 06849*
DUODENUM FISTULAS
02192 02419 04032* 04068 04666 05211 07120 08061
08202 09161 09804
DUODENUM HISTOLOGY
03717 04231* 04271* 04698 05685* 05707 06218* 07815*
08146 09233
DUODENUM MORPHOLOGY
00012* 01013 01259* 02116* 03707* 03717 04232* 04699
05009 05210 05226* 05687* 05691* 05930 07815* 08138
DUODENUM MOTILITY
05797 05799 05800 05801 05930 06190 06857* 06872*
07545* 09054 09936
DUODENUM MUCOSA
02248 03707* 03717 04231* 04271* 04698 04699 04778*
05681* 05685* 07080* 07448* 08101 08200 09202 09256
09273
DUODENUM NEOPLASMS,BENIGN
08146
DUODENUM OBSTRUCTION
06849* 08382 09241
DUODENUM PATHOLOGY
08672 09227
DUODENUM PERFORATION
06301 07108
DUODENUM RADIOLOGY
03950 04244 05930 06234 06308 06313 06843* 07158
08019 08121 08138 08146 08151 08171* 08420 09050
09054 09217 09223 09256 09273
DUODENUM SECRETION
00048* 00849* 01259* 01266 01366 01492* 01762* 01807*
01912 02215* 02408* 03707* 04001 04411* 05777 06214*
07448* 07614 07976
DUODENUM SURGERY
02001 03168 03185* 03201 03324 04244 04254 05237
06153* 06224 06225 06227 06301 06306 07108 07110
07277* 07496* 07539 08045* 08061 08138 08181 08210
08212 08394 08420 08678 08679 08691 08692 08713
08762 09169 09222 09226 09233 09252 09269 09296
09314 09436 09769 09782 09803 09912
DUODENUM TRAUMA
07017* 07108 07118
DUODENUM ULCER DIAGNOSIS
09211
DUODENUM ULCERS
00181* 00399 00405 01310* 01312* 01365 01768* 01962
01982 02211* 02213* 02214* 02215* 02217* 02218* 02219*
02228 02234 02238 02243 02246 02248 02250 02251
02252 02256 02266 02267 02268 02269 02905* 02982*
02998* 03107* 03185* 03188* 03189* 03190* 03191* 03201
03206 03210 03214 03216 03761* 04038* 04245 04256*
04258* 04260* 04261* 04262* 04264* 04266* 04270* 04271*
04272* 04273 04274 04276 04280 04282 04284 04288
04291 04292 04293 04297 04613 04765 04982* 05014
05018 05028 05098 05216* 05217* 05220* 05223* 05224*
05225* 05226* 05227* 05228* 05232* 05236 05237 05242

DUODENUM ULCERS
CONTINUED
05244 05245 05250 05253 05257 05262 05764* 05840*
05863* 05879* 05994* 06008* 06149* 06150* 06177 06235*
06236* 06237* 06239* 06240* 06242* 06244* 06245* 06248*
06250* 06251* 06253* 06254* 06257* 06258* 06259* 06260*
06268 06270 06272 06284 06285 06289 06292 06302
06303 06304 06305 06308 06309 06313 06532* 06843*
06883* 07006 07010* 07062 07071 07115 07125* 07128*
07133* 07134* 07138* 07140 07149 07151 07152 07158
07162 07164 07165 07177 07178 07180 07231 07285
07397 07684 07839* 07873 08059 08073 08108 08165*
08173* 08176* 08181 08187 08196 08197 08199 08201
08203 08204 08208 08209 08210 08212 08216 08729*
08730* 08842 09045 09048 09076 09077 09078 09201
09205 09248 09249 09252 09256 09257 09258 09259
09260 09262 09263 09269 09270 09271 09273 09275
09276 09279 09280 09281 09283 09285 09287 09290
09291 09292 09293 09295 09300 09303 09307 09311
09312 09313 09317 09318 09574 09728 09865 09908
09927
DUODENUM ULTRASTRUCTURE
03707* 07614 08137 08146
DYE STUDY TECHNIQUES
SEE ALSO MARKER STUDIES
01871*
DYSENTERY
SEE ALSO AMEBIASIS
SEE ALSO SHIGELLOSIS
SEE ALSO TYPHOID FEVER
01938* 01980 01988 03882* 05354 06060 06915 06916
07972 08582 08724* 08731* 08781 08814 08834 08838
08839 08863 08892 09879 09937 09944 09962 09977
09978 09983
DYSENTERY DIAGNOSIS
01012 01865* 03987 05888 06870* 06874* 06952 06953
06954 07859* 09943 09962
DYSENTERY EPIDEMIOLOGY
02991* 05888 05993* 07546* 07571 09879 09941 09943
09946 09999
DYSENTERY IMMUNOLOGY
01938* 03987 05888 06874* 07546* 09941 09946
DYSENTERY IN CHILDREN
01919 01938* 01971 03882* 03987 05888 08788 08796
09962
DYSENTERY PATHOLOGY
07210*
DYSENTERY PROPHYLAXIS
09999
DYSENTERY TREATMENT
01938* 03882* 03905* 03917* 05443* 05999* 06006* 07551
08572 08723* 08774 08775 08788 08801 08802 08809
08857 08860 08861 08862
DYSPEPSIA
02100 02211* 04134 05232* 05644* 05984* 07006 07028*
08073 08098 08110 08655* 08709 08898 09179 09192
09277 09840 09986
DYSPHAGIA
05085* 06087 06093 06299 07076
DYSPHAGIA,ESOPHAGUS
00123* 00985 02012* 02020 03069* 03070* 03075* 03076*
04133 04135 04136 04139 04140 04144 04145 04158
04264* 05102 05120 06108* 06109* 06110* 06117 06133
06136 06140 06972* 06974* 06975* 06976* 06983 06986
06998 07001 07002 07006 07985* 07987* 08013 08025
09132 09138 09144 09147 09160 09162 09166
DYSTENTERY TREATMENT
08756

EDEMA
00498 05609* 07114 07575 08085 08888 09214 09726
ELASTASE SECRETION,PANCREAS
00836* 03338* 03339* 03340* 04412*
ELECTROLYTE ABSORPTION
00038 00815* 01412 01740 01824* 02170 02290 02753*
02758* 02764 02770 02775 02776 02777 03002* 03674*
03722* 03730* 03751* 03913* 04023* 04331* 04674* 04703
04713* 04716* 04720* 04720* 04981* 05720* 05724* 05726
05731 06708* 06743* 07566 07622* 07623* 07624* 07627*
07630* 07633* 07636* 07637* 07638* 07642* 07688 08168*
08227* 08874 08944 09399 09737 09954
ELECTROLYTE EXCRETION
03676* 06569
ELECTROLYTE METABOLISM
02126* 02290 02363 02521 03041 03042 03260* 03669*
03955 04070 04180* 04453* 04863* 05368 05410 05731
07065 07124 07371 07543* 07633* 07636* 07637* 07638*
07688 07747* 08055 08516 08915 08939 09316 09701
09738
ELECTROLYTE METABOLISM IN CIRRHOSIS
03600* 03617 04600* 05604* 05613 05881* 06580* 07471*
07478 08634*
ELECTROLYTE METABOLISM IN GASTROINTESTINAL TRACT
00444* 01049 01096 01395* 08058
ELECTROLYTE METABOLISM IN PANCREATITIS
07309*
ELECTROLYTE METABOLISM,LIVER
06784*
ELECTROLYTE SECRETION,KIDNEY
00090* 03774 04445* 08055 08227*
ELECTROLYTE SECRETION,STOMACH
00301* 00305* 00819* 00859 01492* 01754* 01755* 01756*
01757* 01777* 01787 02235 02817 03732* 03747* 03763*
03770* 03772 03796* 04745* 04747* 04763 06744* 06747*
07665* 07678* 07688 08044* 08168* 09316
ELECTROLYTES
01245 01257* 01744 02126* 02220 02320* 02325* 02623
02908* 03260* 03600* 03722* 04376 04799* 06063 06405
06701* 06725* 08132 08369 08811 08812 09022 09458
ELECTROLYTES,SALIVARY GLAND
01772* 03759*
ELECTROLYTES,SERUM
00002* 00046* 00184* 00299* 00303* 00385* 00441* 00444*
00578 00756* 01395* 02337 02571 02753* 02978* 03029
03041 03042 03075* 03367* 03934 04456* 05289* 05604*
05720* 06001* 06017 06020 07139 07574 07807 08055
08072 08425* 08537 09737
ELECTROMYOGRAPHY
00884* 00885* 02831 04768* 04784 04849*
ELECTRON MICROSCOPY
00729* 00811 01015 01287 01451 01513* 01583 01584
01706* 02282* 02576* 02577* 02732* 02988* 03478 03517
03518* 03592*
ELECTROPHORESIS
01919 03579 05865* 06675* 06755 06819 06845* 07111
07683 07745* 07832 07842* 07921 07953 08366 08402
08456 08465 08486 08487 08537 08930 09100 09127
09518 09829 09923
ELECTROPHYSIOLOGY
00079* 01821 02102 02323* 03722* 03740 04674* 04766*
04789 04833 04849* 05445* 05781* 05791* 05795* 05799
05800 05883* 06191 06373 06687 06720* 06722* 06732
06741* 06882* 06967* 07379 07630* 07658* 07660* 07662*
07986* 08051 08939 09710
EMBRYOLOGY
04383* 05689* 07618 07809 08026 08092 08782 09412
09959
EMESIS
SEE VOMITING
ENCEPHALITIS
03337* 06445
ENCEPHALOPATHY,HEPATIC
02486* 02554 02637* 05514 05603* 05605* 06453* 0648
06503 06504 06509 06579* 06597 06605 07405 07406
07407 07466* 07486 07487 08441* 08461 08499 0850
08614 08638* 09522 09561 09639 09710
ENDOMETRIOSIS
00529 01388* 05048 05199 06015
ENDOSCOPY COMPLICATIONS
07993*
ENDOSCOPY IN BLEEDING DIAGNOSIS
00130* 01089 02961 03108* 03149* 05052 07873 07968
08018 09836
ENDOSCOPY IN CANCER DIAGNOSIS
00132* 01137* 02912* 03889* 06859* 06913 06936 07258
08015 08070 08326 09101 09144 09149 09364 09371
ENDOSCOPY IN PEPTIC ULCER DIAGNOSIS
00132* 09254
ENDOSCOPY,ABDOMEN
01902
ENDOSCOPY,BILIARY TRACT
05860* 05862* 07964
ENDOSCOPY,DUODENUM
06843* 06849*
ENDOSCOPY,ESOPHAGUS
00146 00160 00163 00282 00283 00284 00285 01011
01154 01157 01160 01864* 01973 02010* 02011* 02013*
02015* 03009 03076* 03086 03899* 03900* 04141 04154
04165 05107 05108 05113 05125 05929 05962 06109*
06116 06118 06141 06847* 06848* 06905 06913 06941
06942 06943 06945 06946 06951 06968* 06975* 06981
06983 07001 07005 07006 07046 07368 07873 07875
07889 07993* 08005 08007 08011 08012 08014 08016
08813 08855 09094 09095 09139 09140 09149 09162
ENDOSCOPY,GASTROINTESTINAL
00105* 00178 00943* 00979 00981 00989 01104 01897
02904* 02961 04073 06849* 06906 07873 07874 08813
08825
ENDOSCOPY,LARGE INTESTINE
00458 01400* 01428 01865* 02387* 03265* 03304 03920*
04364 04860* 04929 04931 05367 06949 07254 07265
08330 08363 08724* 09035 09427
ENDOSCOPY,RECTUM
00935* 01399* 03265* 04364 04929 04931 06913 06949
07259 08363 08364 08365 08781 09035 09088
ENDOSCOPY,SMALL INTESTINE
00921* 02119* 04897 05887 07861* 07871

NDOSCOPY,STOMACH
00105* 00130* 00132* 00146 00162 00163 00317 00335
00965 01007 01111 01167* 01182* 01186 01203 01209
01216 01232 01242 01332 01858* 01973 02050* 02052*
02080 02098 02099 02886* 02904* 02912* 02921 02942
02954 02971 03108* 03114* 03137 03146 03889* 03907*
03921* 03940 03945 03977 03996 04004 04184 04874
04876 04957 05133* 05835* 05900 05902 05903 05942
05945 05962 06021 06158* 06172 06212 06841* 06843*
06846* 06849* 06850* 06859* 06860* 06918 06919 06927
06936 06941 06946 06951 06985 07013* 07032 07068
07069 07128* 07845* 07849* 07855* 07872 07873 07885
07888 07896 07916 07935 08046 08052 08389 08807
08855 09031 09040 09044 09065 09094 09097 09098
09183 09184 09198 09282 09308
NTERITIS
SEE ALSO COLITIS
SEE ALSO ENTEROCOLITIS
SEE ALSO GASTROENTERITIS
00250 01116 01466* 02130* 02278 03891* 03981 05019
08129 08747* 08848 09230 09243 09845 09863
NTERITIS DIAGNOSIS,REGIONAL
04307*
NTERITIS IN CHILDREN
08737*
NTERITIS TREATMENT
09273
NTERITIS,REGIONAL
00186* 00201 00202 00354* 00420* 00421* 00422* 00423
00424 00442* 00521 00526 01115 01261* 01367* 01368
01369 01370 01403* 01436 01451 01470* 01474 01922*
01925* 01969 02275* 02277 02278 02281* 02290 02299*
02301* 02339 02341 02385* 02387* 03049 03225 03279
03290 03295* 03300* 04209 04301* 04302* 04303 04304
04330* 04374* 04867 05277 05278 05279 05280 05281
05282 05283 05284* 05333 05342 05343 05367 06011
06042 06105 06314* 06315* 06316* 06317 06318 06319
06320 06321 06326* 06360 06366 06385 06844* 07085*
07181* 07182* 07183* 07184 07185 07186 07212* 07251
07252 07263* 07267 07269 07271 07895 07903 07933
08218* 08221 08222 08223 08224 08246 08327 08354
08370 08758 09083 09320 09322 09355 09356 09359
09360 09379 09835 09866 09982
NTEROCOLITIS
SEE ALSO COLITIS
SEE ALSO GASTROENTERITIS
00149 01116 01441 02278 02331* 02339 02377 02894*
02931 03019 03271 04367 04972* 04973* 05006 05007
05477 06042 06069 06315* 06317 06873* 07227 08113*
08289 08335 08341 08751 09243 09843
ENTEROCOLITIS DIAGNOSIS
05838* 05888 06318 07181* 07215* 07915 09321 09356
09379
ENTEROCOLITIS ETIOLOGY
00198* 01393* 01398* 02385* 04059 04108 04345 05183*
05278 05315* 05992* 06010 06031 06053 06336 06360
07185 07186 07210* 07269 07271 07548* 08219* 08727*
08728* 08740* 08874
ENTEROCOLITIS IMMUNOLOGY
07263*
ENTEROCOLITIS IN CHILDREN
00938* 01106 01398* 04027* 04059 04367 05838* 05888
05992* 06048 07548* 07549* 08726* 08727* 08728* 08775
08785 08786 09937 09945 09993
ENTEROCOLITIS NECROTICANS
02308* 02358 03271 03275 04027* 04367 04972* 05183*
05337 08728*
ENTEROCOLITIS PATHOLOGY
00198* 01027* 01474 02299* 02301* 03271 04345 05278
05315* 06011 06053 06356 06366 07210* 07215* 07222
07271 07557 08223 08232* 08727* 09360
ENTEROCOLITIS SURGERY
09993
ENTEROCOLITIS TREATMENT
00938* 01025* 01393* 01441 02301* 03271 04883 05334
05343 05644* 06010 06031 06105 06314* 06318 06360
06366 06375 06385 07094 07181* 07183* 07184 07195
07212* 07549* 08333 08727* 08837 09355 09359 09379
09861 09880 09993 09995
ENTEROCOLITIS,DISEASES ASSOCIATED WITH
09360
ENTEROCOLITIS,PSEUDOMEMBRANOUS
03271 03287
ENTEROCOLITIS,STAPHYLOCOCCAL
07548* 07549* 08785
ENTEROPATHY,PROTEIN-LOSING
00149 00345* 00433* 00436 01025* 01043* 01261* 01274
01289 01376 01431 01451 01868* 01937* 02274 02298*
02324* 03231* 03915* 03981 04003 04029* 04030* 04081
04112 04315 04318 04903 04939 04947 04948 04951
04952 05002 05008 05063 05304 05368 05963 05973
06025 06315* 06873* 06903 07085* 07089 07119 07199
07204 07558 07699* 07919 08085 08129 08246 08760
08888 09450 09726 09957

ENZYME TREATMENT OF GASTROINTESTINAL TRACT
02987* 03785* 03974 05382* 05984* 08790
ENZYME TREATMENT OF LIVER
09645
ENZYMES
SEE ALSO AMYLASE
SEE ALSO PANCREAS SECRETION
SEE ALSO PHOSPHATASES
SEE ALSO STOMACH SECRETION
00001* 00119* 00599* 00602* 00638 01793* 03299* 03786*
05597* 07653 07833 09096 09298
ENZYMES IN CHILDREN,LIVER
00638 03370* 04847*
ENZYMES,BILIARY
04843* 05682* 07596* 07754 09797
ENZYMES,GASTROINTESTINAL
00003* 00018 01793* 02003 02161 02655 02761* 02812
02952 03244* 03683 04685* 05697* 05865* 06260* 06792*
09017 09019
ENZYMES,LARGE INTESTINE
00469 02003 02051* 02812 03254* 03677 07261* 07820
08258* 08263*
ENZYMES,LIVER
00083* 00097 00099 00682* 00690 00715 00891* 00892*
00899 00907 00909 00910 00910 00914 00954* 01541*
01563 01823* 01829* 01830* 01833* 01835 01836 01837
01838 01839 01840 01843 01846 01848 01849 01970
02477* 02490 02537 02566 02576* 02577* 02630* 02737
02837* 02839* 02841* 02842* 02843 02850 02852 02855
02858 02859 02862 02863 02869 02871 02872 02879
02881 02884* 02951 03358* 03380* 03397 03429 03431
03432 03433 03479* 03514 03515 03524* 03537 03550
03592* 03629* 03682 03806* 03807* 03812* 03816* 03818*
03821* 03824* 03825* 03828 03829 03830 03831 03833
03834 03837 03838 03839 03840 03845 03848 03851
03852 03853 03854 03856 03857 03858 03861 03862
03863 03869 03872 03875 03876 04320 04446* 04451*
04506 04518* 04545 04546 04690* 04792* 04796* 04797*
04798* 04807* 04808* 04809* 04810 04811 04814 04817
04818 04820 04821 04822 04826 04830 04831 04832
05049 05438* 05439* 05484 05535* 05539* 05561 05563
05689* 05694* 05701* 05812* 05817* 05820* 05821* 05824
05825 05827 05828 05830 06228 06466* 06519 06531*
06550 06769* 06778* 06780* 06781* 06783* 06786* 06787*
06788* 06789* 06792* 06794* 06795* 06799* 06800* 06801
06811 06812 06813 06814 06815 06818 06819 06823
06827* 07455* 07596* 07697* 07698* 07700* 07701* 07702*
07704* 07707* 07708* 07713* 07714* 07720* 07732* 07734*
07737* 07738* 07739* 07746* 07751* 07753 07754 07757
07758 07761 07766 07768 07769 07772 07777 07782
07784 07787 07788 07789 07791 07794 07796 07797
07798 07801 07804 07807 07829 07934 07946 07962
08476 08487 08495 08506 08508 08543* 08546* 08560*
08626 08643 08930 08959 08962 08963 08964 08965
08966 08967 08968 08970 08972 08973 08974 08984
08986 08994 08996 08997 08998 09000 09001 09006
09008 09013 09015 09016 09089 09099 09100 09555
09556 09563 09598 09601 09637 09641 09649 09653
09654 09675 09676 09694 09698 09719 09797 09972
ENZYMES,LIVER SERUM
SEE ALSO ALKALINE PHOSPHATASE
SEE ALSO LIVER DISEASE DIAGNOSIS
00166 00638 00715 00931* 00937* 00947* 01002 01003
01539* 01543* 01552* 01603 01615* 01616* 01631 01639*
01668 01852* 01863* 01869* 02470* 02576* 02597* 02598*
02602* 02604* 02606 02609 02622 02706* 02916* 02958
02959 03001* 03356* 03365* 03377* 03482* 03484* 03491
03499 03507 03523* 03531 03532 03536 03542 03549
03609 03615 03685 03806* 03835 03962 03967 03984
04020 04441* 04446* 04496 04523* 04541 04585 04813
04843* 04845* 04872 04879 04885 04915 04920 04932
05201 05442* 05466 05494 05536* 05546* 05553* 05560
05572* 05576* 05592* 05599* 05617 05637* 05859* 05866*
05889 05919 05926 05934 05959 06286 06460* 06513
06515 06532* 06545 06553* 06554* 06561 06568 06572
06577 06651 06777* 06789* 06852* 06853* 06854* 06855*
06865* 06867* 06869* 06923 06928 07351* 07369 07418*
07422* 07440 07444* 07449* 07459 07626* 07752* 07763
07806 07840 07842* 07864* 07867* 07870 07914 07962
08366 08424* 08425* 08441* 08474 08537 08541* 08549*
08561* 08562* 08594* 08595* 08598* 08601* 08612 08626
08986 09033 09086 09430 09625 09628 09641 09663
09673 09693
ENZYMES,PANCREAS
00044* 00067 00223 00540* 00545 00583 00594 00804
00805 00836* 00863 00864 00879* 01021* 01116 01484*
01486* 01489* 01494 01509* 01514* 01516* 01522 01697*
01765* 01771* 01933* 02416 02417 02430 02449 02453
02454 02456 02729* 02787* 02810* 02825* 02828 02987*
03325 03338* 03340* 03588* 03677 03683 03685 03686
03689 03690 03691 03692 03699* 03711* 03739 03787
03788 03789 03790 03965 03969 03971 03974 04382*
04389* 04412* 04424 04431 04669* 04687* 04693* 04722*
04734 04878 04920 04921 04960 04961 05203 05292
05382* 05383* 05395 05415 05522 05737* 05738* 05740*
05779* 05850* 05915 05932 06400* 06412 06424* 06425*

ENZYMES,PANCREAS
CONTINUED
06429* 06434 06435 06440 06528 06568 06770* 06776
06827* 06863* 06924 07190* 07274* 07278* 07288 07290
07295 07296 07301* 07306* 07307* 07308* 07315 07321
07323 07326 07329 07416 07446* 07651 07652 07693*
07696 07754 07836* 07838* 07853* 07914 07937 07958
08377 08395* 08402 08412 08415 08659* 08790 08954
09100 09210 09468 09469 09898
ENZYMES,SALIVARY GLAND
01773* 03683 07652 07666* 07672 07936
ENZYMES,SMALL INTESTINE
00011* 00179 00432* 01371* 01373* 01374* 01697* 01731*
01765* 01793* 02003 02051* 02157 02391* 02456 02731*
02812 02824 02826* 02827* 02902* 02913* 03002* 03110*
03228* 03233* 03239* 03243* 03245* 03673* 03677 03681
03682 03685 03687 04227* 04229* 04253 04305* 04306*
04313 04686* 04704 04714* 05160 05181* 05295 05666*
05687* 06669* 06708* 06827* 07448* 07595* 07620 07621
07652 07816* 07829 07833 07915 07976 08129 08225*
08237 08263* 08918 08926 09100 09243 09322 09898
ENZYMES,STOMACH
00001* 00298* 00789* 00838* 00880 01309* 01701* 01761*
01781 01794* 02980* 03110* 03715 04179* 04215 04702
05143* 05147* 05686* 05741* 05768 06148* 06155* 06677*
06690 06737 06740* 06769* 06833 07048 07440 08076
08947 09205
EPINEPHRINE
00002* 00050* 00443* 02779* 03675* 03762* 05776 05807*
05826 06146* 06738* 06759 06826* 07697* 07897 08088
08136 09535
ESCHERICHIA COLI
00198* 00222 00533 00537 00654 00681 00795 00822
00896* 00938* 01093 01367* 01540* 01795 01945 01971
01980 02761* 03288 03329 03892* 04035* 04059 04108
04411* 04676 04679 05006 05010 05077 05180* 05363*
05532* 05671* 05838* 05996* 06053 06092 06377* 06381*
06830 06831 06952 07261* 07262* 07542* 07548* 07904
07909 07911 07951 08238 08547* 08721* 08726* 08732*
08737* 08776 08800 08841 08884 08902 09852 09853
09937 09938 09939 09940 09941 09942 09943 09944
09945 09946
ESOPHAGITIS
00263* 00273 00284 00843* 00887* 00940* 00950* 01056
01057 01113 01139* 01141* 01151 01159 01170* 01946
02007* 02009* 02010* 02011* 02014* 02015* 02030 02887*
02990* 03009 03049 03070* 03076* 03077* 03078* 03088
04131* 04134 04135 04141 04142 04145 04147 04165
04203 04883 04969 04972* 05080* 05090 05097 05098
05099 05101 05102 05119 05120 05121 05122 05123
05124 05897 06124 06133 06137 06189 06696 06847*
06943 06945 06971* 06978 07001 07002 07006 07062
07145 07156 07983* 07987* 08009 08012 08013 08014
08058 08203 09136 09140 09147 09148 09164 09310
ESOPHAGITIS TREATMENT
06113 06114
ESOPHAGUS
SEE ALSO ACHALASIA
SEE ALSO HERNIA
SEE ALSO HIATUS HERNIA
00070* 01108 01154 01959 02283* 03094 03095 03096
04128* 05085* 06167 06974* 07826
ESOPHAGUS ANOMALIES
00271 01156 01165 01946 02013* 02017 02019 02027
02043 02114* 02907* 03080 03081 03087 03093 04077
04121 04139 04145 04162 05097 06140 06253* 06974*
06982 06987 06989 07991* 07992* 08000 08009 08013
08092 09132 09137 09142 09146 09152 09155 09158
ESOPHAGUS ATRESIA
02027 02907* 03081 03084 03087 03095 04164 07991*
07995* 09137 09152 09158
ESOPHAGUS BIOPSY
00146 00284 01888 05123 05929 06116 06118 06946
06971* 07875 09129 09140
ESOPHAGUS CANCER
01020* 01150 01153 01165 02025 02039 02041 03072*
03075* 03082 03109* 04053 04126* 04132 04140 04143
04146 04151 04154 04155 04156 04157 04999 05081*
05083* 05087* 05088* 05089 05103 05104 05106 05107
05117 05119 05124 05698* 05865* 05929 06077 06108*
06109* 06119 06121 06122 06128 06129 06135 06141
06838* 06972* 06987 06990 06996 06997 07003 07587
07875 07989* 07999 08004 08013 08016 08017 08020
08028 08028 08246 08487 08821 09051 09094 09113
09128 09131 09135 09143 09147 09149 09159 09832
ESOPHAGUS CIRCULATION
01934* 05084* 05164 06724* 09133 09708
ESOPHAGUS DEVELOPMENT
00271 01143 03087 06676* 06989 07610 07982* 07991*
ESOPHAGUS DIAGNOSIS
00123* 00146 00147 00160 00163 00268 00272 00281
00282 00283 00284 00285 00887* 00940* 00942* 00950*
00950* 00985 00990 01000 01011 01135* 01137* 01138*
01139* 01141* 01143 01148 01150 01154 01157 01160
01645 01864* 01888 01895 01908 01973 02006* 02007*
02010* 02011* 02012* 02013* 02015* 02035 02885* 02887*
ESOPHAGUS DIAGNOSIS
CONTINUED
02907* 02994* 03009 03070* 03076* 03078* 03083 03086
03093 03899* 03900* 03911* 04127* 04129* 04130* 04131*
04133 04134 04141 04146 04148 04149 04154 04160
04165 04774* 04916 04969 05082* 05091 05093 05094
05101 05102 05107 05108 05110 05111 05113 05114
05119 05120 05121 05123 05125 05126 05929 05957
05962 06109* 06112 06117 06118 06120 06121 06125
06130 06136 06138 06140 06141 06838* 06847* 06848*
06905 06913 06920 06941 06942 06943 06945 06946
06951 06960 06968* 06971* 06972* 06975* 06976* 06979
06981 06982 06983 06987 06994 06998 07001 07004
07046 07368 07858* 07875 07982* 08004 08005 08007
08010 08011 08012 08013 08014 08025 08029 08032
08482 09051 09095 09113 09129 09132 09134 09139
09140 09147 09149 09155 09162 09166 09168
ESOPHAGUS DISEASE ETIOLOGY
09140
ESOPHAGUS DISEASES
00123* 00276 01148 01161 01165 02014* 02895* 03088
03095 03096 04128* 05957 06111* 06987 09151 09156
09163 09166 09933 09966
ESOPHAGUS DIVERTICULA
00283 01034* 01135* 01152 03009 03080 03086 04149
05082* 05126 06974* 08000 08004 08012 09837
ESOPHAGUS DYSPHAGIA
00123* 00985 02012* 02020 03069* 03070* 03075* 03076*
04133 04135 04136 04139 04140 04144 04145 04158
04264* 05102 05120 06108* 06109* 06110* 06117 06133
06136 06140 06972* 06974* 06975* 06976* 06983 06986
06998 07001 07002 07006 07985* 07987* 08013 08025
09132 09138 09144 09147 09160 09162 09166
ESOPHAGUS ENDOSCOPY
00146 00160 00163 00282 00283 00284 00285 01011
01154 01157 01160 01864* 01973 02010* 02011* 02013*
02015* 03009 03076* 03086 03899* 03900* 04141 04154
04165 05107 05108 05113 05125 05929 05962 06109*
06116 06118 06141 06847* 06848* 06905 06913 06941
06942 06943 06945 06946 06951 06968* 06975* 06981
06983 07001 07005 07006 07046 07368 07873 07875
07889 07993* 08005 08007 08011 08012 08014 08016
08813 08855 09094 09095 09139 09140 09149 09162
ESOPHAGUS FISTULAS
02034 02040 03089 03092 04077 04142 04162 04164
05094 06108* 07995* 08006 08011 08016 09145
ESOPHAGUS HISTOLOGY
03074* 05086* 06116 06121 06676* 06971* 06993 07610
07992* 08028 09129
ESOPHAGUS IMMUNOLOGY
06993
ESOPHAGUS IN CHILDREN
01145 01146 01156 02012* 02017 02027 02907* 03069*
03087 03095 04121 04138 04162 04164 04771* 06112
06137 06970* 06982 06989 07600 07982* 07991* 07995*
08023 09152 09165 09167
ESOPHAGUS INJURIES
00266* 01908 05108 05110 05112 05113 05114 06043
06120 06123 06127 06158* 06905 07993* 07994* 08011
09139 09140 09150
ESOPHAGUS MANOMETRY
00887* 00985 00990 01000 01138* 02006* 02013* 02994*
03070* 04127* 04774*
ESOPHAGUS MICROORGANISMS
03076* 09140
ESOPHAGUS MORPHOLOGY
00266* 00275 01150 02043 04130* 04139 05085* 05114
05698* 06676* 06696 06974* 06989 07982* 07992* 07999
08010
ESOPHAGUS MOTILITY
00070* 00074* 00078* 00079* 00263* 00884* 00887* 00940*
00985 00990 01000 01138* 01139* 01141* 01142* 01155
01157 01158 01800* 01806* 01819 01925* 02013* 02020
02024 02831 02832 03070* 03074* 03076* 03078* 03797*
03899* 04101 04127* 04133 04134 04136 04138 04164
04263* 04784 04786 04969 05080* 05085* 05086* 05090
05095 05101 05124 05126 05785* 05957 06052 06140
06696 06724* 06732 06967* 06971* 06974* 07001 07986*
08023 08025 09147
ESOPHAGUS MUCOSA
01705 02013* 02807 03074* 03980 04133 05044 05095
05121 05122 05123 05126 06043 06118 06121 06133
06676* 06696 06945 06971* 06975* 06978 07002 07610
07673 07889 09129 09130 09140 09166
ESOPHAGUS NEOPLASMS
00147 00276 02022 02037 02038 03078* 06118
ESOPHAGUS NEOPLASMS,BENIGN
00281 00985 01147 01153 01162 02028 02039 04145
04148 04154 04969 05117 06117 06134 06138 06981
07989* 08008 08012 09134
ESOPHAGUS NEOPLASMS,MALIGNANT
00278 00279 00963 00985 01033* 01136* 01137* 01144
01152 01160 01969 02008* 02035 02042 02113 02885*
03009 03015 03072* 03079* 04126* 04145 [illegible] 05087*
05117 05698* 06030 06108* 06109* 07003 07999 08015
08028 08719* 08821 09135 09149

ESOPHAGUS OBSTRUCTION
01141* 01145 01151 01156 02011* 02043 03009 03069*
03070* 04141 04145 04155 05080* 05099 05102 05115
05125 06120 06123 06127 06137 06140 06141 06974*
08006 08011 08012 09138 09140
ESOPHAGUS PATHOLOGY
00265* 00278 01140* 01908 02030 02040 02043 03062
03081 04132 05103 06087 06138 07998 08012 08013
08018 08023 09138 09139 09151 09163 09907
ESOPHAGUS PERFORATION
00272 02023 02023 03086 03092 03093 04129* 04130*
04160 05088* 05093 05094 05108 05109 05110 06109*
06983 06985 06992 08005 08007 08011 08013
ESOPHAGUS POLYPS
08008
ESOPHAGUS PROSTHESIS
00277 02029 03094 04157 04163 05089 07988* 08003
08030 09135
ESOPHAGUS RADIOLOGY
00147 00279 00280 00281 00473 00942* 00980 01034*
01035* 01145 01154 01155 01157 01160 01864* 01908
02007* 02010* 02011* 02013* 02040 02990* 03076* 03083
03086 03093 03911* 04130* 04131* 04133 04138 04146
04147 04148 04149 04154 04161 04164 04165 04263*
04846* 04916 04969 05082* 05093 05094 05101 05102
05111 05114 05120 05990* 06112 06119 06121 06130
06131 06133 06136 06140 06141 06253* 06920 06941
06942 06945 06960 06971* 06972* 06975* 06976* 06987
06988 06994 07001 07004 07046 07600 07858* 07875
07889 07982* 07985* 08004 08005 08007 08010 08013
08019 08482 09051 09132 09140 09147 09154 09155
09159 09160
ESOPHAGUS REFLUX
00273 00843* 00887* 00940* 00950* 01139* 01158 01159
02006* 02013* 02015* 02887* 02990* 02994* 03069* 03070*
03074* 03078* 03088 03205 04134 04135 04136 04141
04142 04147 04165 04203 04969 05080* 05090 05092
05097 05101 05121 05123 06113 06114 06132 06189
06945 06971* 06978 06982 06984 06986 06988 07002
07006 07145 07156 07972 07983* 07986* 07987* 07990*
08009 08010 08013 08655* 09136 09138 09140 09148
09153 09155 09164 09291
ESOPHAGUS SPASMS
00269 02026 04145 05073 05085* 05086* 05095 06106
06115 08023
ESOPHAGUS SPASMS,DRUG TREATMENT OF
00269 04141
ESOPHAGUS SPHINCTER
00070* 00884* 00887* 00990 01138* 01152 01155 01158
02006* 02994* 03070* 03078* 04136 04147 04165 04771*
04774* 04784 05080* 05097 05101 05115 06052 06110*
06136 06189 06724* 06835 06920 06971* 06974* 06982
07001 07600 07982* 07983* 07986* 08010 08021 09164
ESOPHAGUS STRICTURE
00273 01141* 02010* 02013* 02014* 02043 02113 02990*
03091 03911* 04135 04139 04145 04147 05092 06116
07002 07006 07989* 08005 08009 08019 08030 08762
09135 09144 09153 09161 09162
ESOPHAGUS SURGERY
00262* 00265* 00270 00273 00274 00277 00285 00286
00473 00673 01059 01085 01135* 01136* 01137* 01140*
01142* 01144 01146 01147 01151 01152 01156 01157
01159 01160 01162 01163 02008* 02011* 02012* 02015*
02016* 02017 02018 02028 02029 02032 02034 02037
02039 02042 03009 03069* 03070* 03073* 03084 03086
03087 03093 03094 03096 03205 04135 04136 04137
04138 04139 04140 04141 04142 04144 04146 04147
04149 04151 04154 04156 04157 04160 04161 04162
04163 04164 04165 04975* 05082* 05084* 05088* 05089
05090 05091 05092 05093 05098 05107 05108 05110
05112 05113 05114 05115 05116 05117 05118 05137*
05164 06112 06115 06117 06122 06123 06124 06126
06127 06130 06131 06132 06134 06135 06137 06696
06942 06945 06969* 06970* 06972* 06974* 06976* 06977
06978 06981 06983 06986 06988 06990 06993 06995
06996 06997 06998 06999 07000 07003 07985* 07986*
07988* 07989* 07991* 07993* 07995* 07996* 07997 08000
08003 08005 08006 08009 08014 08015 08019 08023
08028 08029 08030 08032 08056 08079 08118* 08816
09130 09131 09133 09134 09135 09136 09138 09139
09141 09142 09145 09146 09150 09151 09152 09153
09154 09155 09157 09158 09161 09162 09164 09165
09167 09591 09811
ESOPHAGUS TRANSPLANTATION
06993 09150
ESOPHAGUS TRAUMA
00274 00285 01108 01146 02023 02023 03092 04133
04158 05093 05108 05112 05113 06137 06985 07993*
09145
ESOPHAGUS TREATMENT
05122 05123 06043 06111* 06972* 06976* 07004 07156
07987* 08011 08029 08813 09128 09140 09148 09151
ESOPHAGUS ULCERS
00473 01021* 01946 02016* 04129* 04141 04157 05119
07131* 08011 08730* 08779
ESOPHAGUS ULTRASTRUCTURE
07610
ESOPHAGUS VARICES
00226 00264* 00268 00281 00282 00370 00607* 00620
00942* 01011 01034* 01035* 01057 01112 01596 01642*
01645 01926* 01930* 01934* 02010* 02032 02033 02036
02473* 02484* 02628* 02645 02957 02994* 03046 03049
03073* 03083 03096 03355* 03413 03936 03949 04063
04073 04128* 04137 04285 04490 04598* 04599* 04603*
04606* 04616 04617 04621 04846* 04916 04926 05063
05099 05100 05511 05513 05602* 05603* 05607* 05617
05629 05897 05994* 06002* 06467* 06579* 06583* 06596
06597 06601 06610 06840* 06848* 06960 06968* 06970*
06973* 06980 06992 06995 07046 07341* 07368 07451*
07464* 07465* 07468* 07470* 07858* 07889 07900 07984*
07996* 07998 08013 08387 08428* 08448 08471 08473
08482 08526 08530 08632* 08642 08645 08729* 08813
08816 08836 08845 08900 09060 09098 09130 09236
09310 09479 09496 09539 09565 09613 09703 09708
09724 09726 09730
ESOPHAGUS,AGE EFFECTS ON
05119
ESOPHAGUS,CLINICAL STUDIES OF
00269 00272 00274 00275 00276 00278 00287 01148
01163 01645 01908 02019 02024 02038
ESOPHAGUS,DISEASES ASSOCIATED WITH
01149 03062 03088 03095
ESOPHAGUS,DRUG EFFECTS ON
05785*
ESOPHAGUS,DRUG TREATMENT OF
05099 06113 06114
ESOPHAGUS,FOREIGN BODIES IN
01145 03071* 04145 07004 08005
ESOPHAGUS,NERVOUS CONTROL OF
00887* 01149 01155 01161 01806* 01819 04127* 04136
05086* 05126 05957 06052 06724* 06732 06967* 07005
07992*
ESOPHAGUS,TOXIC EFFECTS ON
00266* 00287 03091 05083* 05112 06043 06724* 07999
08011
ETHIONINE
00590 00802
ETHIONINE IN LIVER INJURIES
01605
ETHNIC FACTORS
03311* 03598* 06241* 06753* 08390 08440*
EXCRETION,ALCOHOL
07827
EXCRETION,AMMONIA
06205 07019* 07040 09592
EXCRETION,BILE
01832* 02122* 02180 02650 03721* 04805* 04873 05796*
06649 06720* 06721* 06799* 07334* 07335* 07533 07628*
07711* 07712* 07715* 07717* 07741* 07774 07802 08306
08443* 08636*
EXCRETION,BILIARY TRACT
00117*
EXCRETION,CALCIUM
01245
EXCRETION,ELECTROLYTE
03676* 06569
EXCRETION,IRON
03676* 03915* 06606
EXCRETION,LIVER AMMONIA
00085* 00100 00597* 00601* 00671 00676 01528* 02509
02630* 03373* 03405 03406 03407 03408 03416 03606
03612 04014 04064 04491 04509 04546 04607* 04608*
04620 04622 04623 04812 04857* 05324 05592* 05828
05881* 06449* 06476 06524 08445 09047 09508 09553
09561
EXCRETION,PHOSPHORUS
01245
EXCRETION,PROTEIN
06034
EXCRETION,SODIUM
00090* 06034 06569
EXCRETION,TRACE ELEMENT AND MINERAL
03676* 03915*
EXFOLIATIVE CYTOLOGY
03916* 04215 05835* 05923 06212 06859* 06936 07869
07875 09308
EXPERIMENTAL GASTRITIS
01303* 05146* 06146*
EXPERIMENTAL PANCREATITIS
00579* 00590 00593 00949* 01044* 02413* 02424 02444*
02451 02453 02454 02456 02458 02462 02781* 03331*
03335* 03343* 03354 03672* 04098 04408* 04411* 04431
05383* 05401* 05856* 06417* 06419* 06420* 06434 06446
06871* 07298* 07306* 07307* 07308* 07310 07320 08395*
08400* 09487
EXPERIMENTAL STUDIES OF CANCER
01581 01582 01583 01585 01586 02077
EXPERIMENTAL STUDIES OF CIRRHOSIS
00729* 00730* 00731* 00732* 01633* 01720* 02579* 02656
07433 09589 09704

EXPERIMENTAL STUDIES OF HEPATITIS
00707 00721 00723 01626 01875* 02623 05442* 06459*
06553* 07345* 07805 08596* 08624 09671

EXPERIMENTAL STUDIES OF LIVER
00087* 01609 02527 02558 02573 02582* 02583 02589

EXPERIMENTAL STUDIES OF PANCREAS
02432

EXPERIMENTAL STUDIES OF SALMONELLOSIS
02384* 08744*

EXPERIMENTAL STUDIES OF SCHISTOSOMIASIS
00680

EXPERIMENTAL SURGICAL TECHNIQUES
02152 02317* 02429 02573 04078 04163 04242 04461
04477 04623 04631* 04640 04742* 04743* 05084* 05424*
05458 06219* 06327* 06413 06447* 06618* 06679* 07024*
07087* 07331* 07353 07403 07500* 07677* 07730* 07988*
08006 08009 08034* 08114* 08489 08658* 09350

EXPERIMENTAL ULCERS
00310* 00397* 00401 00408 00419 00868 01300* 01303*
01314* 01357 01366 02196* 02207* 02384* 02705* 02799*
03197 03198 04190 04259* 04267* 04268* 04279 04742*
05166 05213* 05215* 05275 05360* 06247* 06255* 06287
06288 06307 07127* 07135* 07580 07582 07656* 07676*
08168* 08175* 08201 09195 09264 09319

EXTRACTS,LIVER
00014* 00082* 00088* 00092 00093 00684* 00732* 03818*
04804* 05549* 06794* 07759 07768 07796 07797 07946
08960 08985 08997 08998 09003 09014 09015 09641

EXTRACTS,PANCREAS
03588* 04384* 04722* 05995* 06704*

EXTRACTS,SMALL INTESTINE
07594*

FAMILIAL DISEASES
00308* 00320 00321 00364 00387* 00448* 00450* 00576*
00651 00665 01055 01063 01256* 01379* 01429 01497
01929* 01940* 02133 02233 02312* 02459 02526 02528
02666* 02951 02995* 03053 03107* 03237* 03366* 03390
03421 03462 03960 04058 04097 04218 04234 04304
04313 04320 04492 04499 04502 04516* 04652 05194
05224* 05252 05307 05346 05386* 05397 05409 05426*
05430* 05431* 05608* 05987* 05988* 06240* 06241* 06402*
06427* 06593 06603 07041 07133* 07159 07187* 07201
07208* 07213* 07230 07235 07249 07342* 07393 07466*
07744* 07871 08076 08078 08123 08127 08140 08225*
08227* 08241 08242 08245 08271 08288 08298 08315
08337 08425* 08436* 08462 08495 08503 08518 08527
08746* 08749 08757 08856 08864 08912 08915 09265
09346 09352 09458 09492 09531 09575 09620 09665
09813 09967 09970

FATS
SEE ALSO DIGESTION
SEE ALSO LIPID ABSORPTION
SEE ALSO LIPIDS
SEE ALSO LIVER LIPID METABOLISM
SEE ALSO MALABSORPTION
00073* 00821 00898 01992 02000 02822 03316* 03912*
04080 05600* 05864* 06709 06713 07193* 07195 07577
07643* 07651 07654 07783 08848 09560

FATTY ACID ABSORPTION
00023* 00027* 00028* 00813* 00890 01736* 02636* 02845
03744 03802 03886* 04308* 04729* 05300 05308 05834
06236* 06705* 06735* 06822 07079* 07439 07710* 07726*
07803 07919 07919 08236 08945 08991 08993 09043
09716 09867

FATTY ACIDS
05361* 05551* 05819* 06003* 07640* 07701* 07783 07787

FATTY LIVER
00595* 00606* 00631 00648 00661 00678 00728 00729*
00755* 00947* 01015 01080 01104 01534* 01536* 01542*
01543* 01544* 01552* 01555 01556 01562 01576 01588
01599* 01606 01636* 01675 01730* 02402 02470* 02536
02539 02578* 02579* 02590 02639 03356* 03361* 03362*
03374* 03389 03434 03462 03468 03485* 03492 03495
03497 03503 03507 03517 03520* 03598* 03813* 03822*
03826 03836 03867 03954 04114 04434* 04443* 04443*
04452* 04494 04508 04513 04516* 04517* 04518* 04523*
04534 04535 04656 04683* 04793* 04902 04964 05241
05447* 05448* 05455* 05463 05471 05476 05500 05501
05520 05528* 05529* 05531* 05532* 05537* 05550* 05551*
05623 05629 05813* 05834 05848* 05954 06452* 06485
06494 06501 06517 06520 06521 06536* 06541 06543
06549 06623* 06692 06704* 06797* 06810 06815 06821
06822 06823 06824 06855* 06868* 07021* 07330* 07332*
07346* 07373 07391 07401 07420* 07424 07480 07705*
07729* 07749* 07751* 07779 07789 08385 08427* 08447
08455 08476 08501 08538 08540* 08548* 08589 08637*
08639 08728* 08955 08957 08987 08989 08993 09000
09041 09582 09595 09621 09626 09631 09805

FECES
00005* 00058 00122* 00149 00434* 00878* 00896* 00971
01367* 01384* 01403* 01452 01795 01865* 01883 02180
02291 02365 02374 02760* 02762* 02883* 02999* 03045
03155 03170 03721* 03886* 03892* 03912* 03943 03946
04036* 04854* 04873 05006 05007 05010 05058 05059
05298 05838* 05841* 05846* 05861* 05910 05931 05985*

FECES
CONTINUED
05987* 05988* 06076 06147* 06220* 06828 06862* 06907
06915 06916 06917 07281 07575 07817* 07876 07976
08272 08311 08360* 08456 08721* 08724* 08731* 08797
08800 08882 09024 09025 09106 09126 09218 09299
09450 09810 09818 09871 09915 09948 09955 09994

FIBROSCOPY
00132* 00162 00163 00178 00965 01201 01237 02921

FIBROSIS,CYSTIC
SEE ALSO MUCOVISCIDOSIS
03329

FISSURES,ANUS
00515 02364 05059 05325 05326 05330 07225 09362
09373

FISTULAS,ANUS
00476 01377* 01382* 04357 05316* 05325 05326 06326*
06328* 08293 09390

FISTULAS,BILIARY TRACT
00776 01342 01652* 01657* 01670 01680 01921 01949
02182 02478* 02682 03627* 04006 04007 04661 04871
06618* 06619* 07339* 07507 07508 07511 07513 07537
07791 07793 07800 08202 08530 08669 08700 09249
09659 09744 09771 09774 09785 09786

FISTULAS,DUODENUM
02192 02419 04032* 04068 04666 05211 07120 08061
08202 09161 09804

FISTULAS,ESOPHAGUS
02034 02040 03089 03092 04077 04142 04162 04164
05094 06108* 07995* 08006 08011 08016 09145

FISTULAS,GALLBLADDER
00757* 01655* 01680 01681 02667* 04641 04776* 05011
07511 09774 09804 09807

FISTULAS,GASTROINTESTINAL
00170 00199* 00221 00271 00285 00473 00866 01047*
01049 01123 01127 01152 01156 01342 01921 01949
01987 02017 02182 02290 02299* 02762* 03008 03746*
04032* 04068 04222 04238 04303 04423 04871 05027
05414 05935 06863* 07212* 07537 07563 08049 08327
08910 09082 09774 09854

FISTULAS,LARGE INTESTINE
00199* 01127 01655* 01680 01681 02419 03008 03155
04032* 04068 04344 04346 05027 05935 07183* 07218
07565 08065 08283 08309 08417 09386 09410

FISTULAS,LIVER
00206 00597* 00646 01949 02500 06458* 09720 09990

FISTULAS,PANCREAS
00546 01511* 01751* 01753* 01763* 01779* 01949 02432
02827* 03993 04414 07120 07294 07513 07695 09434
09439 09456 09466 09898

FISTULAS,RECTUM
01382* 01408* 02380 02387* 05312* 05322 07212* 09341
09386

FISTULAS,SMALL INTESTINE
00199* 01038* 01273 01275 01276 01278 01652* 01655*
02149 03008 03030 03066 04032* 04741* 05027 05211
07124 07183* 07565 07626* 08132 08143 08221 08222
09237 09898

FISTULAS,STOMACH
00041* 00046* 00397* 01655* 01754* 01755* 01763* 01767*
01779* 01785 01949 02803* 03746* 03764* 04032* 04741*
04749* 05027 05935 06761 07682* 08065 08309 08910
09200 09774

FOLIC ACID
01250* 01643 03415 04091 04305* 04306* 07194* 08593*
08891

FOLIC ACID ABSORPTION
00036 00311* 00816* 03104* 03242* 03247 03589* 04301*
05287* 07199 08116* 09329

FOLIC ACID IN MALABSORPTION TREATMENT
04301* 05309

FOREIGN BODIES IN BILIARY TRACT
05661

FOREIGN BODIES IN ESOPHAGUS
01145 03071* 04145 07004 08005

FOREIGN BODIES IN GALLBLADDER
05661

FOREIGN BODIES IN GASTROINTESTINAL TRACT
07105 08825

FOREIGN BODIES IN SMALL INTESTINE
08128

FREEZING TREATMENT
00295* 00319 00447* 01205 01557 03128 03128 04438*
07075 07422*

FREEZING TREATMENT OF PEPTIC ULCERS
01356 04216 05270 06160* 09911

FRUCTOSE ABSORPTION
04320 04688* 07647 08245 08495

GALACTOSE ABSORPTION
00024* 01913 02759* 04227* 04313 04837* 05529* 06810
07391 07629* 08241 08245

GALLBLADDER
SEE ALSO BILIARY TRACT

GALLBLADDER
CONTINUED
SEE ALSO CHOLECYSTITIS
SEE ALSO CHOLELITHIASIS
01647* 03633* 04628* 04629* 04630* 05639* 07495* 07832
GALLBLADDER ABSORPTION
01648* 01824* 01825* 03743 05656 05917 09560
GALLBLADDER ANGIOGRAPHY
02944 09067
GALLBLADDER ANOMALIES
00779 00783 02659* 04645 07495* 07503 07504 08434*
08680 08681 08683 08687 09779
GALLBLADDER BIOCHEMISTRY
01824* 02613 02695 04776* 05682* 07489* 07489*
GALLBLADDER BIOPSY
01674
GALLBLADDER CANCER
01531* 01650* 01654* 01674 01679 01942 02975* 02986*
03382* 03625* 04659 06621* 06622* 06627* 06639 07528
07931 08675 08680 08706 08711 09527 09743 09770
09772
GALLBLADDER CIRCULATION
01669 02768 04642 09790
GALLBLADDER DEVELOPMENT
00779 08680 08681 08683
GALLBLADDER DIAGNOSIS
00754* 00767 00954* 00981 01662* 01674 01881 01903
02671 02925 02944 04001 04625* 04861* 04884 05842*
05917 05922 05961 06621* 06628* 06636 06643 06658
06879* 06933 07265 07402 07528 07824 07894 07932
07974 08171* 08468 08668 08681 08684 08688 08694
08714 08943 09034 09037 09055 09067 09124 09584
09762 09776 09783 09791
GALLBLADDER DISEASE EPIDEMIOLOGY
00762 01668 01673 01674 03047
GALLBLADDER DISEASE ETIOLOGY
02679 04637 04642 06627* 06636 08688 08703
GALLBLADDER DISEASE TREATMENT
07382 08659* 09762 09776 09805
GALLBLADDER DISEASES
00750* 00751* 01659* 01662* 01685 02675 02694 03627*
03974 04628* 04629* 04634* 04645 04651 04668 05795*
06508 06637 06640 06646 06648 06652 07304* 07494*
07509 07511 07521 07914 08668 08673 08680 08681
08684 08686 08697 08709 08714 09250 09739 09740
09775 09791 09792
GALLBLADDER DIVERTICULA
07495* 08680 08687 09742
GALLBLADDER FISTULAS
00757* 01655* 01680 01681 02667* 04641 04776* 05011
07511 09774 09804 09807
GALLBLADDER HISTOLOGY
06622* 06671* 08665*
GALLBLADDER IMMUNOLOGY
06664
GALLBLADDER IN CHILDREN
01097 03664 04637 04654
GALLBLADDER MICROORGANISMS
03626* 08778 09765
GALLBLADDER MORPHOLOGY
05643* 05652 05682* 07616 08677 08680 08694 09742
GALLBLADDER MOTILITY
00076* 01796* 02663* 02894* 04085 04557* 04776* 04884
05152 05153 05644* 05648* 05660 05788* 05790* 05792*
05796* 05798 05802 05936 05938 05956 05961 06529
06634 06664 06718* 06721* 06933 06957 07529 07540
07661* 07824 07894 08045* 08051 08677
GALLBLADDER MUCOSA
01650* 01666* 01688 01824* 02669* 03743 05643* 05645*
05652 05703 06622* 06671* 07489* 08677 08680 08684
08697 09739 09795 09806
GALLBLADDER NEOPLASMS
05640* 08686 09743
GALLBLADDER NEOPLASMS,BENIGN
02671 03646 07495* 08680 08681 08703 08709
GALLBLADDER NEOPLASMS,MALIGNANT
00757* 00778 00784 02685 02986* 06617* 06621* 06622*
06627* 06639 07242 08711 09772
GALLBLADDER OBSTRUCTION
01676 03650 04653 05665 07257 08674 08681
GALLBLADDER PATHOLOGY
00754* 01650* 02679 04016 04637 05645* 06463* 06622*
06639 06646 06648 07528 08665* 08677 08680 08684
08687 08688 08697 08703 08704 08704 08709 08711
08714 09760 09783 09805 09806
GALLBLADDER PERFORATION
00750* 00769 01667 01669 01683 02669* 04654 04661
05655 06658 08663* 08683 09807
GALLBLADDER POLYPS
00784 02681 03646 08681 08684 08686 08688 08697
08703
GALLBLADDER RADIOLOGY
00139 00754* 00778 00779 00781 00977 02671 04019
04625* 04856* 04888 05872* 05901 05909 05917 05922
05961 06718* 06879* 06933 06963 07494* 07824 07894
07932 07974 08171* 08681 08684 08686 08694 08697
GALLBLADDER RADIOLOGY
CONTINUED
08703 09034 09037 09055 09067 09776 09791
GALLBLADDER SECRETION
00086* 00776 00839* 03641 05652 05656 05660 05956
06671* 07489* 07807 08677
GALLBLADDER SURGERY
00765 00766 01651* 01658* 01661* 01676 02668* 02675
02919* 03332* 03649 04628* 04632* 04638 04653 05634*
05642* 05649* 05653 05663 05665 06008* 06217* 06426*
06532* 06621* 06628* 06629* 06641 06642 06647 06650
06656 06659 06663 06879* 07491* 07493* 07497* 07506
07510 07522 07528 07531 07532 07878 08059 08118*
08469 08655* 08663* 08668 08668 08671 08672 08681
08686 08692 08702 08705 08707 08709 08714 09560
09745 09754 09757 09762 09770 09776 09778 09781
09783 09787 09788 09792 09806
GALLBLADDER TRAUMA
00769 01683 03910* 06629* 09807
GALLBLADDER ULTRASTRUCTURE
05643* 06671* 08704
GALLBLADDER VOLVULUS
04645 07504
GALLBLADDER,AGE EFFECTS ON
08663*
GALLBLADDER,CLINICAL STUDIES OF
00767
GALLBLADDER,DISEASES ASSOCIATED WITH
01661* 08712
GALLBLADDER,DRUG EFFECTS ON
04884 05956 08664*
GALLBLADDER,DRUG TREATMENT OF
07535
GALLBLADDER,FOREIGN BODIES IN
05661
GALLBLADDER,HORMONAL CONTROL OF
00782 00878* 02861 03648 04557* 04629* 04776* 04800*
05796* 06718* 06957 08045* 08677 09806
GALLBLADDER,NERVOUS CONTROL OF
00086* 00839* 00867 01796* 02663* 04085 04626* 04642
05788* 06718* 07145 07540 07616 08443* 09806
GALLBLADDER,TOXIC EFFECTS ON
07514
GANGLIONEUROMA
02288 06321 07378
GANGRENE
02317* 07103 07114 07997 08316 09236 09788 09790
GARDNERS SYNDROME
00519 01940* 05346 09419
GAS
01976 02510 02531 03103* 04074 04338 04990* 05331
06835 06883* 07537 07577 07579 07825 07901 07952
08087 08118* 08331 08414 08789
GASTRECTASIA
00323
GASTRECTOMY
SEE ALSO MALABSORPTION
SEE ALSO MALIGNANT STOMACH NEOPLASMS
00195* 00302* 00303* 01036* 01089 01090 01231 01302*
01362 01732* 02051* 02053* 02066 02067 02072 02082
02089 02212* 02237 02270 02294 02909* 03047 03107*
03130 03135 03139 03166 03182* 03188* 03189* 03196
03197 03228* 03748* 03749* 04048* 04166* 04203 04260*
04261* 04269* 04280 04292 04869 05035 05053 05092
05135* 05158 05164 05165 05214* 05221* 05231* 05255
06000* 06061 06127 06144* 06174 06199 06201 06202
06246* 06263* 06265 06270 06275 06283 06296 06303
06309 06532* 06846* 07010* 07029* 07032 07034 07035
07043 07044 07054 07066 07151 07153 07163 07177
07179 07871 08037* 08039* 08056 08064 08072 08094
08097 08098 08101 08111 08137 08183 08196 08205
08389 08779 08815 08824 08856 09076 09178 09189
09259 09261 09268 09272 09278 09300 09302 09309
09313 09314 09927
GASTRECTOMY COMPLICATION TREATMENT
00314 01025* 03097* 03142 03143 06152* 06173 06174
07129* 08035* 08049 08182 08184 08185 09191 09192
09196 09200
GASTRECTOMY COMPLICATIONS
00311* 00312* 00326 00338 00400 00418 00761 00971
01047* 01049 01104 01166* 01170* 01171* 01173* 01178*
01183* 01190 01191 01192 01199 01200 01206 01209
01214 01215 01219 01246* 01260* 01278 01304* 01306*
01322* 01331 01333 01343 01353 01358 01673 01719*
01856* 01949 02089 02112 02201* 02203* 02205* 02235
02261 — 02407* 02802* 03007 03058 03097* 03115* 03117*
03142 03143 03183* 03187* 03188* 03189* 03207 03981
04085 04140 04171* 04187 04189 04198 04201 04204
04208 04209 04210 04221 04222 04224 04274 04275
04278 04283 04298 04299 04787 05030 05034 05070
05072 05124 05130* 05137* 05144* 05145* 05150* 05151
05152 05153 05159 05160 05176 05177 05222* 05227*
05233* 05247 05255 05259 05294 05885* 05887 06018
06152* 06153* 06157* 06167 06173 06181 06184 06186
06189 06190 06196 06246* 06264* 06266 06269 06277
06286 06293 06306 06312 06646 07026* 07027* 07033

GASTRECTOMY COMPLICATIONS
CONTINUED
07036 07042 07060 07065 07128* 07132* 07147 07164
07177 07294 07545* 07657* 07935 08034* 08036* 08042*
08049 08050 08051 08055 08058 08060 08061 08062
08063 08065 08074 08082 08102 08170* 08182 08184
08185 08186 08195 08213 08276 08694 08793 09177
09191 09192 09202 09207 09250 09253 09258 09269
09271 09293 09300 09811 09865
GASTRECTOMY COMPLICATIONS,MICROORGANISMS IN
00312*
GASTRECTOMY TECHNIQUES
01033* 01047* 01097 01170* 01193 01211 03117* 03142
03143 03187* 04194 04195 05137* 05227* 06173 06174
06277 06278 06306 07849* 08034* 08170* 09177 09200
09247
GASTRECTOMY-INDUCED AFFERENT LOOP SYNDROME
00326 03142 04201 04204 04224 04299 05150* 06266
07036 07042 08049 08058 08185 08195
GASTRECTOMY-INDUCED ANEMIA
00311* 00312* 01206 02089 02754* 02756* 03097* 03104*
04204 05227* 06266 07177 08036* 08058 08067
GASTRECTOMY-INDUCED BILE DISEASES
04171* 04187 04204 04219 04221 04278 04940 05070
05071 05072 08041* 08103 08885
GASTRECTOMY-INDUCED DUMPING SYNDROME
00186* 00302* 00303* 00314 00787 00791 01100 01211
01322 01331 02011* 02062 02106 03009 03100* 03142
03188* 03189* 03194* 04189 04204 04283 04296 04299
05198 05885* 06147* 06152* 06157* 06173 06231 06266
06277 07036 07060 07062 07129* 07132* 07177 08035*
08049 08058 08062 08182 08185 09093 09175 09196
09271 09300 09314
GASTRECTOMY-INDUCED MALABSORPTION
00434* 01191 02281* 02294 03104* 04178* 04204 04208
04266* 04283 04730* 05144* 05145* 05867* 06029 06153*
06836* 06863* 07177 07197 08049 08050 08058 08077
08185 09176 09300
GASTRECTOMY-INDUCED METABOLIC CHANGES
01049 01996 02089 02203* 03104* 04166* 06155* 06836*
08043* 08045* 08058 08067
GASTRECTOMY-INDUCED PEPTIC ULCERS
00377*

GASTRECTOMY-INDUCED RECURRENT ULCERS
00132* 00312* 00400 00413 01315* 02221 03183* 03187*
03188* 03189* 03205 04204 04260* 04265* 04275 04283
04299 05233* 05255 05263 05840* 06248* 06293 06297
06310 06311 06841* 07036 07128* 07132* 07164 07177
07855* 08049 08058 08185 08730* 08769 08871 09253
09258 09284 09300 09309 02899*
GASTRECTOMY-INDUCED SECONDARY MALABSORPTION
04062 04201 05867* 08041* 09191
GASTRECTOMY,CLINICAL RESULTS OF
00381* 00395* 00411 00761 01170* 01245 01325 01327
01329 01330 01334 01336 01340 01348 01355 01996
02044* 02065 02101 02115* 02226 02262 02423 03142
03184* 03185* 04208 04209 04210 06277 06278 07033
08186 09269
GASTRECTOMY,DISEASES ASSOCIATED WITH
02091 02663* 03117* 05145* 05152 05153
GASTRIC ANALYSIS IN STOMACH DIAGNOSIS
00053 00118* 00317 00340 00343 00383* 00413 00848*
00979 01309* 02064 02087 02094 03945 04881 05133*
05134* 05879* 05879* 06904 09192
GASTRIC ANALYSIS TECHNIQUES
SEE ALSO HEIDENHAIN POUCH STUDIES
00040* 00041* 00104* 00109* 00118* 00133 00134 00291*
00944* 00955 01700* 01769* 01912 02780* 02788* 02792*
03181* 03705* 03751* 03796* 05141* 05751* 05755* 05852*
05879* 06902 06904 06934 07037 07684 07685 07839*
07854* 08006 08200 08949 09097 09308
GASTRIC PARIETAL-CELL ANTIBODY
00308* 01180* 02943 03109* 03111* 03113* 06176
GASTRICSIN
00838*
GASTRIN
SEE ALSO STOMACH
00045* 00048* 00054 00836* 00842* 00846* 00847* 00856
00859 00871 00873 00993 01021* 01051 01090 01308*
01310* 01358 01359 01487* 01698* 01699* 01701* 01716
01748* 01763* 01767* 01768* 01770* 01778* 01782 01783
01789 01790 01809* 01810* 01821 01899 01925* 02235
02265 02699* 02703* 02780* 02785* 02792* 02794* 02796*
02806 02993* 03000* 03004 03197 03209 03708* 03748*
03758* 03760* 03763* 03764* 03765* 03767* 03770* 03773
03776 03784 04037* 04039* 04167* 04407 04741* 04748*
04749* 04751* 04752* 04755 04756 04761 04776* 05031
05053 05167 05274 05695* 05696* 05736* 05740* 05741*
05742* 05743* 05752* 05753* 05755* 05756* 05759* 05760*
05761* 05766 05769 05770 05771 05772 05773 06000*
06014 06240* 06255* 06292 06726* 06737 06749* 06750*
06752* 06756 06760 06763 06765 06767 06768 06769
07584 07675* 07679* 07680* 07682* 07684 07690* 07835
08165* 08176* 08720* 08779 08949 09300
GASTRITIS
00212 01089 01111 01202 01644 01788 01954 02063
02199* 02211* 02957 03044 03102* 03125 03138 03140
03145 03146 03147 04038* 04173* 04192 04201 04223
04299 04310* 04972* 04979* 04985* 05042 05141* 05148*
05240 05625 05863* 05879* 05961 05994* 06002* 06008*
06069 06086 06143* 06150* 06151* 06155* 06166 06197
06212 06858* 07016* 07043 07045 07085* 07361 07362
07562 08054 08058 08098 08108 08211 08725* 08732*
08762 08836 08913 09045 09179 09199 09210 09254
09307 09310 09812 09845 09908 09910
GASTRITIS DIAGNOSIS
00053 00132* 00837* 00920* 00922* 00965 01034* [illegible]
01167* 01185 01209 01858* 02912* 03106* 03114* 03136
03137 03145 03146 03887* 03907* 03945 04177* 04876
04958 05056 05232* 05884* 05897 05942 06188 06192
06740* 06843* 06849* 06860* 06904 06918 06919 06927
07025* 07028* 07873 07916 08052 09183 09211
GASTRITIS DIAGNOSIS,RADIOLOGY IN
00339 03890* 03940 05912
GASTRITIS EPIDEMIOLOGY
08073 08166*
GASTRITIS ETIOLOGY
00312* 01166* 01171* 01209 01231 03113* 03114* 03137
04177* 04922 05056 06162 06200 06205 06582* 06755
07028* 07030* 07031* 07039 08052 08804 09181 09183
09198 09204 09209
GASTRITIS IMMUNOLOGY
06748*
GASTRITIS IN CHILDREN
01106 06048
GASTRITIS PATHOLOGY
01179* 03136 03137 03940 04214 04300 05133* 05147*
06162 06179 06188 06200 07025* 07037 08052 09171
09183
GASTRITIS TREATMENT
01024* 01231 02045* 02894* 02897* 03113* 03145 03190*
03208 03949 04177* 04922 05005 05005 05140* 05146*
05984* 06149* 06162 06192 06211 06249* 07026* [illegible]
07047 07064 07170 07171 08052 08837 09171 09180
09250 09273 09298 09816
GASTRITIS,ATROPHIC
00294* 00298* 00312* 00339 00813* 00834* 00844* 00845*
01027* 01048* 01115 01166* 01177* 01180* 01209 01924*
02980* 03106* 03112* 03114* 03137 03890* 05129* 05133*
05141* 05147* 05232* 06087 06177 06740* 07028* 07030*
07031* 07041 07068 07839* 08073 08076 09098 09171
09176 09180 09181 09210
GASTRITIS,DISEASES ASSOCIATED WITH
01034* 01035* 01924* 01954 03890* 05144* 05418 07046
07274* 07839* 08076 08166* 09183 09197 09210
GASTRITIS,EXPERIMENTAL
01303* 05146* 06146*
GASTRITIS,METABOLIC CHANGES IN
07019*
GASTRODUODENAL ULCERS
00404 02213* 02218* 02244 04279 08780 09183 09292
09297
GASTRODUODENITIS
04979* 06149* 06273
GASTROENTERITIS
SEE ALSO COLITIS
SEE ALSO ENTERITIS
SEE ALSO ENTEROCOLITIS
00938* 01031* 01037* 01093 01095 01113 01247* 01333
01986 01999 02002 02278 02984* 03041 03064 03970
04028* 04047* 04106 04940 05008 05057 05066 05570*
06008* 06053 06851* 07082* 07262* 07453* 07566 08049
08223 08725* 08749 08811 08812 08841 09230 09863
09947 09968 09975
GASTROENTERITIS DIAGNOSIS
07911 09989
GASTROENTERITIS EPIDEMIOLOGY
01106 02385* 02713 04045* 04059 05010 05224* 05992*
06048 07571
GASTROENTERITIS ETIOLOGY
08732*
GASTROENTERITIS IN CHILDREN
05996* 06088 06092 07543* 07544* 08728* 08884 09989
GASTROENTERITIS TREATMENT
05996* 06088 06092 07173 07174 08879
GASTROENTEROSTOMY
00313* 00368 01038* 01047* 01178* 01187 01246* 01298*
01299* 01304* 01305* 01322* 01946 02066 02215* 02274
02541 02663* 03004 03012 03058 03183* 03187* 03188*
03189* 03197 03776 04085 04181* 04195 04198 04222
04242 04260* 04261* 04264* 04265* 04266* 04274 04275
04282 04283 04293 04297 04975* 05124 05137* 05151
05163 05176 05186* 05205 05216* 05221* 05227* 05236
05255 05758* 05983* 06018 06045 06143* 06152* 06181
06186 06246* 06248* 06266 06269 06270 06285 06293
06308 06310 07014* 07027* 07033 07115 07128* 07146
07151 07153 07166 07177 07545* 07657* 08034* 08035*
08087 08141 08165* 08174* 08181 08182 08183 08202
08439* 08815 09200 09207 09248 09269 09284 09300

GASTROENTEROSTOMY,ROUX
02053* 02105 02188
GASTROENTEROSTOMY,ROUX-EN-Y
00294* 03161*
GASTROINTESTINAL ABSORPTION
00029 00117* 00797 00824 00827 01104 01725* 01726*
01741 01898 02752* 02754* 02764 02766 02771 02772
02773 02774 02775 03240* 03588* 03729* 03738 03750*
04718* 04728* 04731* 04738 04889 05711* 05716* 05728
05734 06039 06406 06702* 06711 06712 06713 06714
06828 07199 07555 07631* 07632* 07649 07919 08819
08941 08945 09069
GASTROINTESTINAL ANGIOGRAPHY
00932* 01252* 02315* 05844* 05868* 05873* 06070 06886
06891 06896 06909 07556 08131 09121
GASTROINTESTINAL BIOCHEMISTRY
00003* 00005* 00018 00210 00944* 01104 01713 01793*
02003 02161 02273 02655 02761* 02764 02812 02965
03244* 03683 03703* 04685* 04691* 05697* 05778 05865*
06037 06038 06095 06735* 06737 07631* 07673 07818
07821 07834 09019 09023
GASTROINTESTINAL BIOPOTENTIALS
00021* 00944* 02723 02775 02776
GASTROINTESTINAL BIOPSY
00018 00130* 00788* 00943* 00982 00989 01027* 02283*
02929 04685* 09092
GASTROINTESTINAL BLEEDING
00135 00181* 00215 00216 00217 00218 00267 00356
00361 00932* 01060 01130 01238 03102* 03173 07972
08173* 09964
GASTROINTESTINAL CANCER
02056* 02155 02950 03010 03022 03024 04053 04073
04093 04124 05015 05067 05865* 06001* 06021 06077
06091 06359 06899 07155 07547* 07569 07585 07901
08719* 08764 08821 08876 08903 09094 09813 09838
09892
GASTROINTESTINAL CIRCULATION
00213 00221 00522 00799 00830 00973 01132 01252*
01927* 02130* 02188 02317* 02319* 02514 02523 02698*
02861 02997* 03011 03251* 04067 04069 04118 04353
04460* 04681 04738 04935 04995 05040 05063 05066
05067 05304 05667* 05670* 05844* 06023 06070 06666*
06702* 06817 06835 06886 06891 06896 06899 07550*
07553 07554 07555 07556 07831 07887 08728* 08740*
08792 08817 08831 08850 08853 09091
GASTROINTESTINAL CIRCULATION STUDY TECHNIQUES
00798 00799 00897 00903 01977 01984 01989 01990
01994 02036 02112 02121* 02315* 02353 02510 02514
03903* 09036
GASTROINTESTINAL COMPLICATIONS
00249 01098 01240 02294 02338 02725 02982* 03022
03025 03332* 04062 04972* 05049 05177 05320* 06374
09168 09982
GASTROINTESTINAL CYSTS
09959
GASTROINTESTINAL DEVELOPMENT
00157 01708* 05908
GASTROINTESTINAL DIAGNOSIS
00018 00105* 00126* 00131* 00154 00167 00186* 00191*
00226 00251 00253 00788* 00932* 00943* 00944* 00948*
00971 00979 00980 00981 00982 00983 00989 00998
01006 01012 01027* 01046* 01056 01057 01070 01072
01073 01083 01084 01086 01100 01101 01104 01107
01237 01252* 01401* 01433 01816 01867* 01897 01914
01918 01920 01921 02283* 02904* 02918* 02929 02931
02990* 02992* 03022 03039 03126 03194* 03681 03682
03685 03892* 03895* 03915* 03922* 03923* 03933 03944
03958 03959 03961 03966 03968 03970 03973 03984
04027* 04107 04113 04115 04844* 04855* 04871 04895
04903 04940 04942 04948 04952 04968 05020 05023
05047 05073 05079 05177 05298 05844* 05847* 05868*
05873* 05885* 05921 05933 05935 05958 05967 05973
06016 06021 06031 06032 06039 06051 06056 06104
06106 06870* 06873* 06886 06891 06895 06899 06903
06904 06906 06909 06913 06925 06944 06952 06961
07002 07067 07113 07505 07579 07589 07590 07859*
07862* 07872 07887 07901 07902 07909 07910 07911
07919 07923 07924 07953 07956 07960 07972 07981
08021 08140 08782 08794 08825 08828 08863 08909
09063 09069 09070 09092 09121 09123 09175 09202
09804 09810 09887 09901 09962 09963 09982 09991
GASTROINTESTINAL DISEASE ETIOLOGY
00212 00220 00228 00231 00232 00237 00242 00251
00362 00385* 01055 01063 01064 01076 01115 01291
01440 02135 03005 03020 05282 06005* 06038 06054
08804 09017 09396
GASTROINTESTINAL DISEASES
00074* 00186* 00196* 00221 00230 00233 00237 00249
00348* 00423 00513 01046* 01099 01101 01102 01104
01410* 01972 01977 01980 01986 02210* 02905* 03028
03035 03039 03061 03062 03226* 04115 04843* 04979*
05020 05022 05048 05056 05073 05789* 06021 06031
06032 06040 06067 06078 06080 07569 07972 08054
08731* 08732* 08741* 08753 08757 08771 08783 08790
08794 08837 08843 08849 08874 08885 08909 08913
09182 09230 09400 09821 09842 09844 09858 09859
GASTROINTESTINAL DISEASES
CONTINUED
09860 09873 09891 09917 09935 09973
GASTROINTESTINAL DISEASES IN CHILDREN
00250 01938* 02378 03049 03288 03948 04028* 04070
04089 04090 04113 04364 05008 05010 05016 05997*
06051 06858* 08784 08848
GASTROINTESTINAL DISEASES,BIOCHEMICAL DIAGNOSIS OF
00210 00944* 00989 02238 04859* 04980*
GASTROINTESTINAL DIVERTICULA
00370 00484 01798* 01801* 01969 02322* 02354 04121
06689 08773 08806 09837 09866 09891
GASTROINTESTINAL ENDOSCOPY
00105* 00178 00943* 00979 00981 00989 01104 01897
02904* 02961 04073 06849* 06906 07873 07874 08813
08825
GASTROINTESTINAL ENZYMES
00003* 00018 01793* 02003 02161 02655 02761* 02812
02952 03244* 03683 04685* 05697* 05865* 06260* 06792*
09017 09019
GASTROINTESTINAL FISTULAS
00170 00199* 00221 00271 00285 00473 00866 01047*
01049 01123 01127 01152 01156 01342 01921 01949
01987 02017 02182 02290 02299* 02762* 03008 03746*
04032* 04068 04222 04238 04303 04423 04871 05027
05414 05935 06863* 07212* 07537 07563 08049 08327
08910 09082 09774 09854
GASTROINTESTINAL FUNCTION
00806 03997 04203 04224 04240 04343 06835 08074
08341 09023 09177 09916 09926
GASTROINTESTINAL HISTOLOGY
07073 07617 07669 07830 08848
GASTROINTESTINAL IMMUNOLOGY
00238 00239 00436 01055 01056 01115 01230 01741
01868* 02235 02385* 02392* 02410* 02411* 02773 02971
03895* 03958 03959 03960 03961 03987 04003 04050*
04112 04317 04369* 04402 04403 04404 04671* 04691*
04924 05016 05056 05284* 05299 05385* 05705 05712*
05838* 05905 06037 06069 07544* 08644 08760 09821
09835
GASTROINTESTINAL INJURIES
02162 02725 03014 03031 07131*
GASTROINTESTINAL LYMPHOGRAPHY
01992 04933 06899
GASTROINTESTINAL METABOLISM
00230 00298* 02158 02715 02718 03060 03322 08927
09023 09826
GASTROINTESTINAL MICROORGANISMS
00003* 00005* 00158 00177 00196* 00222 00795 00896*
00946* 01095 01115 01367* 01795 01811* 01883 01922*
01938* 01980 01981 01983 01988 02705* 02708 02727
02984* 02985* 03040 03045 03150* 03882* 03892* 03905*
03974 04014 04035* 04036* 04069 04074 04106 04108
04111 04241 04259* 04411* 04671* 04679 04862* 04887
04913 05006 05019 05035 05077 05526* 05598* 05713*
05780 05838* 05986* 05992* 05996* 05998* 06057 06060
06825* 06829 06830 06831 06832 06907 06915 06916
06952 06953 06954 07027* 07216* 07253 07262* 07544*
07548* 07566 07640* 07791 07830 07833 07859* 07909
07911 07951 08067 08145 08228* 08429* 08664* 08726*
08732* 08734* 08737* 08740* 08747* 08758 08778 08796
08798 08800 08804 08838 08841 08847 08849 08852
08881 08884 08888 08898 08927 09230 09810 09817
09819 09863 09871 09872 09881 09885 09938 09939
09940 09941 09942 09943 09944 09945 09946 09977
09978 09979
GASTROINTESTINAL MORPHOLOGY
00788* 01043* 01713 02164 02328* 02740 05001 05990*
06666* 06689
GASTROINTESTINAL MOTILITY
00068* 00069* 00074* 00080 00123* 00127* 00187* 00193*
00229 00788* 00883* 01725* 01808* 01809* 01811* 01815
01816 01818 01893 02117* 02151 02834 02835 02836
02990* 03757* 03793* 03798 04092 04097 04101 04110
04263* 04779* 04786 04787 04993 05079 05175 05784*
05792* 05803 05847* 05933 06013 06031 06039 06058
06080 06101 06106 06264* 06412 06689 06702* 06719*
06730 07656* 07823 07901 08767 08772 08847 08858
09023 09093 09291 09825 09839 09868 09873
GASTROINTESTINAL MUCOSA
00003* 00005* 00019 00021* 00827 01045* 01115 01705
01725* 01905 02130* 02295 02720 02725 02738 02747
02762* 02764 02766 02767 02974* 03703* 03722* 03729*
03731* 03738 04046* 04067 04095 04115 04685* 04691*
04738 04983* 04989* 04994 05020 05044 05045 05048
05066 05364 05697* 05705 05728 05865* 05973 06015
06025 06689 06702* 06735* 06792* 07044 07148 07631*
07667* 07669 07670 07821 07830 08179 08189 08738*
08740* 08791 08848 09121 09835 09935
GASTROINTESTINAL NEOPLASMS
00089* 00200* 00945* 01042* 01055 01430 01487* 01918
01993 02000 02004 02997* 03006 03023 03062 05023
05042 05047 06104 06368 09866

GASTROINTESTINAL NEOPLASMS,BENIGN
00224 00948* 01077 04042* 04068 06015 08748 08817
08824 08912 09823
GASTROINTESTINAL NEOPLASMS,MALIGNANT
00224 00227 00937* 00948* 00963 00969 00983 01033*
01081 01256* 01928* 01943 01967 01987 02292 03023
03904* 03923* 04050* 05067 06001* 06009 06030 06055
06077 06913 07547* 07559 08719* 08821 09813 09892
GASTROINTESTINAL NEOPLASMS,VILLOUS
00461 00524
GASTROINTESTINAL OBSTRUCTION
00200* 00220 00231 00362 00764 00795 01028* 01042*
01044* 01056 01057 01060 01103 01107 01109 01125
01392* 01401* 01430 01495 01655* 01927* 01928* 01950
01967 02000 02117* 02151 02199* 02290 02708 02974*
02977* 03003 03012 03040 03115* 03266 03970 03973
04070 04077 04078 04094 04103 04113 04121 04222
04228* 04299 04976* 04993 04996 05000 05001 05003
05023 05032 05041 05044 05066 05321 06015 06019
06051 06056 06098 06106 06181 06196 06689 06991
07111 07537 07542* 07566 08128 08272 08717* 08734*
08751 08763 08767 08770 08772 08792 08832 08841
08846 08895 09235 09866 09886 09900
GASTROINTESTINAL PATHOLOGY
00211 00220 00258 00338 01043* 01440 01928* 02283*
02683 03016 04046* 04055 04972* 05044 05045 05047
05079 05177 05908 06005* 06040 07542* 08740* 08792
08814 08858 08911 08912 09175 09182 09320 09821
09860 09887 09891 09904 09907 09912 09922 09954
09991
GASTROINTESTINAL PERFORATION
00191* 00232 00400 00511 00939* 00976 01028* 01039*
01042* 01056 01927* 01928* 02162 03043 04027* 04103
06082 08189 08728* 08757 08789 08806 08895 09297
GASTROINTESTINAL POLYPS
01060 01083 01957 07917 08140 09361 09823 09897
09982
GASTROINTESTINAL RADIOLOGY
SEE ALSO GASTROINTESTINAL DIAGNOSIS
00074* 00126* 00131* 00148 00154 00164 00167 00170
00191* 00242 00243 00244 00253 00512 00932* 00943*
00969 00980 00989 00998 01006 01027* 01050 01057
01100 01101 01252* 01797* 01800* 01867* 01909 01918
01920 01921 01985 02182 02918* 02961 02990* 03033
03126 03182* 03922* 03947 03973 04019 04068 04096
04101 04107 04115 04123 04222 04263* 04786 04844*
04855* 04871 04903 04933 04940 04942 04962 04968
04996 05020 05044 05045 05047 05079 05248 05844*
05847* 05873* 05921 05933 05935 05958 05967 06016
06021 06056 06070 06106 06186 06264* 06730 06886
06891 06909 06944 06961 06962 07068 07074 07113
07589 07590 07872 07901 07910 07923 07924 07956
07960 07981 08021 08073 08402 08434* 08782 08807
08825 08828 08855 08874 09063 09069 09070 09121
09774 09868 09982
GASTROINTESTINAL SECRETION
00402 00403 00797 02071 02816 02823 03017 03062
03195 03746* 04115 04677 05020 05778 06828 07656*
GASTROINTESTINAL SPASMS
04668
GASTROINTESTINAL SURGERY
SEE ALSO GASTROINTESTINAL TREATMENT
SEE ALSO ROUX-EN-Y GASTROENTEROSTOMY
00143 00184* 00199* 00221 00261 00262* 00286 00294*
00338 00348* 00363 00381* 00440* 00800 00939* 00973
01023* 01028* 01029* 01033* 01045* 01049 01056 01057
01059 01084 01085 01086 01087 01096 01342 01401*
01430 01480 01607 01911 01948 01967 01978 02021
02083 02152 02210* 02349 02743 02924 02974* 03010
03013 03018 03027 03040 03345 04027* 04032* 04043*
04055 04064 04068 04070 04071 04073 04078 04095
04113 04241 04365 04446* 04972* 04986* 04993 04995
04996 04999 05000 05009 05027 05029 05035 05050
05150* 05165 05321 05873* 05983* 06007* 06024 06027
06029 06032 06034 06056 06059 06077 06078 06689
06964 07093 07505 07552 08035* 08105 08215 08401
08717* 08763 08773 08782 08785 08815 08823 08825
08828 08832 08842 08852 08854 08858 08878 09161
09490 09754 09774 09802 09834 09854 09900 09991
GASTROINTESTINAL TRACT
00018 01236 06835 07577
GASTROINTESTINAL TRACT ANOMALIES
00157 00232 00998 01088 01094 01187 01217 01221
01950 03266 04055 04077 04121 05001 06019 06032
06056 06689 07902 08782 09992
GASTROINTESTINAL TRACT COMPLICATIONS
09935
GASTROINTESTINAL TRACT DEVELOPMENT
01094 02743 04055 04115 04691* 05020 05705 05908
06056 07667* 08782 09959
GASTROINTESTINAL TRACT DISEASE EPIDEMIOLOGY
00233 00237 00250 01055 01081 02991*
GASTROINTESTINAL TRACT HISTOLOGY
08929
GASTROINTESTINAL TRACT IN CHILDREN
00939* 00998 01094 01919 01950 02002 02004 02029
02421 04027* 04055 04077 04121 04691* 04976* 05001
05050 05847* 06019 06032 06047 06056 06106 06107
07111 07566 08731* 08733* 08751 08761 08806 08811
08812 08823 08825 08828 08830 08881 09070 09899
09957 09964 09969
GASTROINTESTINAL TRACT MORPHOLOGY
07616 07826 07830 08928
GASTROINTESTINAL TRACT,AGE EFFECTS ON
00233 01028* 01371* 01978 03034 03677 04036* 04691*
08792 08819 09198
GASTROINTESTINAL TRACT,CLINICAL STUDIES OF
00154 00211 00231 01062 01102 01898 02135 02182
GASTROINTESTINAL TRACT,DISEASES ASSOCIATED WITH
00213 00235 00237 00242 00258 01063 01065 01966
07956 08911 09933 09996
GASTROINTESTINAL TRACT,DRUG EFFECTS ON
00158 00167 00516 00518 01062 01820 01822 01937*
01938* 01995 02048* 02223 02992* 03014 03108* 03113*
03232* 03258* 03275 03725* 03884* 03980 04091 04094
04305* 04306* 04345 04382* 04600* 04779* 04786 04983*
05005 05181* 05784* 05789* 05885* 05981* 06080 06084
06090 06149* 06241* 06249* 06582* 06672* 06678* 06906
06912 07135* 07254 07656* 07658* 07664 07815* 07823
07981 08040* 08783 08791 08874 08879 08896 08927
08929 09123 09198 09297 09393 09400 09401 09504
09625 09839 09844 09851 09870 09887 09894 09899
09910
GASTROINTESTINAL TRACT,DRUG TREATMENT OF
00122* 00127* 00177 00384* 00946* 00953* 00986 00997
01024* 01093 01095 01219 01331 01364 01367* 01495
01522 01808* 01853* 01905 01909 01938* 01971 01972
01976 02098 02181 02252 02287 02302* 02305* 02397
02893* 02894* 02895* 02896* 02897* 02963 02984* 02987*
03048 03076* 03284 03298* 03372* 03617 03734* 03882*
03897* 03905* 03917* 03928 03932 03948 03956 03974
03988 04028* 04061 04075 04106 04595* 04865 04883
04923 04989* 04998 04998 05175 05334 05598* 05803
05993* 05999* 06006* 06007* 06012 06039 06059 06073
06074 06075 06083 06085 06088 06089 06091 06092
06094 06095 06098 06099 06100 06101 06102 06103
06105 06106 06107 06156* 06210 06213 06314* 06953
07064 07181* 07184 07382 07541* 07549* 07551 07560
07563 07564 07574 07578 07588 07590 07591 07744*
07981 08178* 08198 08216 08222 08267* 08286 08356
08722* 08723* 08724* 08739* 08742* 08745* 08750 08754
08756 08765 08766 08774 08775 08778 08781 08783
08795 08799 08801 08802 08819 08827 08837 08844
08847 08857 08860 08861 08862 08865 08866 08882
08883 08887 08889 08890 08892 08910 09343 09354
09393 09504 09816 09818 09825 09840 09842 09843
09854 09861 09870 09873 09877 09887 09896 09905
09910 09914 09918 09924 09934 09936 09947 09948
09950 09978 09984 09995
GASTROINTESTINAL TRACT,ELECTROLYTE METABOLISM IN
00444* 01049 01096 01395* 08058
GASTROINTESTINAL TRACT,ENZYME TREATMENT OF
02987* 03785* 03974 05382* 05984* 08790
GASTROINTESTINAL TRACT,FOREIGN BODIES IN
07105 08825
GASTROINTESTINAL TRACT,HORMONAL CONTROL OF
00883* 01815 02187 02829* 02861 02993* 04116 04117
05778 06039 08819 08824 09195
GASTROINTESTINAL TRACT,HORMONAL TREATMENT OF
01319* 01521 04310* 06035 07144 09444
GASTROINTESTINAL TRACT,NERVOUS CONTROL OF
00081 00127* 00197* 01081 01815 02151 02187 02894*
02895* 02918* 03016 03757* 03792* 04040* 04097 04766*
04924 05250 05677 05678 05732 05784* 05847* 06058
06157* 07579 07616 08767 08858 09291 09812 09962
09966
GASTROINTESTINAL TRACT,PHYSICAL TREATMENT OF
01005 09860
GASTROINTESTINAL TRACT,TOXIC EFFECTS ON
00182* 00218 01113 02158 02159 02160 02161 02162
02163 02176 02177 02718 02719 02720 02724 02725
02726 02744 02747 03031 03032 03033 03034 05728
06039 06047 07542* 07664 07681* 07831 08722* 08740*
08805 09070 09198 09204 09394 09400 09851 09879
09894 09910
GASTROINTESTINAL TRANSPLANTATION
09835
GASTROINTESTINAL TRAUMA
00573 00976 01927* 01944 02005 03345 04073 04113
06482 08852 09928
GASTROINTESTINAL TREATMENT
SEE ALSO GASTROINTESTINAL SURGERY
00122* 00127* 00164 00211 00251 00253 00267 00314
00356 00358 00423 00440* 00477 00482 00489 00490
00513 00933* 00946* 00953* 00958 00986 00997 01024*
01025* 01045* 01064 01066 01067 01093 01095 01100
01219 01331 01364 01367* 01401* 01440 01495 01521
01522 01808* 01853* 01893 01905 01909 01910 01911
01938* 01971 01972 01976 01983 01993 02135 02287
02302* 02305* 02347 02349 02362 02375 02377 02397

GASTROINTESTINAL TREATMENT
CONTINUED
02758* 02893* 02894* 02896* 02897* 02908* 02963 02984*
02987* 03027 03039 03048 03076* 03151* 03284 03298*
03372* 03475 03617 03734* 03785* 03882* 03897* 03905*
03913* 03917* 03928 03932 03948 03956 03974 03988
03991 04028* 04061 04075 04106 04174* 04584 04586
04595* 04605* 04863* 04865 04883 04923 04972* 04986*
04998 04998 05010 05027 05050 05282 05334 05372
05382* 05598* 05605* 05734 05735 05803 05982* 05984*
06006* 06012 06017 06020 06032 06039 06040 06051
06059 06073 06074 06075 06080 06083 06084 06085
06088 06089 06091 06092 06093 06094 06095 06097
06098 06099 06100 06101 06102 06103 06105 06106
06107 06213 06273 06282 06302 06314* 06375 07222
07541* 07542* 07551 07563 07564 07578 07590 07591
07834 08058 08356 08721* 08772 08774 08778 08783
08790 08794 08795 08799 08801 08802 08804 08813
08819 08820 08827 08842 08860 08861 08862 08879
09175 09182 09354 09393 09754 09816 09821 09825
09838 09839 09840 09844 09854 09858 09860 09870
09877 09887 09899 09918 09934 09963 09991
GASTROINTESTINAL ULTRASTRUCTURE
02163 02295 02328* 02740 07073 07617 07670 07818
08863 09019
GASTROINTESTINAL VARICES
08817
GASTROINTESTINAL WATER METABOLISM
00441* 01049 02129* 02290 02730* 02924 02978* 03041
03774 03955 04070 04180* 04376 04863* 04981* 05604*
06017 06020 07065 07124 07139 07543* 07633*
GASTRONE
00833*
GASTROPEXY
00288
GASTROSCHISIS
01223
GASTROSCOPY
SEE STOMACH ENDOSCOPY
GASTROSTOMY
01299* 03075* 04054 05170 06109* 07070 08471 09162
09441
GAUCHERS DISEASE
09616
GENETIC RELATIONSHIPS
SEE ALSO FAMILIAL DISEASES
00224 00364 00407 00435* 00439 00450* 00453* 00530
00555 00576* 00757* 01063 01173* 01317* 01453 01536*
01773* 02133 02233 02281* 02305* 02312* 02385* 02389*
02420 02631* 02632* 02635* 03741 04082 04092 04121
04150 04175* 04200 04227* 04270* 04325 04379 04402
04403 04404 04492 04499 04502 04624* 04693* 05131*
05224* 05252 05286* 05397 05409 05426* 05524 05569*
05610* 05987* 05988* 06038 06260* 06397 06563 06753*
06845* 07133* 07155 07208* 07213* 07264* 07393 08020
08078 08089 08204 08225* 08337 08390 08399* 08425*
09190 09265 09346 09353 09813 09992
GENETIC RELATIONSHIPS,BLOOD GROUP
00389* 01317* 01617* 02060 05149* 05616 08244 09017
09734
GIARDIASIS
08882 08883 08889 08890
GILBERTS DISEASE
04499 08478 09593
GLUCAGON
00836* 01697* 01783 04690* 05400* 05739* 05821* 05950
06737 06800* 07697* 08088 08971 08982 09444
GLUCOSE ABSORPTION
00817* 01722* 01724* 01825* 01870* 02293 02698* 02712
02748* 02758* 02759* 02763* 02913* 02999* 03110* 03116*
03245* 03373* 03673* 03727* 03728* 03913* 04170* 04313
04713* 04715* 04721* 04735 04736 04863* 05654 05714*
05719* 05724* 05732 06228 06691 06699* 06701* 06703*
06706* 06717 06826* 07639* 07760 08043* 08234* 08241
08245 08508 09175 09336 09452 09668 09718
GLUCOSE METABOLISM
05811* 06004* 07277*
GLUCOSE METABOLISM,LIVER
07756
GLYCOGEN STORAGE DISEASE
01589 02559 02951 03397 04079 05033 05049 05438*
05439* 05446* 09531
GLYCOGEN,LIVER
00092 00093 01831* 02499 02867 03358* 03696* 03698*
03819* 03821* 03832 04792* 04822 05033 05049 05810*
06543 06550 06778* 07187* 07430 07436 07743* 07760
07773 07786 07796 07801 08088 08451 08476 08546*
08557* 08587 08924 08933 08968 09010
GLYCOPROTEINS
00047* 02441 03894* 04819 05672* 05683* 05750* 05751*
08539* 08947 09760
GNOTOBIOSIS
00003* 01044* 02667* 02708 04672* 05713* 06694 07640*
GRANULOMATOUS COLITIS
SEE CROHNS DISEASE OF COLON

HAMARTOMA
07495* 08493
HEIDENHAIN POUCH STUDIES
00042* 00043* 00045* 00048* 00051* 00305* 00309* 00807
00841* 00842* 00847* 00857 00870 00871 00873 00874
01314* 01779* 01782 02046* 02780* 02788* 02792* 03181*
03736 03756* 03758* 03764* 03767* 03770* 04740* 04741*
04747* 04748* 04749* 04752* 04761 05455* 05740* 05745*
05751* 05758* 05760* 06760 07682* 07685 08168* 08458
08952
HEMANGIOMA
00165 01272 01439 02037 02123* 05209 05898 06233
08147 08308 09520 09559 09609
HEMANGIOPERICYTOMA
06233 08440*
HEMATEMESIS
02141 02218* 03089 05897 06257* 08027 09479 09906
HEMATOLOGICAL DISEASES
00026* 04580 08402 09545
HEMATOLOGY
00252 02089
HEMATOMA
02137 04254 08420
HEMOBILIA
SEE ALSO BILIARY TRACT
00656 01679 01686 01944 03636 04659 05490 05516
05880* 06464* 06654 06657 06892 07389 07390 08467
09623 09630 09638 09928
HEMOCHROMATOSIS
02555 02590 03017 03487* 04602* 06468* 08068 08531
08631*
HEMOLYTIC ANEMIA
01244 03133 03299* 05317* 05318* 05373 05875* 0765
07480 08444 08466
HEMORRHAGE
SEE ALSO BLEEDING
00673
HEMORRHAGIC PANCREATITIS
00588 02433 03336* 03337* 03339* 04408* 04411* 04412*
06438 07298* 07312 08379 08408 08411
HEMORRHAGIC RECTOCOLITIS
08370 08375*
HEMORRHOIDS
00516 01377* 01408* 01419 01423 05325 05344 05348
05349 05355 06328* 08294 08303 08321 08343 08349
08351 09343 09982
HEMOSIDEROSIS
00039 00096 00651 00678 01530* 01568 01719* 01968
02472* 02629* 02900* 03374* 03487* 03607 03739 04005
04436* 04590 04732 04733 05430* 05444* 05522 05681*
05875* 06468* 06716 07374 07467* 07840 08068 09546
HEPATECTOMY
SEE ALSO LIVER REGENERATION
00017 00091* 00092 00093 00632 00656 00680 01531*
01532* 01549* 01682 01834* 02490 02507 02515 02517
02706* 02714 02880 03377* 03382* 03427 03429 03431
03432 03433 03434 03435 03436 03437 03438 03439
03440 03441 03442 03443 03445 03446 03448 03449
03451 03453 03454 03455 03456 03808* 03815* 03841
03865 03866 04393 04433* 04438* 04461 04466 04477
04500 04525* 04526* 04529* 04544 04817 05470 05503
05530* 05536* 05541* 05551* 05567 05819* 05831 05832
05833 06464* 06478 06541 06549 06777* 06790* 06801
06805 06814 07333* 07350* 07355 07601 07705* 07722*
07723* 07730* 07736* 07751* 07755 07766 07795 08433*
08441* 08480 08488 08489 08491 08492 08504 08510
08514 08517 08535 08581 08654 08876 08968 08973
09509 09513 09516
HEPATIC COMA
SEE LIVER COMA
HEPATIC ENCEPHALOPATHY
02486* 02554 02637* 05514 05603* 05605* 06453* 06488
06503 06504 06509 06579* 06597 06605 07405 07406
07407 07466* 07486 07487 08441* 08461 08499 08500
08614 08638* 09522 09561 09639 09710
HEPATIC VEIN THROMBOSIS
SEE BUDD-CHIARI SYNDROME
HEPATITIS
SEE ALSO JAUNDICE
SEE ALSO LIVER
SEE ALSO LIVER COMA
00370 01567 01625 02588 02639 02641 02884* 03359*
03393 03464 03477 03624* 04466 04886 06462* 07349*
07356 07476 07957 08553* 08591 08698 09576 09683
09983
HEPATITIS COMPLICATIONS
00695* 00697* 00722 01612* 01615* 01618* 01619* 01625
02471* 02599* 02605* 02627* 02628* 02629* 03366* 03402
03520* 03523* 03535 03557 03558 03559 03560 03561
03562 03563 03570 03577 03580 04442* 04471 04556
04557* 04560* 04563* 04564* 04589 04593 04594 05473
05480 05485 05493 05570* 05584 05585 05608* 06570
07462 09629 09670 09683 09686 09696

HEPATITIS COMPLICATIONS,VIRAL
06556* 06559* 06571 06865* 09574
HEPATITIS DIAGNOSIS
00107* 00110* 00113* 00600* 00635 00677 00678 00701
00724 00927* 00941* 00954* 00992 01543* 01614* 01627
01647* 01860* 01917 02472* 02481* 02602* 02891* 03383*
03526* 03894* 03902* 03954 03962 03963 03967 04005
04008 04009 04011 04012 04020 04474 04478 04536
04568* 04571 04593 04837* 04845* 04873 04902 04934
04953 04954 04963 04964 05472 05497 05580 05836*
05892 05919 05924 05925 05926 05959 06469* 06473
06497 06500 06510 06520 06574 06894 06900 06901
06923 06932 06933 06947 07391 07402 07837* 07877
07889 07899 07913 07914 08427* 08454 08463 08523
08537 08569 08580 08611 09047 09099 09120 09694
HEPATITIS DIAGNOSIS,VIRAL
00107* 00711 00712 00713 00715 00721 00725 00727
01555 01611* 01613* 01615* 01616* 01629 01631 01852*
01863* 01886 01894 02598* 02606 02608 02959 02967
03521* 03523* 03531 03532 03533 03536 03538 03548
03549 03550 03553 03555 03557 03559 03560 03562
03575 03919* 03976 04559* 04562* 04577 04581 04582
04583 04872 04905 05572* 05579 05587 05588 05591
05837* 05870* 06465* 06554* 06555* 06557* 06561 06562
06573 06852* 06854* 06865* 06867* 06868* 06869* 06939
06957 07278* 07442* 07444* 07454* 07457 07459 07840
07867* 07883 07890 07893 07904 08474 08523 08592*
08593* 08595* 08598* 08610 08612 09029 09046 09081
09089 09664 09667 09673 09677 09684 09685 09686
09691 09693 09695
HEPATITIS EPIDEMIOLOGY
00703 00708 00717 01625 01627 03527* 03569 03571
03579 05422* 06512 08463 08502 08580
HEPATITIS EPIDEMIOLOGY,VIRAL
00603* 00695* 00705 01613* 01615* 01617* 01623 01624
01628 01629 01630 01631 02598* 02599* 02604* 02619
02624 02959 02991* 03383* 03519* 03547 03551 03559
03566 03567 03583 03919* 04565* 04566* 04567* 04575
04577 04579 04582 04583 04585 04589 05462 05569*
05572* 05577 05580 05588 05591 06554* 06560 06563
06566 06570 06573 06574 07418* 07447* 07452* 07457
07461 07463 07890 08592* 08596* 08599* 08599* 08600*
08602* 08608 08611 08613 08622 08624 08629 08630
09089 09665 09666 09672 09685 09690 09700
HEPATITIS ETIOLOGY
00014* 00600* 00605* 00639 00641 00695* 00705 00717
00721 00725 00954* 01566 01575 01608 01875* 02481*
02608 02610 03389 03519* 03544 03566 03567 04474
04479 04480 04515 04533 04536 04537 04565* 04567*
04571 04573 04574 04578 04587 04588 04591 05447*
05448* 05449* 05465 05497 05523 05555 05572* 05588
05591 05674 06462* 06465* 06470 06473 06490 06496
06497 06500 06501 06520 06522 06523 06545 06558*
06574 07021* 07137* 07345* 07365 07370 07399 07423*
07425 07457 07499* 08050 08454 08463 08523 08569
08582 08584 08608 08615 08619 08628 09624 09628
09647 09667 09672 09675 09684 09696 09789
HEPATITIS ETIOLOGY,VIRAL
07890
HEPATITIS IMMUNOLOGY
00709 00927* 01608 01627 01875* 02600* 03534 03621
03919* 03963 04021 04443* 04444* 04483 04511 04512
04554 04581 04588 04963 05523 05583 06470 06473
06473 06498 06874* 07345* 07408 07411 07415 07450*
07891 07913 09633
HEPATITIS IMMUNOLOGY,VIRAL
00723 01613* 01626 01630 01875* 02611 03521* 03525*
03533 03551 03553 03567 03914* 04562* 04569* 04577
05590 05870* 06554* 06868* 07411 07443* 08598* 08599*
08610 09029 09665 09666 09678 09684 09685 09690
HEPATITIS IN CHILDREN
03528* 03571 03572 03579 03581 03902* 04563* 04564*
04567* 04572 04574 04591 05446* 05493 06462* 06855*
07347* 07841* 08455 08462 08503 08524 08628 09100
09563 09615 09667 09683 09684
HEPATITIS IN CHILDREN,VIRAL
01613* 01615* 01617* 01622 02601* 02604* 02619 02631*
03049 03527* 03531 03539 03547 03548 03554 03558
03564 03573 03574 03575 03576 03577 03578 03582
04566* 04579 04583 04589 05576* 05577 05584 06556*
06560 06562 06563 06575 06576 06577 06852* 06854*
06869* 07278* 07442* 07444* 07448* 07452* 07459 07462
07484 07883 08463 08524 08593* 08594* 08597* 08598*
08599* 08600* 08602* 08604* 08605 08606 08607 08609
08610 08616 08619 08629 08630 09540 09664 09670
09676 09677 09679 09690 09695 09697 09700
HEPATITIS IN PREGNANCY
00708 03383* 03561 03562 08617
HEPATITIS PATHOLOGY
00634 00695* 00722 00724 00725 00726 00727 01626
02592 02593 02598* 02604* 02607 03378* 03387* 03522*
03550 03560 03568 04483 04569* 04578 05447* 05448*
05449* 05497 05581 05586 05587 05924 06469* 06497
06500 06518 06520 06521 06522 06523 06533* 07365
07424 07454* 07455* 07891 08523 08596* 08608 08614
08617 08618 08619 08636* 09628 09636 09645 09683
HEPATITIS PATHOLOGY
CONTINUED
09697
HEPATITIS PATHOLOGY,VIRAL
05442* 05543* 05574* 05575* 05582 06556* 06561 06572
06865* 07134* 07442* 07445* 07458 08595* 08615 08616
08621 09669 09671 09673 09679 09681 09682 09684
09686 09698
HEPATITIS PROPHYLAXIS,VIRAL
00718 01613* 01615* 01623 01624 01628 01629 01630
01631 02598* 03519* 03525* 03529* 03552 03569 04566*
04577 04579 05572* 05590 06563 07461 08611 08621
08629 09665 09666
HEPATITIS TREATMENT
01612* 02597* 02600* 02607 03415 03418 03497 03542
03572 03653 04444* 04494 04568* 04570* 04591 04592
05461 05489 05497 05555 05580 06469* 06517 06574
07347* 07351* 07361 07362 07365 07370 07404 07408
07425 07805 08424* 08445 08446 08474 08483 08485
08524 08541* 08569 08611 08820 09561 09636 09645
09692 09694
HEPATITIS TREATMENT,VIRAL
00700 00706 00711 00713 00714 00716 00720 01568
01596 01613* 01615* 01621 01622 01624 01631 02604*
02605* 02621 03383* 03523* 03535 03538 03539 03540
03541 03543 03544 03545 03555 03556 03559 03564
03565 03576 03582 04577 04578 04584 04586 05573*
05576* 05578 05579 05587 05588 05590 05644* 06495
06556* 06560 06562 06564 06565 06567 06575 06576
06577 06865* 06867* 07278* 07462 07963 08594* 08598*
08606 08607 08619 08621 08625 08626 08627 09540
09668 09671 09674 09677 09680 09685 09691 09695
09699
HEPATITIS,ACUTE
00698 00701 00931* 03374* 03389 03418 03524* 03534
03542 04593 05494 05497 05926 06488 06868* 07453*
07867* 07913 08424* 08442* 08475 08486 08573 08598*
09555 09573 09684 09686
HEPATITIS,ACUTE VIRAL
00603* 00704 00720 00726 00927* 01612* 01621 02603*
02614 03356* 03383* 03408 03416 03520* 03521* 0353
03555 03557 03563 03576 04442* 04556 04557* 04558*
04578 04580 04584 04586 05534* 05571* 05581 05583
05586 05588 05589 06556* 06558* 06567 06568 06569
06571 06572 06579* 06787* 06957 07443* 07454* 07455*
07456 07963 08463 09610 09676 09681
HEPATITIS,AGE FACTORS IN
02607 03523* 03547 03557 03558 03559 03560 03564
04567* 05571* 06556* 06558* 06852* 08524 08599* 08602*
08604* 09681
HEPATITIS,ANICTERIC
00641 00642 02481* 06854* 06869* 07442* 07444* 07452*
07840 08580 08595* 09684 09686
HEPATITIS,BIOCHEMICAL DIAGNOSIS OF
00107* 00145 00927* 00931* 01894 02601* 03574 03575
03578 04872 05571* 05579 06852* 06854* 08537 08618
09667
HEPATITIS,CHRONIC
00020 00634 00635 00639 00641 00642 00659 00697*
00702 00709 00710 00931* 01543* 01571 01612* 01619*
02597* 02600* 02613 02618 03356* 03358* 03378* 03383*
03389 03424 03503 03555 03621 03976 04478 04511
04553 04568* 04570* 04571 04588 04591 04592 04593
05433* 05434* 05444* 05519 05570* 05580 05584 05602*
06469* 06470 06473 06491 06494 06497 06500 06501
06503 06528 06570 06571 06577 06787* 06855* 06868*
06901 06947 07134* 07354 07362 07365 07370 07391
07404 07408 07411 07416 07450* 07451* 07453* 07778
07889 07891 07913 07914 08424* 08442* 08449 08463
08483 08485 08502 08513 08523 08530 08541* 08603*
08611 08613 09120 09633 09663 09686 09692 09696
09867
HEPATITIS,CHRONIC VIRAL
02604* 04443* 04444* 05571* 05585 05587 06558* 07454*
09573 09681 09687
HEPATITIS,CIRCULATION IN
00020 02511 05611* 06459* 09574 09682 09683
HEPATITIS,CLINICAL STUDIES OF
00020 00634 00635 00639 00704 00705 00706 00710
00717 00722 01875* 01894 02593 02607 02611 02614
08523 08604* 08628
HEPATITIS,DISEASES ASSOCIATED WITH
00699 00703 00719 01619* 02511 02600* 02602* 02603*
02645 03477 03570 04553 04558* 04575 04593 05260
05493 05522 06475 06508 06528 07016* 07134* 07344*
07416 08449 08463 08465 08521 08522 09614 09629
09672 09683 09789
HEPATITIS,DISEASES ASSOCIATED WITH VIRAL
00720 02615 02620 02625 03402 03527* 03554 03580
03590* 04559* 04580 04590 04594 05488 05570* 05573*
05578 05584 05587 05589 06430 06553* 06558* 06559*
06564 06566 06570 06573 06575 07446* 07449* 07451*
07453* 07454* 07458 07460 07462 07484 08609 08613
08614 08618 09675 09682 09684

ATITIS,DRUG TREATMENT OF
09692
ATITIS,EXPERIMENTAL STUDIES OF
00707 00721 00723 01626 01875* 02623 05442* 06459*
06553* 07345* 07805 08596* 08624 09671
ATITIS,LUPOID
00600* 00927* 01553 01571 01720* 04512 04591 04592
04593 06461* 09686
ATITIS,METABOLIC CHANGES IN
02597* 03524* 03573 03574 03575 03580 03581 04495
04561* 05444* 07337* 07448* 07805 07963 08442* 08593*
08597* 08598* 08601* 08617 08618 08623 08626 09628
09663 09693
ATITIS,METABOLIC CHANGES IN VIRAL
09491 09668 09669
ATITIS,MICROORGANISMS IN
04569*
ATITIS,SERUM
02486* 03383* 03522* 03529* 04562* 04569* 04575 04585
04590 04965 05572* 05588 06545 06556* 06563 06574
07447* 08592* 08608 08628
ATITIS,TOXIC
E ALSO TOXIC EFFECTS ON LIVER
00927*
ATITIS,VIRAL
00696* 00697* 00707 00708 00728 00773 01530* 01611*
01614* 01618* 01627 01632 01659* 01720* 01875* 02492
02609 02611 02615 02616 02617 02620 02622 02625
02884* 03378* 03440 03518* 03537 03561 03568 03569
03577 03580 03582 03583 03835 04009 04561* 04576
05440* 05475 05481 05533* 06461* 07450* 07778 07891
08500 08601* 08617 08618 08620 08621 08623 08625
08626 08627 09079 09546 09688 09689 09691
ATOLENTICULAR DEGENERATION
01541* 02472* 02551 02590 04447* 04492 04516* 04838*
05426* 05456* 06451* 06515 06603 07397 08425* 09534
09572 09611
ATOMA
02561 02566 03463 06579* 08514 08960 09013 09125
09596 09599
ATOMEGALY
00697* 01560 01579 02470* 02602* 02604* 02631* 02635*
02989* 03462 03897* 04796* 05036 05426* 05431* 05550*
05914 07351* 07788 08425* 08456 08463 08505 08755
08892 09236 09430 09563 09616 09657 09661 09681
ATOSPLENOMEGALY
00204 00675 03528* 04022* 04058 04076 04088 04114
04457* 04573 04621 05452* 05472 05495 05496 05607*
05608* 05631 06467* 06474 06959 07511 07570 08733*
08833 09635 09878
RNIA
E ALSO DIAPHRAGM HERNIA
00240 00245 00246 00248 00362 00475 00512 00795
01028* 01042* 01050 01055 01085 01125 01246* 02152
02346 05000 05032 05195 06181 07121 07552 08195
08846 09219 09228 09345 09815 09820 09866 09868
09900 09922 09985
RNIA IN CHILDREN
00286 01040* 01907 02128* 04152 04153 05105 05116
05118 06081 06126 07940 08019 08031 08914 09103
09142
RNIA,DIAPHRAGM
00288 00462 01164 03900* 04060 04125* 04150 04152
04153 05091 05096 05105 05116 05118 05125 05171
06120 06125 06125 06126 06139 06942 06991 06994
06999 07987* 08001 08002 08024 08026 08029 08759
08914 09103 09154 09157 09827 09869 09874 09895
RNIA,HIATUS
00139 00187* 00263* 00265* 00286 00843* 00940* 00950*
00985 00990 01021* 01034* 01057 01139* 01140* 01141*
01151 01153 01158 01159 01164 01864* 02009* 02010*
02011* 02015* 02018 02019 02031 02199* 02887* 02897*
02904* 02994* 03009 03077* 03078* 03624* 03900* 03911*
04043* 04131* 04134 04135 04147 04159 04161 04165
04202 04883 05042 05080* 05082* 05095 05098 05099
05101 05111 05115 05119 05122 05124 06019 06112
06114 06124 06125 06126 06130 06132 06696 06847*
06920 06942 06945 06967* 06979 06982 06983 06984
06986 06988 06994 07000 07002 07006 07074 07873
07982* 07983* 07985* 07990* 07997 08001 08005 08010
08014 08019 08021 08022 08026 08029 08032 08079
08169* 08655* 08762 09103 09136 09138 09139 09142
09147 09154 09155 09156 09157 09165 09167 09298
09908
RNIA,INGUINAL
00257 00365 01023* 01133 01907 06139 07117 07940
08304 08872
ATUS HERNIA
00139 00187* 00263* 00265* 00286 00843* 00940* 00950*
00985 00990 01021* 01034* 01057 01139* 01140* 01141*
01151 01153 01158 01159 01164 01864* 02009* 02010*
02011* 02015* 02018 02019 02031 02199* 02887* 02897*
02904* 02994* 03009 03077* 03078* 03624* 03900* 03911*
04043* 04131* 04134 04135 04147 04159 04161 04165
04202 04883 05042 05080* 05082* 05095 05098 05099
05101 05111 05115 05119 05122 05124 06019 06112

HIATUS HERNIA
CONTINUED
06114 06124 06125 06126 06130 06132 06696 06847*
06920 06942 06945 06967* 06979 06982 06983 06984
06986 06988 06994 07000 07002 07006 07074 07873
07982* 07983* 07985* 07990* 07997 08001 08005 08010
08014 08019 08021 08022 08026 08029 08032 08079
08169* 08655* 08762 09103 09136 09138 09139 09142
09147 09154 09155 09156 09157 09165 09167 09298
09908
HIRSCHSPRUNGS DISEASE
01398* 01462 02313* 02316* 02321* 03282 03289 04329*
04349 04359 04367 05059 05062 05291* 05324 05331
05336 05349 05688* 06372 06680* 07224 07236 07246
07863* 08249* 08250* 08269* 08278 08335 08341 09040
09368 09375 09407 09408 09412 09413 09417 09913
09966
HISTAMINE
00048* 00051* 00060 00084* 00252 00289* 00313* 00402
00403 00832* 00835* 00840* 00852* 00857 00870 00871
00872 00873 00882* 00936* 01090 01181* 01305* 01308*
01313* 01703* 01748* 01749* 01750* 01754* 01755* 01756*
01757* 01764* 01767* 01774* 01775* 01782 01785 01810*
01925* 02196* 02212* 02235 02709 02779* 02789* 02792*
02794* 02795* 02796* 02798* 02800* 02801* 02806 02808
02821 02892* 03000* 03180* 03701* 03748* 03749* 03760*
03761* 03762* 03763* 03764* 03772 03773 03776 03895*
04114 04172* 04256* 04258* 04702 04718* 04741* 04746*
04752* 04754 04758 04763 04765 04780* 04881 04914
04979* 05167 05215* 05228* 05229* 05266 05686* 05710*
05741* 05742* 05743* 05745* 05751* 05753* 05755* 05761*
05762* 05763* 05773 05840* 05884* 06210 06240* 06245*
06255* 06292 06677* 06690 06727 06737 06738* 06739*
06742* 06746* 06747* 06748* 06749* 06750* 06752* 06754*
06757 06758 06761 06763 06765 06841* 06904 06922
06950 06956 06958 07016* 07127* 07151 07469* 07674*
07675* 07680* 07681* 07682* 07684 07839* 08145 08165*
08168* 08175* 08176* 08206 08687 08948 09077 09078
09114 09176
HODGKINS DISEASE
00528 01290 01966 05047 05863* 06229 08150 08476
09875
HOMOCYSTINURIA
00604*
HORMONAL CONTROL IN CIRRHOSIS
00737 02605* 03424
HORMONAL CONTROL OF ABSORPTION
00812* 00815* 00823 02760* 03730* 04310* 07623* 07633*
07803
HORMONAL CONTROL OF BILIARY TRACT
05938 07495*
HORMONAL CONTROL OF GALLBLADDER
00782 00878* 02861 03648 04557* 04629* 04776* 04800*
05796* 06718* 06957 08045* 08677 09806
HORMONAL CONTROL OF GASTROINTESTINAL TRACT
00883* 01815 02187 02829* 02861 02993* 04116 04117
05778 06039 08819 08824 09195
HORMONAL CONTROL OF LARGE INTESTINE
01468* 06390 06392 07655 07663 08270
HORMONAL CONTROL OF LIVER
00099 00621 00904 00913 00915 01606 01836 01840
01849 02547 02699* 02845 02861 02862 02867 02869
03469 03698* 03808* 03816* 03819* 03824* 03874 04471
04496 04514 04697 04797* 04819 04820 04822 04823
04826 04926 05450* 05468 05476 05526* 05562 05567
05573* 05700* 05807* 05808* 05810* 05815* 05818* 05826
06786* 06791* 06794* 06798* 06800* 06804 06817 06820
07377 07381 07418* 07427 07439 07447* 07697* 07698*
07708* 07720* 07725* 07731* 07733* 07737* 07753 07756
07769 07788 07798 07801 08556* 08564 08970 08978
08982 08984 09613
HORMONAL CONTROL OF MOTILITY
00883* 05786* 05797 05803 08677 08877
HORMONAL CONTROL OF PANCREAS
00044* 00099 00193* 00836* 00860 00869 01484* 01494
01505 01512* 01521 01523 01715 01751* 01752* 01759*
02409* 02699* 02782* 02786* 02787* 02791* 02803* 02810*
02827* 03672* 03752* 03753* 03765* 03766* 03777 04380*
04389* 04399 04407 04677 04750* 04762 04875 04961
05403 05522 05737* 05739* 05740* 05747* 05752* 05761*
05774 05895 05896 06407 06772* 06773* 06775 07289
07321 07323 07665* 07690* 07691* 07692* 07695 08375*
08400*
HORMONAL CONTROL OF PEPTIC ULCERS
00410 01319* 01344 01758* 01925* 02202* 02235 02240
04289 05264 05271 09315
HORMONAL CONTROL OF SALIVARY GLANDS
00050* 01773*
HORMONAL CONTROL OF SMALL INTESTINE
00812* 01722* 02734* 04785 04941 05070 05071 05072
05203 05307 05707 05777 06722* 06723* 06727 07060
07655
HORMONAL CONTROL OF STOMACH
01701* 01807* 01809* 02196* 03144 03712* 03713* 03765*
05146* 05668* 06672* 06726* 07063 07135* 07659* 07835
09315

HORMONAL CONTROL OF STOMACH SECRETION
00046* 00049* 00054 00057 00831* 00832* 00833* 00840*
00841* 00846* 00847* 00848* 00850* 00852* 00859 00862
00870 00871 00873 00993 01310* 01314* 01358 01359
01748* 01749* 01750* 01754* 01755* 01758* 01774* 01775*
01776* 01777* 01782 01789 01790 01791 01810* 01899
02235 02703* 02779* 02780* 02785* 02788* 02792* 02794*
02796* 02800* 02801* 02806 02814 03000* 03693 03755*
03756* 03758* 03760* 03764* 03767* 03768* 03770* 03772
03773 03776 03784 04037* 04267* 04740* 04741* 04745*
04746* 04748* 04749* 04752* 04755 04756 04758 04763
05017 05018 05213* 05229* 05686* 05695* 05696* 05736*
05741* 05742* 05745* 05746* 05753* 05754* 05757* 05758*
05759* 05760* 05763* 05766 05769 05770 05771 05772
05773 05775 05776 06000* 06014 06213 06247* 06255*
06739* 06741* 06742* 06746* 06747* 06749* 06750* 06752*
06756 06757 06758 06760 06761 06767 06768 06769
06922 06956 07127* 07152 07154 07169 07665* 07675*
07676* 07679* 07682* 07683 07684 08176* 08190 08720*
08769 08948 08949 09315
HORMONAL TREATMENT OF GASTROINTESTINAL TRACT
01319* 01521 04310* 06035 07144 09444
HORMONAL TREATMENT OF LIVER DISEASE
00706 02870 03471 06867* 08618 09729
HORMONES,PANCREAS
00541* 01939* 03898* 04393 04684* 05696* 07274*
HOST REACTIONS
00683*
HYPERBARIA
09828
HYPERBILIRUBINEMIA
09009 09593 09600 09605
HYPERCALCEMIA
02206* 03401 03746* 08136 09458
HYPERCHLORHYDRIA
00974 01305* 01872* 03887* 04037* 04258* 05031 05098
05218* 05764* 05879* 06014 06018 06210 06858* 07176
07581 07582 07584 07846* 08176* 08458 08779 08780
09298 09313
HYPERGLYCEMIA
03675* 07790 08060
HYPERTENSION DIAGNOSIS,PORTAL
00942* 01862* 01930* 03355* 03936 04449* 04621 04916
05848* 05869* 06467* 06474 06601 06840* 06948 06960
07889 07897 08471 09060 09115 09733
HYPERTENSION DIAGNOSIS,RADIOLOGY IN PORTAL
01117 01889 02906* 03379* 04621 05966 06421* 07860*
08448 09059
HYPERTENSION ETIOLOGY,PORTAL
01538* 01926* 01930* 02644 03474 03502 03504 03599*
04449* 04515 04621 05607* 06474 06588 07318 07331*
07393 07405 07406 08448 08459 08640 09059 09496
09512 09521 09541 09548 09575 09707 09730
HYPERTENSION IN CHILDREN,PORTAL
06970* 08473 08729* 09496 09538 09539
HYPERTENSION PATHOLOGY,PORTAL
03379* 04621 08647 09528
HYPERTENSION TREATMENT,PORTACAVAL SHUNT IN PORTAL
00264* 00653 00672 00674 01032* 01089 01642*
HYPERTENSION TREATMENT,PORTAL
00607* 00672 00673 00674 00747 01642* 01926* 01930*
02021 02473* 02533 03119 03355* 03413 03423 03593*
03616 03949 04010 04014 04137 04140 04449* 04454*
04468 04490 04598* 04599* 04606* 04613 04623 04916
04926 05100 05451* 05455* 05603* 05617 05620 05869*
06450* 06454* 06457* 06467* 06578* 06579* 06582* 06583*
06586 06587 06594 06596 06597 06599 06601 06602
06613 06616 06679* 06970* 06973* 07331* 07340* 07341*
07347* 07353 07359 07396 07403 07405 07406 07407
07465* 07468* 07472 07473 07483 07486 07487 07984*
07996* 07998 08380 08387 08448 08457 08471 08472
08482 08632* 08633* 08635* 08640 08647 08729* 08845
09060 09130 09236 09497 09504 09528 09538 09539
09541 09547 09567 09613 09702 09703 09724
HYPERTROPHY
00498 02026 02109 04205 05138* 05174 05175 05939
06143* 06187 06192 06974* 07012* 07045 07046 07058
07228 07257 07872 08085 08098 08246 09199 09201
09256 09616 09619
HYPOCALCEMIA
01521
HYPOCHLORHYDRIA
00118* 00291* 00294* 00312* 00970 00970 00974 01182*
01872* 05418 05764* 05879* 05884* 06145* 07014* 07846*
08044* 08072
HYPOGLYCEMIA
08088 08245
HYPOGLYCEMIC AGENTS
00094 00102
HYPOKALEMIA
09458
HYPOTHERMIA
SEE ALSO COOLING TREATMENT OF BLEEDING
01043* 01356 03335* 03337* 03367* 03395 05270 05428*
05429* 05994* 06002* 06514 06770* 06771* 07009* 07075
08033* 08435* 08504 08545* 09507 09517 09828
ICTERUS
SEE JAUNDICE
ILEITIS
06315* 07181* 07903 07933 08221 08222 08223
ILEITIS,REGIONAL
01967 03891* 04302* 05277 05280 05283 06011 06316*
06319 06320 06321 07182* 07212* 08219* 08220* 08230*
08232* 09321
ILEOCECAL VALVE
00009* 00498 01292 02142 03174 03795* 06316* 06944
09926
ILEOSTOMY
00349* 01480 02144 02276* 02825* 03018 03162 04371*
08254* 09361
ILEOSTOMY COMPLICATIONS
02124* 03162 03955 08255*
ILEUM
SEE ALSO SMALL INTESTINE
00201 00202 00224 00365 00366 00370 00424 00498
00815* 00816* 00817* 00825 00882* 00968 01050 01056
01069 01249* 01253* 01271 01275 01282 01283 01285
01286 01368 01369 01375 01403* 01705 01724* 01814
02702* 02769 02776 02777 02860 02893* 02983* 03150*
03151* 03162 03167 03173 03227* 03228* 03633* 03686
03721* 03726* 03736 03756* 03795* 03804 03980 04032*
04239 04245 04250 04323 04780* 04913 05025 05084*
05183* 05197 05205 05786* 05908 06026 06027 06028
06067 06327* 07078* 07081* 07083* 07086* 07096 07109
07117 07124 07193* 07492* 07579 07594* 07624* 07627*
07673 07825 08122 08124 08133 08362 09236 09936
ILEUS
SEE ALSO LARGE INTESTINE OBSTRUCTION
SEE ALSO SMALL INTESTINE OBSTRUCTION
00120* 00499 00786* 00968 01049 01109 01187 01249*
01267 01277 01369 01396* 01413 01435 01495 01518
03058 03176 03230* 03239* 03627* 03647 03973 04107
04110 04117 04198 04209 04235 04247 04661 04976*
04986* 04993 05066 06196 06228 06625* 07091 07093
07098 07102 07103 07105 07106 07111 07241 07505
07537 07542* 07657* 07930 08118* 08127 08164 08227*
08751 08763 08767 08770 08772 08848 08895 09104
09220 09221 09229 09347 09746 09761 09785 09841
09985
ILEUS,BILIARY TRACT
00764 02182 04666 04667 08152 08156 09759
IMMUNE REACTIONS
SEE ALSO MICROORGANISMS
00198* 00537 00619 00892* 01026* 01044* 02701* 02979*
03153* 03525* 03895* 05770 05829 05830 07030* 07345*
07443* 07450* 07544* 07891 07904 09353 09835
IMMUNOGLOBULINS
01405* 01406* 01698* 01699* 02979* 03153* 03476 03525*
03533 03534 03608 03610 03914* 03958 03959 03960
03961 04021 04058 04440* 04581 04671* 04963 05284*
05370 05464 05548* 05583 05606* 05705 05849* 05870*
05991* 06315* 06380* 06394 06473 06498 06845* 07085*
07292 07354 07411 07450* 07488 07745* 07862* 07953
08226* 08402 08463 08476 08486 08583 08598* 08603*
08610 08777 09029 09030 09209 09337 09633 09665
09666 09678 09685 09980
IMMUNOLOGY
SEE ALSO MICROORGANISMS
00189* 02703* 03054 03895* 05297 07544* 08841 09180
09447
IMMUNOLOGY IN CIRRHOSIS
00739 02647 03534 03608 03610 03621 03963 04443*
04482 04483 04511 04512 04609 04963 05606* 05621
07913
IMMUNOLOGY IN ULCER DISEASE
02943 03109* 03111* 03113*
IMMUNOLOGY IN ULCERATIVE COLITIS
00533 00537 01406* 01465* 01470* 01471* 01871* 02389*
02398 02405 04379 05363* 05370 09425
IMMUNOSUPPRESSION
01593 02652 03310* 04435* 07277* 07750* 09518 09835
INFARCTION
SEE ALSO CARDIOVASCULAR SYSTEM
01088 01518 02111 02317* 02319* 03167 04487 05040
05190 07256 07565 07922 07942 08195 08484 08501
08789 08840 08853 09099 09133 09237 09262 09565
09790 09835 09857
INFECTIONS,ABDOMEN
SEE ALSO PERITONITIS
01065 01394* 01969 01979 01997 02331* 02687 02688
03179 03288 04041* 04103 04241 05199 06036 07207*
07254 07500* 08266* 08785 08806 09294 09817 09846
09852
INFILTRATION,CELLULAR
01208 06810 07565 08071 09480
INFLAMMATION
02135 02135 04975* 05135* 06374 07005 07068 07114
07182* 07183* 07239 07256 07346* 07526 07527 07895
07953 08223 08311 08316 08321 08363 08467 08589
08609 08670 08697 08698 08717* 08757 09140 09214
09224 09273 09365 09386 09389 09395 09542 09559
09647 09748

GUINAL HERNIA
00257 00365 01023* 01133 01907 06139 07117 07940
08304 08872
STRUMENTS
01911 04646 05162 05165 05680* 05767 05851* 05860*
05894 05900 05912 05962 05983* 06447* 06659 06843*
06846* 06847* 06848* 06849* 06850* 06859* 06860* 06918
06919 06927 06930 06941 06943 06946 06951 07004
07259 07855* 07674 07881 07888 07916 08034* 08658*
09031 09092 09135 09238 09834
SULIN
00046* 00059 00102 00109* 00737 00820 00855 00869
00934* 01090 01194 01235 01305* 01308* 01507 01745
01750* 01774* 01776* 01777* 01783 01827* 01828* 01925*
01939* 02129* 02235 02425 02431 02782* 02791* 02805
02814 02815 02899* 03180* 03194* 03373* 03755* 03761*
03770* 03772 03776 03777 03823* 03839 03898* 03978
04170* 04388* 04393 04394 04395 04399 04528* 04673*
04677 04680 04734 04743* 05075 05167 05229* 05400*
05407 05476 05593* 05654 05739* 05762* 05840* 06255*
06480 06761 06763 06765 06791* 06794* 07274* 07509
07648 07767 07801 06060 08088 08165* 08422* 08768
08780 08826 08948 09444 09452 09468 09809
TOLERANCE,CARBOHYDRATE
05985* 05986* 07187*
TOLERANCE,MILK
00426* 00923* 01374* 01868* 02051* 02391* 02392* 02393*
03228* 03238* 04325 04880 05293 05364 06145* 06851*
07589 08244 09425 09859 09964
TOLERANCE,SUGAR
00065 00153 01589 08237 08495
TRINSIC FACTOR
00308* 00829 01180* 07594* 07856* 07857* 08242 08746*
09176
TRINSIC FACTOR,STOMACH
00040* 00189* 00858 00859 01180* 01230 01310* 02749*
03000* 03109* 03237* 03241* 04031* 04213 05127* 05129*
05148* 05149* 05167 06707* 06748* 06751* 07031* 07190*
09180
TUSSUSCEPTION
00200* 01109 01122 01125 01256* 01280 01284 01493*
01928* 01950 02000 02119* 02150 02189 03149* 03158
04107 04113 04228* 04324 04976* 05025 05055 05323
07114 07119 07214* 07244 07248 08115* 08729* 08743*
08784 08823 08828 09207 09231 09238 09347 09382
09996
N TRANSPORT
00021* 00031 00305* 00814* 01733* 01734* 01744 01847
02126* 02320* 02325* 02768 02769 02770 02775 02776
02777 03041 03042 03260* 03367* 03600* 03669* 03674*
03722* 03736 03738 03740 03742 03745 03747* 03751*
03759* 03814* 03913* 03955 04023* 04070 04180* 04331*
04453* 04674* 04703 04711* 04713* 04716* 04835 04863*
04981* 05368 05410 05445* 05682* 05724* 05726 06701*
06708* 06743* 06784* 06791* 06813 07009* 07065 07543*
07575 07623* 07624* 07627* 07630* 07631* 07633* 07636*
07637* 07638* 07641* 07642* 07688 07765 07784 08055
08231* 08372* 08516 08874 08939 09022 09464 09737
ON ABSORPTION
00019 00026* 00039 00311* 00439 00684* 00696* 00844*
00845* 01530* 01719* 01720* 01728* 01730* 01732* 01733*
01744 02629* 02750* 02751* 02754* 02756* 02920* 03017
03077* 03240* 03241* 03301* 03588* 03592* 03723* 03724*
03726* 03733* 03739 03742 03986 04384* 04436* 04602*
04724* 04725* 04726* 04727* 04732 04733 04950 05127*
05444* 05629 05713* 05729 05733 06704* 06709 06712
06713 06714 06715 06716 07645 07646 07647 07840
08036* 08531 08941 08942 08951 09725 09845
ON EXCRETION
03676* 03915* 06606
ON METABOLISM
04678 05681* 08068
ON METABOLISM,LIVER
00082* 00096 00651 00657 00678 00684* 01530* 01568
01968 01969 02629* 02900* 02920* 03097* 03464 03588*
03592* 03843 04087 04590 04675* 04915 04950 05430*
05444* 05453* 05617 05875* 06468* 06606 06709 06816
07332* 07374 08476 08525 08631* 08941 09646
ON STORAGE DISEASES
03487* 04675* 05024
ON-DEFICIENCY ANEMIA
00308* 02057* 03017 03104* 04088 05127* 05452* 05629
05875* 06332* 07112 07120 07194* 07199 07464* 07645
07646 08036* 08177* 08865 08951 09160 09836
ON,SERUM
02534 03104* 04384* 04980* 05368 05444* 06606 08525
09725
RITABLE COLON
01402* 01805* 01865* 02893* 02894* 03016 03038* 05073
05354 06031 06101 06348 06882* 08328 09381 09393
09396 09397 09950
SCHEMIA
02130* 02185 02188 02997* 03011 03103* 03148* 03251*
03255* 03257* 04353 04460* 04695* 04705 04782 04783
04995 05029 05040 05065 05183* 05190 05201 05225*
05315* 05358 05445* 05518 05528* 05536* 05559 05562

ISCHEMIA
CONTINUED
06023 06070 06472 06514 06545 06547 06637 06724*
06817 06896 07084* 07105 07122 07123 07126* 07141
07264* 07366 07375 07403 07423* 07542* 07553 07556
07763 07887 08179 08451 08452 08484 08489 08551*
08647 08717* 08728* 08738* 08792 08831 08852 08853
08867 08954 09236 09428 09507 09518 09706 09786
09828

JAUNDICE
01615* 01616* 02114* 02503 02564 02567 02604* 02610
02633* 02884* 03380* 03381* 03385 03590* 04111 04566*
05440* 06569 07062 07311 07373 07446* 07464* 07467*
07957 08121 08463 08500 08511 08538 08705 09236
09453 09532 09563 09608 09657 09715 09779
JAUNDICE DIAGNOSIS
00111* 00171 00941* 01009 01539* 01555 01852* 01860*
01862* 01863* 01886 01891 02665* 03553 03885* 03893*
03902* 03919* 03954 04005 04009 04012 04020 04443*
04471 04484 04501 04562* 04615 04837* 04839* 04843*
04852* 04873 04886 04902 04904 04915 04927 04964
04965 04966 05487 05515 05525 05822* 05836* 05837*
05858* 05870* 05882* 05941 05947 05955 05959 05960
05979 06465* 06479 06492 06566 06568 06643 06651
06653 06660 06861* 06867* 06914 06923 06928 06931
06932 06937 06939 07349* 07363 07377 07386 07387
07417 07490* 07516 07524 07840 07841* 07883 07890
07893 07898 07918 07928 07938 07943 07953 07978
08436* 08466 08474 09029 09079 09100 09490 09533
09607 09620 09637
JAUNDICE DIAGNOSIS,RADIOLOGY IN
00996 02964 04842* 05880* 06962 09558
JAUNDICE EPIDEMIOLOGY
00637 05515 08428* 08716
JAUNDICE ETIOLOGY
00596* 00603* 00633 00637 00643 00651 00652 00661
00697* 00701 00702 00782 00913 00964 01555 01577
01603 01608 01618* 01852* 01886 02488 02492 02526
02570 02578* 02608 02666* 02977* 03359* 03389 03555
03827 03893* 03894* 04018 04033* 04067 04237 04462
04463 04484 04486 04501 04550 04657 04659 04873
04886 05152 05153 05186* 05479 05515 05525 05557
05837* 05959 05960 06448* 06461* 06462* 06465* 06479
06484 06486 06492 06525 06537* 06544 06545 06560
06609 06619* 06660 06962 07058 07265 07349* 07363
07364 07371 07377 07381 07385 07386 07387 07402
07417 07425 07426 07427 07445* 07449* 07461 07490*
07516 07518 07521 07742* 07743* 07809 07943 07978
08454 08462 08511 08527 08538 08564 08586 08716
08716 09430 09490 09515 09531 09533 09549 09558
09603 09645
JAUNDICE IN CHILDREN
06479 06484 06488 07058 07524 08227* 08426* 08478
08507 09524 09617
JAUNDICE PATHOLOGY
01646* 02488 03827 04471 05504 05947 06781* 07445*
07499* 07809 08095 08462 08538 08622 08716 09525
09549 09558
JAUNDICE SYNDROMES
00643 00648 00738 01665* 02470* 02488 02503 02546
02666* 03366* 03390 03528* 03827 04458* 04499 04557*
04652 05431* 05432* 05435* 05487 06501 06861* 06937
07364 07376 07399 07418* 07480 07527 07893 08095
08411 08436* 08466 08477 08478 08511 09492 09515
09584 09620 09629
JAUNDICE TREATMENT
02495 02546 03359* 04439* 04463 04471 04501 05485
05487 05576* 05646* 06449* 06479 06492 06620* 07348*
07367 07371 07520 07538 08432* 08436* 08530 08625
08716 09549 09558 09637 09645
JAUNDICE,BIOCHEMICAL DIAGNOSIS OF
00145 01009 02916* 03894* 05515 05870* 05881* 05947
06861* 07337* 07837* 08462
JAUNDICE,CLINICAL STUDIES OF
00596* 00633 00637 00643 00679 02599*
JAUNDICE,DISEASES ASSOCIATED WITH
00600* 00698 00916 02488 03380* 03381* 03893* 04445*
05393 05435* 07058 07348* 07349* 07866* 07890 08095
08477 08478 08511 08521 09576 09675
JAUNDICE,NEONATAL
00636 00637 02666* 03902* 04041* 04439* 04462 04463
04501 04563* 04564* 04572 04574 05595* 06462* 06479
06492 06505 06552 07369 07371 07742* 07743* 07809
07841* 07928 08453 08455 08462 08466 08508 08511
09600 09605 09620
JAUNDICE,OBSTRUCTIVE
00171 00603* 00670 00782 00996 01113 01247* 01539*
01576 01577 01603 01656* 01658* 01684 01690 01863*
02492 02890* 02916* 02976* 03003 03356* 03555 03631*
03653 03827 03834 03893* 03894* 03902* 03914* 03954
03963 04009 04067 04443* 04445* 04463 04465 04471
04496 04507 04527* 04538 04542 04543 04550 04564*
04647 04650 04657 04839* 04845* 04886 04905 05485
05487 05515 05521 05573* 05646* 05822* 05837* 05870*
05881* 05960 06087 06418* 06449* 06465* 06466* 06483

JAUNDICE,OBSTRUCTIVE
CONTINUED
06505 06590 06621* 06645 06660 06781* 06861* 07358
07360 07385 07387 07415 07417 07426 07449* 07507
07516 07517 07536 07778 07837* 07890 07904 07928
07953 07978 08436* 08507 08656* 08716 09046 09079
09100 09432 09491 09494 09515 09525 09558 09576
09773 09784
JAUNDICE,SERUM BILE PIGMENTS IN
00596* 00756* 01618* 01677 03380* 03381* 03827 04501
04873 05494 07402 07809 08511
JEJUNUM
SEE ALSO ABSORPTION
SEE ALSO REGIONAL ENTERITIS
SEE ALSO SMALL INTESTINE
00165 00179 00186* 00223 00326 00367 00371 00400
00411 00436 00437 00439 00780 00785* 00787* 00814*
00815* 00816* 00817* 00818* 00823 00825 00841* 00921*
00922* 00923* 00925* 00972 01029* 01038* 01048* 01079
01080 01082 01121 01122 01177* 01246* 01249* 01253*
01255* 01260* 01267 01269 01270 01273 01274 01279
01284 01315* 01333 01723* 01724* 01736* 01870* 01923*
02136 02143 02150 02190 02282* 02287 02289 02698*
02702* 02713 02731* 02777 02829* 02888* 02913* 02914*
02980* 02986* 02989* 03110* 03158 03167 03183* 03228*
03234* 03235* 03238* 03242* 03243* 03246* 03248 03686
03726* 03729* 03731* 03756* 03792* 03891* 03980 03989
04002 04032* 04042* 04046* 04051* 04194 04203 04227*
04230* 04236 04239 04250 04303 04319 04322 04324
04686* 04688* 04695* 04717* 04723* 05159 05160 05189
05205 05217* 05233* 05246 05295 05712* 05716* 05726
05787* 05851* 05886 05887 06045 06167 06216* 06220*
06251* 06304 06325 06687 06701* 06735* 07044 07079*
07081* 07082* 07085* 07086* 07087* 07101 07122 07123
07124 07196 07520 07595* 07624* 07627* 07812* 07814*
08061 08074 08113* 08126 08134 08145 08155 08226*
08233* 08469 08652 08908 09164 09169 09177 09207
09235 09244 09755
JEJUNUM MORPHOLOGY
05691*

KERNICTERUS
03380* 08453 08455 08508 09620
KETOSIS
02572
KIDNEY
02149 02512 02658 02738 03422 03587* 03731* 04022*
04445* 04447* 04522* 04594 04683* 05454* 05458 05480
05496 05533* 05922 06034 06180 06290 06502 06514
06569 06611 06614 06817 07085* 07179 07218 07257
07310 07415 07466* 07488 07527 07543* 07649 07655
07906 07926 07953 08539* 08638* 08652 08746* 08761
08771 08874 08972 08994 08998 09014 09105 09399
09701 09712 09722 09736 09738 09756 09867
KIDNEY ELECTROLYTE SECRETION
00090* 03774 04445* 08055 08227*
KININS
SEE ALSO BRADYKININ
04116 04117 08200
KWASHIORKOR
00428* 01048* 02581* 02978* 03244* 04062 04980* 05008
05985* 05986* 05997* 06004* 06033 07276* 08891 09997

LACTASE
00129* 00428* 00435* 00923* 01374* 02281* 02391* 02392*
02393* 02456 02902* 02913* 03228* 03238* 03244* 03245*
04227* 04325 04880 05386* 05986* 06669* 06851* 08241
08244
LACTIC ACID ABSORPTION
02771 08938
LACTOSE ABSORPTION
00129* 00428* 00435* 00923* 02051* 02748* 02913* 03228*
03244* 03245* 04044* 04227* 04312 04325 05986* 08244
LARGE INTESTINE
SEE ALSO CECUM
SEE ALSO COLITIS
SEE ALSO DIARRHEA
SEE ALSO DYSENTERY
SEE ALSO ENTEROCOLITIS
SEE ALSO ILEOCECAL VALVE
SEE ALSO ILEUS
SEE ALSO INTUSSUSCEPTION
SEE ALSO RECTUM
SEE ALSO SIGMOID
00081 00081 00262* 00441* 00442* 00462 00500 00502
00505 00508 00509 00527 00528 01053 01871* 02893*
03061 03260* 03300* 03466 04023* 05073 06031 06348
06680* 07826
LARGE INTESTINE ABSORPTION
00022* 00035 00798 00815* 00816* 00817* 00828 00951*
01403* 01412 01431 01729* 01740 01742 01746 02320*
02757* 02758* 02767 02777 02778 02883* 03720* 03730*
04023* 05717* 05724* 05726 05731 05733 06379* 06711
07216* 07575 07628* 07633* 08160 08255* 08306 08934
08944 09074 09327 09916
LARGE INTESTINE ANGIOGRAPHY
00420* 00442* 02315* 02353 06887 06888 06889 09420
09427
LARGE INTESTINE ANOMALIES
00245 00471 00511 01429 01434 01449 01455 01799*
01900 01991 02114* 02303* 02333 02334 03287 04228*
04280 04359 05323 05324 05331 05336 06335 06362
06372 07211* 07219 07224 07236 07246 07250 08026
08092 08249* 08250* 08251* 08287 08319 08331 08348
08901 08916 09040 09146 09219 09345 09358 09368
09380 09388
LARGE INTESTINE ATRESIA
06335 08287
LARGE INTESTINE BIOCHEMISTRY
01405* 01406* 02745 02812 03254* 03669* 03677 03778
03780 03781 04023* 04672* 04685* 04707 05362* 05380
06675* 06678* 07205* 07205* 07636* 07637* 07638* 07667*
07820 08258* 08263*
LARGE INTESTINE BIOPSY
00422* 01865* 01880 02935 04330* 04929 07236 08246
08263* 08330 08359* 08365 09363 09889 09920
LARGE INTESTINE CANCER
00106* 00114* 00259 00448* 00454* 00460 00464 00465
00534 00948* 00963 01017* 01020* 01033* 01047* 01128
01380* 01383* 01396* 01403* 01426 01427 01447 01457
01936* 01941* 01969 02290 02298* 02306* 02307* 02309*
02310* 02312* 02329* 02332* 02336 02340 02369 02373
02379 02387* 02390* 02394* 02395* 02901* 02975* 03053
03254* 03256* 03270 03272 03276 03292 03296* 03297*
03941 03997 03998 04053 04082 04327* 04328* 04332*
04333* 04334* 04348 04351 04352 04355 04357 04361
04362 04381* 04867 04999 05310* 05311* 05313* 05338
05339 05346 05356 05865* 05943 06049 06061 06330*
06357 06361 06363 06367 06882* 06949 07207* 07208*
07209* 07213* 07221 07226 07232 07234 07237 07238
07247 07258 07273 07557 08220* 08252* 08253* 08258*
08260* 08261* 08262* 08265* 08271 08274 08296 08300
08302 08305 08309 08310 08314 08318 08320 08337
08355 08368 08722* 08742* 09101 09353 09357 09377
09384 09391 09415 09577 09832 09889 09892 09897
09922
LARGE INTESTINE CARCINOGENESIS
01437 02356 05357 05359 07229
LARGE INTESTINE CIRCULATION
00798 02298* 02319* 02327* 03263* 04353 04373* 05029
05315* 05317* 05337 05355 05358 05360* 06386 06887
07254 07557 08301 09133
LARGE INTESTINE CYSTS
04338 08330
LARGE INTESTINE DEVELOPMENT
00803 06335 08287 09345
LARGE INTESTINE DIAGNOSIS
00106* 00120* 00121* 00131* 00254 00420* 00422* 00442*
00454* 00455* 00458 00467 00501 00506 00928* 00936*
00957 00984 01091 01392* 01397* 01400* 01411 01428
01436 01441 01442 01443 01457 01460 01801* 01805*
01865* 01880 01882 01883 02299* 02300* 02323* 02333
02341 02350 02372 02382* 02387* 02901* 02935 02949
03156 03161 03251* 03252* 03253* 03256* 03257* 03269
03270 03290 03296* 03304 03920* 03931 03941 03946
03997 03998 03999 04330* 04339 04356 04364 04775*
04860* 04867 04896 04929 04931 04970* 04971* 05060
05315* 05318* 05319* 05341 05346 05354 05356 05357
05358 05359 05688* 05690* 05845* 05943 05978 06042
06171 06326* 06329* 06334 06347 06348 06349 06351
06352 06354 06378* 06882* 06887 06888 06889 06921
07211* 07215* 07220 07222 07223 07226 07236 07254
07258 07863* 07874 07917 07952 07961 08065 08153
08249* 08259* 08259* 08264* 08277 08281 08284 08289
08300 08305 08323 08330 08332 08353 08354 08355
08359* 08363 08370 08784 09075 09088 09104 09108
09122 09123 09238 09355 09363 09366 09382 09385
09389 09399 09402 09404 09405 09407 09420 09426
09427 09889 09950 09996
LARGE INTESTINE DIAGNOSIS,RADIOLOGY IN
08164
LARGE INTESTINE DISEASE EPIDEMIOLOGY
00183* 05347 06334 09937 09940
LARGE INTESTINE DISEASE ETIOLOGY
08250* 09395
LARGE INTESTINE DISEASES
00122* 00452* 00467 00477 00478 00479 00480 00481
00482 00483 00484 00485 00486 00487 00488 00489
00490 00491 00492 00493 00494 00495 00521 00620
01087 01418 01422 02400 02894* 02895* 02901* 03257*
03300* 04338 04970* 05059 05061 05208 05352 05360*
06028 06333* 06874* 07222 07230 07744* 07895 08160
08289 08295 08299 08313 08328 08330 08339 08342
08347 08354 08371 08892 09122 09366 09369 09378
09381 09396 09397 09402 09411 09416 09417 09426
09919 09933
LARGE INTESTINE DISEASES,BIOCHEMICAL DIAGNOSIS OF
00149

LARGE INTESTINE DIVERTICULA
00187* 01402* 02314* 02318* 02322* 03067 03277 03283
03997 04335* 04341 04360 04368 05352 06339 06342
06345 06921 07100 07227 07233 07270 08153 08284
08303 08354 09365 09402 09913
LARGE INTESTINE DIVERTICULITIS
00449* 00451* 00465 00946* 01414 01459 01805* 01882
02010* 02299* 02311* 02314* 02372* 02332* 02349 02370
02901* 03161 03261* 03264* 03286 04337* 04341 04342
04344 04358 04360 04867 05352 05353 06339 06342
06345 06354 06887 07238 08282 08283 08284 08332
09367 09369 09370 09402 09403 09891
LARGE INTESTINE ENDOSCOPY
00458 01400* 01428 01865* 02387* 03265* 03304 03920*
04364 04860* 04929 04931 05367 06949 07254 07265
08330 08363 08724* 09035 09427
LARGE INTESTINE ENZYMES
00469 02003 02051* 02812 03254* 03677 07261* 07820
08258* 08263*
LARGE INTESTINE FISTULAS
00199* 01127 01655* 01680 01681 02419 03008 03155
04032* 04068 04344 04346 05027 05935 07183* 07218
07565 08065 08283 08309 08417 09386 09410
LARGE INTESTINE HISTOLOGY
03700* 03888* 04368 05367 05688* 05706 05980* 06389
06694 07210* 07215* 07261* 08160 08263* 08280 08317
08336 08371 09227
LARGE INTESTINE IMMUNOLOGY
00537 00803 01468* 01871* 02384* 02394* 02701* 03019
04369* 05363* 05370 06314* 06360 06381* 06675* 07261*
07263* 07264* 09425
LARGE INTESTINE IN CHILDREN
00472 00511 01434 01455 02303* 02305* 02313* 02348
02366 04228* 04349 04359 04364 05059 05317* 05318*
05324 05331 05336 06081 06335 06349 06362 07211*
07224 07234 07246 07744* 08249* 08278 08287 08331
08335 08348 08350 08901 09108 09342 09414 09417
LARGE INTESTINE INJURIES
02315* 03263* 05029 05975 06352 07565 08256* 08353
08822 09349 09856
LARGE INTESTINE METABOLISM
07817* 07820
LARGE INTESTINE MICROORGANISMS
00369 00790* 00933* 01393* 01694* 01735* 02883* 03892*
04336* 04338 04345 04987* 04990* 05007 05363* 05605*
06147* 06381* 06390 06825* 07210* 07617 07820 08344
08356 08360* 08721* 08898 08904 09024 09025 09937
09938 09939 09940 09941 09942 09943 09944 09945
09946 09977 09983
LARGE INTESTINE MORPHOLOGY
00497 01706* 01707* 01802* 02327* 02330* 03700* 03997
03999 04280 04378 05690* 05783* 05989* 06302 06680*
06688 08253* 08276 08280 08932 09394 09412
LARGE INTESTINE MOTILITY
00071* 00074* 00079* 00186* 00889 00984 01381* 01402*
01410* 01798* 01801* 01802* 01805* 01808* 01809* 01810*
01882 02319* 02323* 02334 02893* 02894* 02972* 03258*
03304 03793* 03799 03805 03888* 04086 04373* 04707
04770* 04775* 04777* 04789 05058 05059 05062 05208
05349 05352 05354 05783* 05788* 05794* 06101 06302
06327* 06719* 06725* 07589 08276 08306 08328 08335
08342 08356 08371 08851 09075 09229 09396 09397
09950
LARGE INTESTINE MUCOSA
00310* 00473 01027* 01080 01144 01378* 01451 01478
01706* 01707* 01802* 01910 02302* 02308* 02318* 02319*
02320* 02343 02358 02384* 02388* 02390* 02745 02777
02935 02983* 03253* 03254* 03304 03669* 03740 03778
03780 03781 03888* 04330* 04335* 04343 04345 04360
04368 04703 04970* 05352 05354 05359 05360* 05362*
05363* 05367 05376 05696* 05697* 05726 05980* 06318
06326* 06336 06337 06356 06376* 06378* 06379* 06386
06389 06390 06394 06675* 06678* 06688 06694 07183*
07210* 07216* 07222 07268 07269 07270 07636* 07637*
07638* 07673 08160 08255* 08256* 08259* 08299 08328
08781 08934 09025 09394 09410 09412 09920
LARGE INTESTINE NEOPLASMS
00450* 00469 00503 00509 00956 01380* 02356 02372
03067 03269 03281 05332 05357 06026 06171 06368
06889 07256 08285 08315 08316 08324 08337 09346
09352 09389 09394 09419 09893
LARGE INTESTINE NEOPLASMS,BENIGN
00441* 01083 01437 02153 02305* 02343 02361 03254*
04334* 04340 04352 04354 04356 04361 04366 05060
05319* 06330* 06334 06337 06357 07223 07229 07230
07231 07234 07237 07242 08259* 08281 08308 08323
08336 08347 09395
LARGE INTESTINE NEOPLASMS,MALIGNANT
00464 00474 00504 00508 00530 01029* 01083 01096
01378* 01391* 01392* 01414 01416 01418 01420 01432
01437 02183 02320* 02348 02355 02359 02362 03254*
03260* 03262* 04065 04333* 04334* 04350 04352 05319*
05359 06027 06326* 06330* 06347 06363 06370 07068
07221 07226 07232 07234 07240 07242 07260 08253*
08265* 08305 08355 09391 09415 09889
LARGE INTESTINE NEOPLASMS,VILLOUS
00446* 01049 01144 02320* 02325* 02337 04334* 05319*
06330* 06357 06949 07242 08317 09399
LARGE INTESTINE OBSTRUCTION
00120* 00472 00503 00508 01091 01124 01125 01387*
01395* 01413 01415 01434 01435 01452 01454 01457
01493* 02300* 02327* 02334 02356 02378 03037 03038*
03155 03170 03280 03297* 03997 04101 04107 04328*
04335* 04339 04351 04993 04996 05278 05323 05352
05353 06026 06063 06090 06343 06351 06355 06365
06372 07183* 07241 07244 07256 07565 07579 07863*
08164 08249* 08287 08335 08341 08347 08350 08784
08916 09104 09122 09229 09347 09375 09384 09385
09391 09406 09841 09847 09866 09890 09895 09933
09985
LARGE INTESTINE PATHOLOGY
00257 00442* 00459 00803 00958 01377* 01378* 01415
01416 01422 01451 01453 01458 01865* 02003 02299*
02301* 02302* 02326* 02327* 02330* 02357 02388* 03251*
04334* 04335* 04341 04354 04360 04970* 05278 05316*
05333 05335 05337 05341 05342 05358 06042 06062
06063 06317 06334 06351 06354 06356 06384 06389
07212* 07224 07236 07238 07251 07252 07269 07270
07271 07557 07895 08160 08250* 08253* 08256* 08263*
08280 08299 08317 08324 08327 08342 08347 08354
08370 08371 08728* 08904 09040 09075 09227 09346
09356 09359 09366 09367 09369 09370 09378 09381
09389 09393 09395 09396 09397 09407 09427 09889
09890
LARGE INTESTINE PERFORATION
00245 00472 01383* 01387* 01398* 01414 01428 01436
02350 02357 02996* 03264* 03267 03291 03297* 03700*
04027* 05317* 05318* 05845* 05975 06329* 06349 06352
07207* 07233 07240 07256 07573 08150 08266* 08283
08284 08304 08309 08316 08832 08901 09035 09088
09349 09363 09370 09391 09876
LARGE INTESTINE PERFUSION
00035 02777 07633* 07636* 07637* 07638*
LARGE INTESTINE POLYPS
00224 00349* 00448* 00450* 00500 00501 00502 00503
00504 00505 00507 00509 00510 00530 00948* 00956
01379* 01416 01437 01445 01453 02305* 02312* 02343
02344 02361 02901* 02995* 03254* 04334* 04340 04352
04361 05319* 05335 05346 05357 05359 06049 06328*
06330* 06357 06361 06882* 06921 06949 07208* 07213*
07229 07230 07231 07235 07237 07244 07249 07268
07557 07917 08259* 08271 08315 08317 08336 08337
08368 08729* 09346 09352 09357 09408 09419 09823
09897
LARGE INTESTINE PSEUDOPOLYPS
03253*
LARGE INTESTINE RADIOLOGY
00120* 00121* 00131* 00254 00420* 00442* 00450* 00454*
00455* 00456 00471 00472 00474 00501 00507 00507
00508 01053 01091 01124 01388* 01389* 01411 01431
01432 01442 01443 01453 01454 01801* 01805* 02299*
02300* 02315* 02341 02350 02382* 02383* 02387* 02742
02949 03156 03252* 03253* 03269 03278 03296* 03304
03888* 03931 03941 03997 03998 03999 04344 04350
04366 04368 04370* 04378 04770* 04775* 04867 04971*
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06349 06351 06378* 06887 06921 06944 07226 07895
07917 08065 08249* 08276 08284 08317 08330 08337
08348 08354 08363 08369 08370 09075 09088 09104
09356 09366 09385 09395 09402 09420 09426 09427
09996
LARGE INTESTINE SECRETION
02003 02325* 04343 07448* 07636* 07637* 07638*
LARGE INTESTINE SURGERY
00207 00349* 00441* 00445* 00446* 00449* 00455* 00465
00478 00479 00480 00481 00483 00485 00486 00487
00488 00491 00492 00493 00494 00495 00500 00512
00520 00523 00939* 01047* 01058 01091 01379* 01381*
01387* 01389* 01392* 01394* 01396* 01397* 01400* 01403*
01411 01422 01434 01437 01449 01450 01475 01940*
02276* 02300* 02301* 02303* 02306* 02307* 02308* 02309*
02310* 02311* 02313* 02314* 02316* 02318* 02321* 02322*
02326* 02329* 02330* 02331* 02332* 02334 02340 02343
02347 02362 02374 02387* 02390* 02395* 02399 02401
02473* 02637* 02996* 03026 03091 03155 03251* 03252*
03253* 03258* 03261* 03264* 03276 03278 03280 03282
03289 03297* 03306 03955 04095 04096 04327* 04332*
04336* 04337* 04339 04340 04341 04342 04344 04346
04347 04348 04349 04350 04351 04354 04355 04358
04360 04366 04370* 04371* 04928 04975* 05030 05310*
05311* 05313* 05319* 05320* 05323 05324 05337 05338
05339 05340 05343 05345 05347 05351 06049 06061
06067 06137 06318 06327* 06328* 06329* 06331* 06337
06338 06339 06340 06343 06344 06345 06346 06348
06352 06354 06355 06357 06363 06365 06366 06367
06368 06369 06371 06372 06374 06385 06393 06605
06882* 07078* 07098 07099 07183* 07205* 07208* 07209*
07211* 07212* 07217* 07218 07219 07220 07227 07231
07232 07234 07238 07239 07240 07241 07242 07244
07246 07247 07248 07250 07251 07252 07256 07259

LARGE INTESTINE SURGERY
CONTINUED
07267 07273 07565 07817* 08030 08087 08144 08163
08249* 08251* 08252* 08254* 08255* 08265* 08269* 08271
08272 08274 08277 08278 08281 08282 08284 08291
08292 08296 08301 08307 08308 08311 08312 08313
08314 08315 08316 08317 08319 08323 08327 08332
08335 08337 08341 08353 08355 08357 08361 08362
08369 08370 08371 08717* 08743* 08784 08851 08901
08916 09146 09151 09158 09187 09344 09348 09349
09350 09355 09358 09361 09363 09367 09368 09369
09370 09374 09375 09378 09379 09382 09383 09384
09385 09389 09395 09398 09409 09413 09416 09417
09418 09421 09427 09428 09764 09847 09857 09868
09888 09893 09901 09926 09930 09985
LARGE INTESTINE TRANSPLANTATION
00473 01144 01147 01151 02016* 02042 04138 04141
04156 05051 06131 06137 06990 07989* 08815 09133
09151 09158 09164
LARGE INTESTINE TRAUMA
00164 00512 01435 01436 02996* 05845* 07256 08026
08353 09398 09404
LARGE INTESTINE TREATMENT
03268 03270 06334 06354 06373 07211* 07223 08251*
08267* 08286 08289 08328 08332 08743* 08851 09343
09359 09366 09367 09384 09395 09398 09413 09857
LARGE INTESTINE ULCERS
02326* 02327* 03161 03257* 04970* 06354 07131* 07210*
08333 09378
LARGE INTESTINE ULTRASTRUCTURE
04363 05367 05690* 06688 07210* 08246 08299 08934
09357 09920
LARGE INTESTINE VOLVULUS
00445* 01125 01463 01493* 02300* 02334 02351 03268
04339 04351 05311* 06026 06343 07241 07259 08251*
08264* 08357 09383 09403 09409
LARGE INTESTINE,AGE EFFECTS ON
01402* 03252* 03273 03277 05058 05338 05358 07247
08264*
LARGE INTESTINE,CLINICAL STUDIES OF
00457 00467 00475 00497 00516 00958 01458 01463
01740 02311* 02326* 02330* 02341
LARGE INTESTINE,DISEASES ASSOCIATED WITH
00062* 00501 00502 00503 00504 00505 00509 00510
03061 09890
LARGE INTESTINE,DRUG EFFECTS ON
04775* 07216* 07663 07820 08721* 08847
LARGE INTESTINE,DRUG TREATMENT OF
07272 08321 08333
LARGE INTESTINE,HORMONAL CONTROL OF
01468* 06390 06392 07655 07663 08270
LARGE INTESTINE,NERVOUS CONTROL OF
00889 02302* 02316* 02321* 02323* 02333 02334 02894*
02895* 03805 04329* 04363 04707 05688* 05706 05788*
05794* 06390 06680* 06719* 07663 07863* 08249* 08264*
08267* 08270 08335 08342 09040 09229 09397 09407
09412 09913
LARGE INTESTINE,TOXIC EFFECTS ON
00471 01257* 01469* 02747 02983* 03263* 04378 04735
05058 05317* 05318* 06010 06336 07210* 07216* 08256*
08259* 08302 09040
LAXATIVES
00933* 02302* 04047* 06098 08356
LEAD
06727
LEIOMYOBLASTOMA
09170 09172
LEIOMYOMA
00280 02038 02184 02345 03126 05128* 06134 06223
06981 07073 08146 08147 08148 08151 08155 08336
09208 09216 09387 09905
LEIOMYOSARCOMA
01265 01417 02359 03281 04225 05128* 06223 06225
07067 07071 07092 08149
LEPTOSPIROSIS
04090 05046 09634 09637 09849
LIPASE
07306* 07307* 07701* 07781
LIPASE SECRETION,PANCREAS
00027* 00836* 00860 00930* 01371* 01489* 01759* 02407*
02729* 02784* 02804 03699* 03983 04080 04268* 04389*
04921 05382* 05779* 05850* 05932 06423* 06435 06773*
07278* 07651 07954 08239 08415 09210
LIPID ABSORPTION
00027* 00028* 00029 00034 00037 00431* 00813* 00821
00826 00830 00890 00911 01191 01260* 01372* 01723*
01731* 01736* 02087 02172 02279* 02289 02291 02392*
02483* 02603* 02636* 02700* 02707 02757* 02760* 02762*
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03741 03744 03802 03886* 04000 04080 04124 04166*
04178* 04240 04242 04308* 04673* 04681 04719* 04722*
04728* 04729* 04854* 04939 04949 05031 05206 05296
05300 05304 05361* 05382* 05666* 05676 05725* 05834
05864* 05910 06029 06322 06698* 06705* 06713 06822
06881* 06897 07079* 07090 07112 07188* 07191* 07193*
07195 07197 07202 07290 07439 07492* 07629* 07634*
LIPID ABSORPTION
CONTINUED
07635* 07640* 07643* 07651 07653 07654 07688 07726*
07803 07919 08116* 08229* 08230* 08236 08239 08243
08377 08548* 08938 08945 08991 08993 09017 09043
09126 09218 09329 09332 09535 09631 09716 09920
LIPID DIGESTION
00034 00066 00890 00895* 00898 01731* 01736* 01738*
02392* 02603* 02707 02822 03246* 03633* 03802 03886*
04080 05676 05779* 05796* 07290 07643* 07651 07654
07688 07783
LIPID METABOLISM
01738* 02087 02636* 02700* 02710 02711 02728 02882
03573 03710* 04058 04061 04080 04294 04382* 04673*
04949 05077 05593* 05673* 05841* 06003* 06007* 06406
06735* 06802 06826* 06827* 07188* 07629* 07634* 07640*
07643* 07688 07727* 07752* 07781 07786 07803 08228*
08668 08686 08688 08830 08978 09020 09127 09332
09867
LIPID METABOLISM,LIVER
00037 00171 00595* 00612 00647 00650 00685* 00895*
00898 00911 00912 01534* 01542* 01544* 01556 01562
01588 01605 01637* 01743 01840 02540 02601* 02636*
02673 02845 02851 03388* 03427 03434 03476 03492
03629* 03696* 03813* 03819* 03823* 03824* 03826 03828
03830 03836 03842 03850 03852 03860 03861 03862
03867 03877 04452* 04508 04534 04535 04548 04682
04683* 04793* 04803* 04815 04816 04825 04827 04828
05144* 05308 05431* 05432* 05433* 05471 05528* 05537*
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07785 07786 08447 08540* 08579 08637* 08955 08957
08971 08975 08987 08989 09010 09011 09597 09716
09805
LIPIDS
SEE ALSO ABSORPTION
00024* 00029 00034 01525 01637* 01743 02000 02673
02874 03703* 03881 04029* 04458* 04859* 05530* 05542*
05850* 05861* 06236* 06700* 07593* 08233*
LIPOMA
08008 08147 08323 08336
LIPOPROTEINS
05530* 05551* 05672*
LITHIASIS,PANCREAS
00550
LIVER
SEE ALSO AMEBIASIS
SEE ALSO BILE
SEE ALSO BILIARY TRACT
SEE ALSO BLOOD COAGULATION
SEE ALSO CHOLESTASIS
SEE ALSO CIRRHOSIS
SEE ALSO FOLIC ACID
SEE ALSO GLYCOGEN STORAGE DISEASE
SEE ALSO HEMOSIDEROSIS
SEE ALSO HEPATITIS
SEE ALSO PARASITES AND PARASITIC DISEASES
SEE ALSO PORPHYRIA
SEE ALSO SCHISTOSOMIASIS
SEE ALSO SULFOBROMOPHTHALEIN
SEE ALSO VITAMINS
02738 03469 03473 03513 03885* 04694* 05669* 06876*
LIVER ABSCESSES
00206 00666 00667 00677 01564 01874* 02479* 02485*
02485* 02530 02560 02891* 02991* 03363* 03372* 03375*
03393 03417 04470 04943 04973* 05443* 05469 05477
05498 05869* 06100 06458* 06874* 07400 07414 07877
08222 08456 08500 08570 08571 08572 08582 08583
08584 08585 08756 09080 09125 09630 09644 09990
LIVER ABSCESSES,DIAGNOSIS OF
03363*
LIVER AMINO ACID METABOLISM
05825 06800* 06808 07698*
LIVER AMMONIA EXCRETION
00085* 00100 00597* 00601* 00671 00676 01528* 02509
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03612 04014 04064 04491 04509 04546 04607* 04608*
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05881* 06449* 06476 06524 08445 09047 09508 09553
09561
LIVER ANGIOGRAPHY
00112* 00116* 00173 00175 00627 01117 01554 02500
02542 03376* 03486* 04701 04900 04938 04953 05516
05848* 05869* 05898 05966 06493 06840* 06842* 06864*
06876* 06884 06893 06894 06959 06965 07550* 07889
07922 07926 08433* 08647 09026 09038 09053 09087
09519 09599 09705 09720 09730 09733
LIVER ANOMALIES
00689 01693 02568 03466 03470 03488* 04471 05492
06551 06938 07393 07413 07908 07942 08434* 08494
08526 09544 09548 09563 09603

ER BIOCHEMISTRY
00037 00082* 00083* 00084* 00085* 00088* 00091* 00094
00095 00097 00099 00100 00102 00145 00597* 00601*
00613 00614 00615 00617 00647 00657 00658 00676
00682* 00683* 00684* 00685* 00715 00730* 00732* 00742
00891* 00892* 00893* 00894* 00898 00899 00900 00901
00904 00907 00909 00910 00912 00913 00914 00916
00918 01008 01235 01528* 01541* 01542* 01547* 01563
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03879 03880 04064 04320 04439* 04446* 04451* 04469
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05818* 05819* 05820* 05821* 05823* 05824 05825 05827
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08577 08587 08626 08636* 08643 08652 08657* 08921
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08982 08983 08984 08985 08986 08988 08992 08994
08996 08997 08998 09000 09001 09002 09003 09004
09006 09008 09010 09011 09013 09014 09015 09016
09047 09086 09105 09508 09553 09555 09556 09557
09563 09581 09597 09598 09601 09625 09637 09641
09648 09649 09651 09653 09654 09656 09673 09675
09676 09677 09698 09719 09972
ER BIOPSY
00112* 00146 00156 00603* 00623 00646 00668 00697*
00700 00729* 00738 00782 00937* 00941* 00947* 00981
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01552* 01579 01592 01597 01603 01608 01612* 01675
01860* 01880 02499 02500 02540 02562 02570 02635*
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03883* 03902* 03951 04005 04008 04470 04494 04587
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05506 05516 05582 05612* 05693* 05694* 05892 05937
05948 05960 05972 05979 06387 06452* 06465* 06469*
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06948 07021* 07187* 07332* 07334* 07335* 07338* 07342*
07377 07420* 07427 07445* 07490* 07841* 07934 07941
07957 07971 08088 08427* 08430* 08431* 08447 08454
08474 08486 08491 08500 08502 08507 08523 08525
08528 08538 08569 08580 08583 09041 09116 09236
09538 09555 09556 09591 09603 09610 09618 09662
09697
ER CANCER
00113* 00114* 00609 00624 00937* 01033* 01531* 01533*
01537* 01563 01578 01581 01583 01585 01586 01593
01837 01852* 01861* 01862* 01863* 01887 02487 02497

LIVER CANCER
CONTINUED
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06493 06527 06604 06788* 06837* 06842* 06893 07338*
07342* 07343* 07349* 07372 07380 07394 07476 07585
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08498 08500 08505 08510 08514 08517 08529 08530
08535 08550* 08553* 08582 08644 08645 08725* 08742*
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09499 09502 09527 09536 09537 09562 09569 09570
09577 09588 09589 09590 09599 09609 09614 09656
09707 09717
LIVER CARBOHYDRATE METABOLISM
00083* 00091* 00094 00102 00612 00622 00732* 00737
00743 00947* 01589 01823* 01831* 01836 02518 02699*
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03862 03863 03876 04320 04518* 04528* 04545 04604*
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05823* 05826 05831 05832 05950 06228 06480 06543
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07773 07785 07796 07801 08543* 08546* 08560* 08563
08597* 08657* 08933 08971 08982 09531
LIVER CARCINOGENESIS
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02532 02561 02642 03391 03452 03453 03846 03873
03874 03875 04469 04505 04510 04514 04531 04831
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07380 07421* 07430 07437 07438 07441 07587 07739*
07740* 07741* 08506 08542* 08543* 08544* 08550* 08552*
08553* 08555* 08556* 09536 09537 09588 09589 09614
09656 09660
LIVER CHEMICAL COMPOSITION
01547* 03501 03822* 05456*
LIVER CHOLESTEROL METABOLISM
00037 00208 00595* 00665 00687 00755* 00911 01637*
01663* 02528 02601* 02838* 02860 02868 03721* 03809*
03817* 03826 03860 04508 04682 04803* 04816 04824
04825 05432* 05805* 05808* 05815* 06536* 06783* 06813
07494* 07673 07725* 07756 07764 07780 07785 07821
08617 08980 09805
LIVER CIRCULATION
00090* 00264* 00607* 00647 00897 00902 00903 00916
00919 01358 01536* 01538* 01549* 01550* 01551* 01565
01574 01600* 01845 01877* 02476* 02480* 02510 02514
02517 02531 02542 02558 02574 02575 02645 02721
02821 02861 03360* 03365* 03367* 03403 03412 03444
03445 03460 03465 03481* 03486* 03591* 03593* 03599*
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04487 04490 04497 04498 04526* 04584 04586 04597*
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07375 07384 07388 07403 07412 07423* 07429 07470*
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09548 09550 09552 09565 09574 09577 09592 09596
09599 09606 09702 09703 09720 09730 09733 09790
09828
LIVER COMA
00206 00597* 00608* 00676 01537* 01596 01621 01622
01636* 01638* 01642* 02473* 02486* 02508 02509 02517
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03405 03406 03408 03410 03416 03472 03475 03520*
03523* 03539 03602 03612 03616 04015 04064 04198
04437* 04491 04530 04546 04560* 04598* 04599* 04605*

LIVER COMA
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05602* 05603* 05605* 05607* 05617 05618 05619 06449*
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06565 06582* 06605 06607 06613 07344* 07347* 07355
07485 07837* 07840 08438* 08441* 08455 08461 08499
08638* 08645 09505 09522 09530 09532 09553 09561
09568 09574 09604 09674 09710 09719
LIVER COPPER METABOLISM
01541* 03597* 04447* 04516* 04915 05426* 05456* 06451*
06515 07397 07770 07893 08425* 09572 09611
LIVER CYSTS
00655 01527* 01531* 01533* 01548* 01554 01556 01561
01861* 01918 01947 02478* 02498 02519 02541 02936
03422 03466 03938 03939 03954 04466 04503 04943
05486 05492 05499 05893 06499 06511 06619* 06842*
06893 06894 06938 07339* 07395 07409 07410 07417
08434* 08464 08468 08490 08494 08512 08530 09087
09503 09509 09510 09542 09559 09586 09619 09622
09659 09990
LIVER DEVELOPMENT
00015* 00083* 00097 00662 00900 01830* 02843 02853
02867 03403 03447 03840 04697 04831 05446* 05675
05689* 05700* 05701* 05825 06505 06551 06783* 06806
07598* 07607 07618 07700* 07713* 07714* 07734* 07735*
07742* 07743* 07746* 08963 08970 09006 09015 09548
LIVER DIAGNOSIS
00112* 00114* 00116* 00131* 00146 00150 00156 00171
00173 00175 00603* 00627 00646 00697* 00700 00729*
00746 00782 00937* 00941* 00947* 00957 00987 00989
00991 00992 00996 01003 01008 01009 01010 01015
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01612* 01675 01860* 01861* 01862* 01863* 01869* 01877*
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02471* 02474* 02487 02499 02500 02504 02513 02516
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03371* 03372* 03375* 03376* 03393 03417 03418 03461
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03536 03595* 03596* 03706* 03883* 03885* 03901* 03902*
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06583* 06786* 06787* 06788* 06789* 06837* 06839* 06840*
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07914 07922 07925 07926 07941 07942 07943 07947
07948 07952 07962 07965 07969 07971 07975 07976
08384 08427* 08447 08455 08456 08461 08468 08483
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09030 09032 09033 09038 09039 09041 09042 09049
09053 09059 09080 09081 09087 09089 09099 09102
09112 09115 09116 09120 09127 09491 09493 09499
09500 09503 09508 09519 09521 09529 09544 09545
09550 09554 09570 09571 09579 09583 09586 09587
09591 09593 09596 09599 09606 09614 09619 09621
09625 09627 09644 09661 09688 09705 09720 09730
09733
LIVER DISEASE COMPLICATIONS
09546
LIVER DISEASE DIAGNOSIS
SEE ALSO LIVER FUNCTION TESTS
09125 09510 09610
LIVER DISEASE EPIDEMIOLOGY
00183* 00613 00614 00615 00640 02570 05422* 05459
05462 05507 05508 06512 07452* 07463
LIVER DISEASE ETIOLOGY
01595 02545 04506 04515 05049 05422* 05436* 05438*
05439* 05483 05948 06452* 06460* 06510 07452* 08526
08637*
LIVER DISEASE TREATMENT
00674 00716 01527* 01566 01596 02485* 02486* 02541
02552 02553 02556 02596 02870 02895* 03361* 03362
03386 03398 03404 03405 03406 03407 03410 03468
03471 03472 03497 03539 03897* 03974 04437* 04447
04452* 04459* 04506 04532 04560* 04606* 05461 05482
05483 05486 05489 05513 05619 06453* 06491 06506
06509 06538* 06620* 06965 07347* 07351* 07361 07367
07382 07401 07404 07410 08426* 08432* 08438* 08445
08446 08456 08480 08571 08573 08585 08638* 0875
08862 09500 09504 09505 09509 09519 09522 0953
09532 09540 09552 09553 09561 09562 09604 09621
09636 09637 09645 09647 09674 09719 09918
LIVER DISEASE,HORMONAL TREATMENT OF
00706 02870 03471 06867* 08618 09729
LIVER DISEASES
00110* 00113* 00205 00206 00239 00608* 00644 00655
00665 00666 00667 00668 00677 00697* 00719 01055
01099 01482 01527* 01531* 01533* 01548* 01554 01556
01560 01561 01564 01567 01568 01572 01579 01594
01861* 01874* 01917 01947 02470* 02478* 02479* 02485
02498 02508 02511 02512 02522 02528 02538 02541
02547 02550 02574 02602* 02604* 02631* 02635* 02683
02891* 02932 02936 02937 02938 02941 02951 02991
03372* 03373* 03375* 03387* 03388* 03394 03397 03398
03403 03404 03409 03417 03421 03422 03458 03462
03464 03466 03466 03471 03478 03498 03500 03587
03591* 03660 03885* 03897* 03938 03939 03954 04466
04470 04493 04503 04838* 04943 04973* 05022 05061
05433* 05434* 05443* 05457* 05460 05469 05483 05486
05498 05499 05505 05506 05507 05508 05509 05512
05517 05525 05602* 05611* 05693* 05858* 05859* 05882
05893 05970 06044 06096 06100 06451* 06458* 06468
06486 06487 06499 06502 06508 06513 06840* 06917
07016* 07339* 07372 07373 07374 07376 07379 07387
07393 07395 07398 07400 07409 07410 07413 07414
07427 07570 07573 07781 07791 07889 07921 07942
07955 07962 07971 07976 08054 08176* 08422* 08444
08464 08468 08475 08486 08500 08503 08521 08523
08525 08526 08530 08534 08537 08578 08584 08698
08755 08756 08992 09041 09042 09081 09115 09496
09503 09507 09512 09524 09525 09529 09534 09550
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09639 09640 09644 09652 09658 09659 09661 09689
09723 09732 09756 09797 09845 09849 09867 09990
LIVER DISEASES IN CHILDREN
08426* 09538 09615 09670
LIVER DISEASES,BIOCHEMICAL DIAGNOSIS OF
00159 00596* 00599* 00602* 00616 00617 00619 00623
00626 00660 00690 00937* 01543* 01585 01639* 01896
02474* 02534 02535 02553 02884* 03373* 03467 03500
03954 04020 04606* 05438* 05439* 05866* 05970 05987
06792* 07899 08523 08537 08611 09047 09086 09089
09508 09576
LIVER DISEASES,BLOOD COAGULATION IN
00599* 00644 00659 03535 04504 04537 04885 05427
05493 06559* 07350* 08477 08639 09529 09554 09573
09683
LIVER DISEASES,METABOLIC CHANGES IN
09555
LIVER DRUG METABOLISM
00088* 01559 02492 02863 02877 03482* 03483* 0388
04486 04524* 04525* 04550 04670* 04796* 04802* 04804
04814 04818 04830 05589 05817* 05827 06541 06549
06795* 06811 06812 06818 06823 07711* 07712* 07715
07716* 07741* 07758 07759 08444 08636* 08967 08973
08979 08995 08999 09000 09001 09024 09598
LIVER ELECTROLYTE METABOLISM
06784*
LIVER ENZYMES
00083* 00097 00099 00682* 00690 00715 00891* 00892
00899 00907 00909 00910 00910 00914 00954* 01541
01563 01823* 01829* 01830* 01833* 01835 01836 01837
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02477* 02490 02537 02566 02576* 02577* 02630* 02737
02837* 02839* 02841* 02842* 02843 02850 02852 02855
02858 02859 02862 02863 02869 02871 02872 02879
02881 02884* 02951 03358* 03380* 03397 03429 03431
03432 03433 03479* 03514 03515 03524* 03537 0355
03592* 03629* 03682 03806* 03807* 03812* 03816* 03818
03821* 03824* 03825* 03828 03829 03830 03831 03833
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03852 03853 03854 03856 03857 03858 03861 03862
03863 03869 03872 03875 03876 04320 04446* 04451
04506 04518* 04545 04546 04690* 04792* 04796* 04797
04798* 04807* 04808* 04809* 04810 04811 04814 04817
04818 04820 04821 04822 04826 04830 04831 04832
05049 05438* 05439* 05484 05535* 05539* 05561 05563
05689* 05694* 05701* 05812* 05817* 05820* 05821* 05824
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06550 06769* 06778* 06780* 06781* 06783* 06786* 06787
06788* 06789* 06792* 06794* 06795* 06799* 06800* 06801
06811 06812 06813 06814 06815 06818 06819 06823
06827* 07455* 07596* 07697* 07698* 07700* 07701* 07702

R ENZYMES
TINUED
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7784 07787 07788 07789 07791 07794 07796 07797
7798 07801 07804 07807 07829 07934 07946 07962
8476 08487 08495 08506 08508 08543* 08546* 08560*
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8966 08967 08968 08970 08972 08973 08974 08984
8986 08994 08996 08997 08998 09000 09001 09006
9008 09013 09015 09016 09089 09099 09100 09555
9556 09563 09598 09601 09637 09641 09649 09653
9654 09675 09676 09694 09698 09719 09797 09972
R ENZYMES IN CHILDREN
538 03370* 04847*
R EXTRACTS
014* 00082* 00088* 00092 00093 00684* 00732* 03818*
804* 05549* 06794* 07759 07768 07796 07797 07946
8960 08985 08997 08998 09003 09014 09015 09641
R FISTULAS
0206 00597* 00646 01949 02500 06458* 09720 09990
R FUNCTION TESTS
0110* 00111* 00113* 00166 00168 00531* 00626 00670
0729* 00927* 00931* 00937* 00947* 00992 01002 01003
1008 01371* 01533* 01535* 01537* 01538* 01543* 01552*
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1913 01917 01996 02279* 02400 02470* 02471* 02475*
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8457 08474 08484 08500 08525 08528 08541* 08549*
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9046 09079 09089 09102 09120 09125 09430 09508
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9628 09629 09663 09664 09688 09693 09694 09700
9753 09775 09797
R GLUCOSE METABOLISM
7756
R GLYCOGEN
0092 00093 01831* 02499 02867 03358* 03696* 03698*
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6543 06550 06778* 07187* 07430 07436 07743* 07760
7773 07786 07796 07801 08088 08451 08476 08546*
8557* 08587 08924 08933 08968 09010
R HISTOLOGY
2424 02481* 02538 02543 02592 02593 02654 02656
2657 03358* 03392 03463 03464 03478 03479* 03518*
3523* 03592* 03599* 03696* 03698* 03709* 03716 03822*
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4573 04578 04580 04597* 05003 05144* 05215* 05431*
5441* 05449* 05451* 05453* 05458 05472 05474 05481
5484 05492 05523 05527* 05540* 05541* 05545* 05552*
5585 05586 05587 05615 05623 05693* 05700* 05701*
5834 05954 05959 06455* 06462* 06466* 06469* 06494
6520 06522 06526 06533* 06534* 06543 06546 06552
6583* 06685 06697 06790* 06814 06885 07021* 07330*
7332* 07343* 07346* 07377 07386 07393 07397 07398
7399 07418* 07436 07450* 07453* 07454* 07455* 07474
7598* 07607 07608 07706* 07722* 07730* 07736* 07768
7804 07870 07971 08440* 08449 08451 08455 08457
8478 08493 08500 08502 08509 08513 08521 08523
8523 08526 08527 08528 08533 08536 08539* 08546*
8551* 08562* 08588 08591 08608 08631* 08755 08921
8922 08930 08933 08958 08973 08976 08989 08992
9014 09089 09492 09521 09535 09551 09557 09575
9587 09595 09596 09603 09610 09618 09621 09626
9628 09629 09636 09645 09650 09660 09662 09670
9687 09696 09713 09719
LIVER IMMUNOLOGY
00014* 00600* 00618 00663 00709 00723 02472* 02540
02550 03413 03436 03521* 03678 03901* 03963 04021
04440* 04443* 04471 04474 04479 04480 04482 04483
04501 04511 04512 04554 04568* 04574 04587 04588
04591 04592 04650 04963 05016 05434* 05464 05523
05527* 05543* 05545* 05546* 05583 05585 05606* 05621
05674 05870* 05905 06461* 06470 06492 06498 06552
06590 06602 06685 06868* 07345* 07354 07415 07443*
07450* 07734* 07735* 07750* 07770 07802 07867* 07891
07913 08423* 08439* 08445 08476 08481 08486 08495
08537 08551* 08553* 08561* 08583 08599* 08603* 08613
08974 08977 09087 09518 09540 09557 09633 09663
09678 09687 09696
LIVER IN CHILDREN
00088* 00612 00652 00660 01540* 01579 01860* 02505
03370* 03371* 03461 03481* 04013 04058 04435* 04459*
04515 04530 04572 04821 05446* 05456* 05479 05608*
05893 06450* 06471 06488 06552 06938 07334* 07335*
07413 07746* 08455 08491 08497 08503 08733* 08830
09116 09496 09503 09524 09540 09562 09563 09575
09605 09609 09655
LIVER INJURIES
00683* 00690 00691 00702 00809* 01559 01609 01656*
02500 02576* 02583 02584 02842* 02875 03001* 03368*
03420 03458 03483* 03484* 03488* 03494 03496 03499
03501 03507 03512 03521* 03592* 03813* 03814* 03908*
04441* 04446* 04450* 04452* 04480 04514 04520* 04521*
04524* 04530 04531 04532 04544 04551 04552 04553
04555 04845* 04956 05399 05442* 05451* 05453* 05490
05494 05516 05537* 05540* 05548* 05549* 05552* 05553*
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05565 05566 05568 05575* 05585 05807* 06286 06464*
06477 06531* 06533* 06538* 06539 06541 06545 06546
06547 06548 06549 06551 06552 06568 06588 06792*
06884 06914 07332* 07334* 07335* 07422* 07423* 07428
07429 07431 07432 07433 07434 07435 07436 07440
07456 07463 07706* 07716* 07864* 07870 07908 08050
08422* 08425* 08426* 08467 08533 08536 08540* 08545*
08548* 08551* 08566 08567 08568 08575 08590 08822
08956 08990 09000 09007 09029 09102 09105 09563
09581 09587 09623 09632 09635 09638 09653 09654
09655 09658 09786 09856
LIVER INJURIES,ANESTHETICS IN
00683* 00688 02578* 02591 03482* 03511 04507 04582
05534* 06532* 06534* 06542 07422* 08549* 08587 09643
LIVER INJURIES,ANTIBIOTICS IN
00683* 01113 01606 03363* 03492 05538* 05564 06537*
08568
LIVER INJURIES,CONTRACEPTIVE DRUGS IN
01558 01850 06525 06544 07376 07377 07418* 07427
07439 07725* 08558* 08564 08586 09545
LIVER INJURIES,DIETARY DEFICIENCIES IN
00729* 00730* 01544* 01599* 01641* 02579* 02627* 02630*
02639 02641 03374* 04515 05501 05600* 06033 06549
06778* 06808 06810 07330* 07780 08050 08557*
LIVER INJURIES,DRUG-INDUCED
00683* 03481* 03719 03834 04094 04486 04533 04537
04538 04542 04543 04545 04547 04548 04549 04550
04554 05476 05526* 05534* 05539* 05544* 05547* 05554
05557 05596* 05813* 06247* 06460* 06534* 06537* 06540
06822 06823 06868* 07364 07425 07438 07441 07740*
07771 08522 08539* 08555* 08574 08586 08588 09561
09624 09646
LIVER INJURIES,ETHIONINE IN
01605
LIVER IRON METABOLISM
00082* 00096 00651 00657 00678 00684* 01530* 01568
01968 01969 02629* 02900* 02920* 03097* 03464 03588*
03592* 03843 04087 04590 04675* 04915 04950 05430*
05444* 05453* 05617 05875* 06468* 06606 06709 06816
07332* 07374 08476 08525 08631* 08941 09646
LIVER LIPID METABOLISM
00037 00171 00595* 00612 00647 00650 00685* 00895*
00898 00911 00912 01534* 01542* 01544* 01556 01562
01588 01605 01637* 01743 01840 02540 02601* 02636*
02673 02845 02851 03388* 03427 03434 03476 03492
03629* 03696* 03813* 03819* 03823* 03824* 03826 03828
03830 03836 03842 03850 03852 03860 03861 03862
03867 03877 04452* 04508 04534 04535 04548 04682
04683* 04793* 04803* 04815 04816 04825 04827 04828
05144* 05308 05431* 05432* 05433* 05471 05528* 05537*
05549* 05551* 05689* 05804* 05808* 05810* 05813* 05819*
05834 06501 06536* 06538* 06692 06779* 06797* 06820
06822 07083* 07420* 07436 07439 07502* 07701* 07704*
07705* 07709* 07710* 07718* 07720* 07725* 07726* 07727*
07728* 07729* 07749* 07751* 07756 07779 07781 07783
07785 07786 08447 08540* 08579 08637* 08955 08957
08971 08975 08987 08989 09010 09011 09597 09716
09805
LIVER LYMPHOGRAPHY
01545* 04934 04955 05611* 05969
LIVER MAGNESIUM METABOLISM
01842 06595

LIVER METABOLISM
00051* 00084* 00089* 00095 00608* 00654 00731* 00790*
00906 00907 00915 00954* 01586 01665* 01833* 01839
02487 02491 02536 02572 02594 02837* 02843 02864
02865 02882 03364* 03373* 03403 03688 03810* 03811*
03826 03838 03871 03873 04446* 04461 04539 04556
04608* 04795* 04798* 04801* 04805* 04808* 04813 04833
04834 04835 04853* 05429* 05454* 05457* 05460 05474
05476 05506 05524 05547* 05558 05820* 05824 05987*
06472 06534* 06549 06790* 06798* 06804 06809 06814
06815 07350* 07392 07619 07697* 07717* 07728* 07735*
07742* 07743* 07744* 07771 07772 07775 07790 07794
07800 07810* 07899 08230* 08422* 08431* 08432* 08437*
08450 08483 08497 08515 08516 08517 08518 08525
08527 08558* 08577 08587 08649 08664* 08956 08961
08968 08975 08988 08996 09002 09519 09530 09555
09560 09578 09615 09621 09625 09637 09651 09653
09712
LIVER MICROORGANISMS
03951 07363 07400 07431 08504 09507 09765 09981
LIVER MORPHOLOGY
00015* 00017 00595* 00625 00628 00629 00686* 00689
00724 00809* 00909 00918 00919 01565 01597 01656*
01841 02493 02494 02515 02532 02538 02576* 02577*
02580* 02591 02631* 02634* 02673 02704* 02706* 02721
02735* 03364* 03368* 03378* 03413 03449 03454 03480*
03578 03590* 03596* 03706* 03709* 03865 03866 03942
04473 04694* 04697 04700 04701 04705 04906 05481
05503 05564 05669* 05675 05680* 05692* 05693* 05699*
05704 05816* 05817* 05907 05948 05989* 06505 06516
06553* 06682 06683 06876* 06948 07021* 07137* 07332*
07418* 07422* 07444* 07550* 07601 07608 07722* 07775
07877 08509 08510 08517 08523 08530 08535 08536
08567 08923 09489 09506 09511 09513 09516 09550
09568 09650 09654 09660
LIVER NEOPLASMS
00632 00677 02966 03399 04901 05491 05609* 05898
05965 06478 07441 07738* 07778 07942 08440* 08491
08506 08552* 08995 09013 09500 09614
LIVER NEOPLASMS,BENIGN
01580 02502 02506 03371* 03939 04476 06464* 06894
07378 08469 08493 08501 08512 08520 09516 09520
09551 09566 09619
LIVER NEOPLASMS,MALIGNANT
00148 00205 00650 01556 01557 02480* 02482* 02502
02505 02506 02513 02561 02565 03408 03463 03904*
04515 05437* 05874* 06456* 06464* 06837* 06876* 06894
07338* 07343* 07429 08428* 08433* 08460 08469 08498
08620 08645 08719* 09125 09493 09499 09501 09516
09562 09656
LIVER NUCLEIC ACID METABOLISM
00906 01547* 01563 01835 02609 02846 02869 02871
02873 02876 02879 02881 03358* 03439 03442 03489
03496 03497 03512 03550 03585* 03592* 03709* 03815*
03818* 03825* 03841 03843 03844 03845 03847 03848
03851 03864 03865 03866 03869 03874 03875 03879
04469 04508 04510 04518* 04522* 04555 04794* 04796*
05704 05806* 05816* 05817* 06535* 06794* 06805 06806
06807 06823 07419* 07421* 07455* 07456 07703* 07706*
07721* 07724* 07733* 07740* 07766 07795 08559* 08560*
08962 08985 09687
LIVER PATHOLOGY
00010 00051* 00159 00611 00612 00615 00640 00662
00671 00689 00692 00694 00712 00726 00727 00729*
00910 01079 01536* 01539* 01540* 01553 01557 01567
01572 01580 01584 01597 01599* 01601 01618* 01926*
02424 02470* 02471* 02480* 02481* 02494 02550 02579*
02600* 02602* 02604* 02608 02628* 02644 02739 02877
02973* 03360* 03364* 03374* 03384 03387* 03392 03396
03402 03403 03414 03421 03479* 03480* 03488* 03495
03503 03520* 03522* 03524* 03568 03598* 03620 03706*
04005 04432* 04460* 04462 04464 04476 04485 04516*
04519* 04520* 04521* 04549 04552 04572 04574 04575
04590 04596* 04610 04616 04791* 05003 05005 05144*
05241 05365 05440* 05441* 05442* 05446* 05449* 05451*
05453* 05456* 05460 05464 05468 05472 05473 05474
05478 05481 05488 05491 05492 05495 05499 05500
05504 05506 05517 05519 05520 05521 05527* 05564
05574* 05575* 05581 05582 05584 05585 05586 05595*
05674 05693* 06033 06087 06153* 06387 06455* 06469*
06470 06471 06489 06493 06496 06497 06499 06500
06508 06521 06523 06525 06538* 06554* 06583* 06589
06600 06604 06692 07021* 07137* 07312 07330* 07338*
07343* 07346* 07378 07413 07421* 07423* 07430 07445*
07456 07475 07477 07482 07484 07499* 07542* 07580
07781 07789 07806 07877 07955 07957 07962 07963
07969 07971 07975 07976 08430* 08431* 08455 08457
08476 08493 08494 08497 08499 08502 08503 08506
08509 08511 08512 08515 08516 08517 08518 08519
08520 08521 08525 08526 08527 08533 08534 08537
08538 08542* 08543* 08547* 08552* 08554* 08557* 08562*
08565 08571 08573 08575 08582 08583 08588 08589
08596* 08613 08615 08616 08625 08626 08631* 08646
08652 08755 08870 08955 08992 09007 09041 09086
09430 09492 09503 09504 09509 09512 09520 09524
09527 09531 09535 09550 09551 09552 09559 09563
LIVER PATHOLOGY
CONTINUED
09566 09587 09595 09596 09599 09610 09611 09618
09621 09625 09626 09635 09636 09647 09661 09670
09673 09674 09675 09676 09677 09703 09711 09713
LIVER PERFUSION
00091* 00809* 00902 00905 00908 01569 01827* 01831*
02475* 02518 02699* 02735* 02844 02851 03395 03410
03472 03491 03599* 03819* 03842 03844 03870 03904*
04433* 04437* 04481 04500 04670* 04694* 04794* 04795*
04799* 05428* 05429* 05458 05482 05558 05619 05804*
05809* 05810* 05811* 05821* 05826 06449* 06453* 06524
06795* 06798* 06800* 06820 07355 07485 07724* 07759
07776 07788 07822 07925 08435* 08437* 08441* 08504
08554* 08876 08971 08982 09344 09502 09505 09522
09532 09574 09828
LIVER PHOSPHOLIPID METABOLISM
00912 01648* 02880 03852 03867 04508 04521* 04541
04682 04683* 04816 05804* 05805* 06779* 06821 06824
07705* 07784 08969
LIVER PLASMA PROTEINS
00900 01297* 02706* 03377* 03533 03534 03608 03610
03615 07752* 08561* 08603*
LIVER PROTEIN METABOLISM
00082* 00604* 00671 00685* 00745 00897 00900 00903
00905 00908 01002 01261* 01547* 01605 01606 01829*
01846 01848 02595 02597* 02623 02839* 02841* 02847
02869 02870 02872 03358* 03409 03476 03488* 03524*
03822* 03841 03842 03843 03844 03845 03847 03849
03859 03864 03865 03866 03870 03876 04014 04519*
04607* 04676* 04794* 04817 04832 04836 04932 04947
04952 04977* 05324 05427* 05505 05512 05548* 05598*
05601* 05622 05689* 05721* 05806* 05809* 05810* 05813*
05818* 05831 05832 05833 06033 06481 06530 06533*
06535* 06557* 06591 06611 06612 06780* 06793* 06795*
06801 06823 06824 07355 07379 07421* 07424 07428
07437 07455* 07699* 07703* 07708* 07716* 07721* 07735*
07740* 07753 07756 07763 07767 07770 07789 07799
07805 07806 07862* 08451 08465 08485 08509 08539*
08560* 08563 08617 08972 08976 08979 08981 08983
08992 09030 09518 09673 09710 09719
LIVER RADIOLOGY
SEE ALSO LIVER DIAGNOSIS
00131* 00137 00148 00150 00173 00175 00623 00627
00646 00667 00677 00746 00941* 00987 01117 01533*
01537* 01538* 01545* 01554 01561 01856* 01861* 01869*
01877* 01879* 01885 01887 01889 02500 02519 02530
02542 02605* 02651 02906* 02936 02937 02938 02939
02941 02953 02966 02967 03376* 03417 03457 03465
03486* 03526* 03595* 03596* 03601* 03885* 03936 03938
03939 03942 04010 04013 04076 04457* 04487 04551
04694* 04701 04846* 04848* 04900 04906 04934 04938
04942 04943 04953 04955 04956 05440* 05465 05472
05516 05556 05611* 05675 05843* 05844* 05874* 05891
05892 05893 05898 05907 05913 05918 05924 05927
05937 05944 05948 05959 05966 05969 05974 06482
06493 06600 06840* 06842* 06853* 06864* 06876* 06884
06885 06893 06894 06930 06938 06940 06959 06960
06965 07351* 07402 07844* 07860* 07877 07889 07892
07908 07922 07925 07926 07931 07941 07942 07943
07948 07949 07965 08454 08456 08468 08647 09026
09028 09032 09033 09038 09039 09041 09042 09053
09080 09087 09115 09489 09493 09503 09519 09523
09593 09596 09599 09660 09705 09720 09730 09733
LIVER REGENERATION
00656 00662 00906 01104 01531* 01557 01834* 01838
02477* 02480* 02490 02506 02515 02517 02576* 02642
02704* 02706* 02714 02842* 02880 03378* 03382* 03382*
03427 03429 03430 03431 03432 03433 03434 03435
03436 03437 03438 03439 03440 03441 03442 03443
03445 03446 03448 03449 03450 03451 03453 03454
03455 03456 03457 03504 03513 03620 03808* 03815*
03841 03847 03849 03865 03866 03924* 04514 04529*
04544 04817 05530* 05538* 05540* 05541* 05552* 05567
05568 05692* 05704 05816* 05831 05832 06535* 06549
06777* 06790* 06798* 06801 06805 06806 06817 07137*
07333* 07419* 07440 07454* 07455* 07703* 07705* 07706*
07708* 07722* 07723* 07724* 07730* 07731* 07733* 07736*
07738* 07752* 07755 07762 07766 07795 08423* 08433*
08445 08492 08502 08510 08536 08559* 08581 08593*
08616 08641 08654 08968 08973 08983 08985 09574
09615 09616 09618 09619 09645 09697 09698 09719
LIVER RETICULOENDOTHELIAL SYSTEM
00017 00595* 03391 03924* 04076 04457* 05540* 05669*
06059 06557* 06692 06697 06838* 07335* 07433 07442*
07836* 09608 09635
LIVER SECRETION
00101 00839* 01996 02071 03369* 03641 04794* 05537*
06792* 07615 07726* 07729* 07807 07969 08527 08563
08636* 09508 09540
LIVER SERUM ENZYMES
SEE ALSO ALKALINE PHOSPHATASE
SEE ALSO LIVER DISEASE DIAGNOSIS
00166 00638 00715 00931* 00937* 00947* 01002 01003
01539* 01543* 01552* 01603 01615* 01616* 01631 01639*
01668 01852* 01863* 01869* 02470* 02576* 02597* 02598*

R SERUM ENZYMES
TINUED
2602* 02604* 02606 02609 02622 02706* 02916* 02958
2959 03001* 03356* 03365* 03377* 03482* 03484* 03491
3499 03507 03523* 03531 03532 03536 03542 03549
3609 03615 03685 03806* 03835 03962 03967 03984
020 04441* 04446* 04496 04523* 04541 04585 04813
843* 04845* 04872 04879 04885 04915 04920 04932
5201 05442* 05466 05494 05536* 05546* 05553* 05560
5572* 05576* 05592* 05599* 05617 05637* 05859* 05866*
5889 05919 05926 05934 05959 06286 06460* 06513
5515 06532* 06545 06553* 06554* 06561 06568 06572
5577 06651 06777* 06789* 06852* 06853* 06854* 06855*
5865* 06867* 06869* 06923 06928 07351* 07369 07418*
7422* 07440 07444* 07449* 07459 07626* 07752* 07763
7806 07840 07842* 07864* 07867* 07870 07914 07962
3366 08424* 08425* 08441* 08474 08537 08541* 08549*
3561* 08562* 08594* 08595* 08598* 08601* 08612 08626
3986 09033 09086 09430 09625 09628 09641 09663
9673 09693
R STEROID METABOLISM
2875 03398 04619 04797* 04823 05475 05512 05594*
5689* 05830 08475
R SURGERY
0087* 00649 00663 00673 00735 00736 01049 01059
1531* 01532* 01546* 01548* 01549* 01550* 01551* 01565
1569 01570 01593 01600* 01602 01607 01652* 01844
2478* 02480* 02484* 02485* 02493 02507 02515 02517
2520 02563 02573 02575 03365* 03371* 03377* 03413
3423 03428 03457 03465 03467 03475 03593* 04435*
4438* 04448* 04450* 04459* 04461 04466 04497 04500
4503 04551 04606* 05424* 05427* 05428* 05443* 05469
5485 05490 05499 05502 05503 05510 05517 05525
5564 05565 05620 05859* 06447* 06464* 06478 06516
5583* 06586 06599 06619* 06654 06777* 06798* 07332*
7333* 07336* 07350* 07355 07366 07383 07384 07388
7395 07400 07410 07414 07435 07470* 07550* 07601
7730* 07731* 08434* 08435* 08437* 08441* 08457 08465
3480 08481 08488 08491 08510 08512 08514 08517
3519 08520 08529 08533 08535 08536 08566 08567
3568 08590 09087 09489 09506 09509 09511 09513
9516 09526 09554 09557 09561 09562 09569 09622
9627 09632 09659 09778 09781 09990
R TRACE ELEMENT METABOLISM
0756* 01541* 01640* 01842 02474* 02630* 03597* 03812*
3814* 03855 03857 04447* 04516* 04633* 04915 05426*
5453* 06451* 06515 06595 06816 07397 07477 07893
3425* 09491 09572
R TRANSPLANTATION
0087* 00647 00663 00809* 01546* 01550* 01551* 01565
1570 01593 01705 02475* 02517 02518 02520 02527
2558 02573 02575 02735* 02844 03360* 03382* 03413
3428 03852 03924* 04433* 04435* 04440* 04448* 04459*
4477 04481 04500 05424* 05428* 05429* 05445* 05458
5470 05510 05536* 06514 06531* 06798* 07336* 07384
7550* 07738* 07750* 08423* 08435* 08439* 08445 08481
3488 08489 08504 09518 09526 09557 09828
R TRAUMA
0649 00652 00656 00687 00691 00961 01069 01352
1529* 01557 01598* 01600* 01602 01607 01610 01944
2563 02584 03467 03475 03810* 03910* 04441* 04450*
4462 04551 04833 05516 05564 05565 05566 06894
7350* 07435 08050 08533 08536 08566 08568 09039
9559 09623 09627 09630 09632 09638
R TREATMENT
5984* 06511 08441* 08491 08491 09548 09579 09585
9586 09606 09632 09644 09692
R ULTRASTRUCTURE
2538 02594 02735* 03378* 03387* 03447 03456 03488*
3505 03516 03517 03522* 03524* 03568 03592* 03620
3696* 03698* 03706* 03709* 03716 03719 03813* 03834
3865 03866 04432* 04460* 04473 04516* 04519* 04555
4633* 04690* 04700 04793* 04803* 04809* 04834 04835
5447* 05449* 05453* 05456* 05460 05472 05481 05484
5542* 05544* 05545* 05574* 05575* 05581 05669* 05680*
5694* 05699* 05704 05820* 05827 05834 05954 06455*
6525 06534* 06551 06623* 06682 06683 06692 06697
6780* 06784* 06818 06819 06820 06821 06823 07017*
7187* 07334* 07335* 07420* 07421* 07430 07432 07436
7438 07445* 07455* 07477 07598* 07607 07615 07618
7619 07708* 07718* 07721* 07721* 07758 07765 07767
7769 07775 07777 07782 07784 07790 07793 07795
7809 07809 08430* 08431* 08449 08451 08460 08497
8539* 08542* 08543* 08545* 08546* 08547* 08551* 08552*
8554* 08557* 08574 08591 08595* 08615 08921 08922
8933 08958 08965 08966 08968 08969 08974 08983
8999 09000 09001 09006 09011 09492 09555 09556
9563 09566 09582 09595 09601 09612 09642 09648
9650 09656 09658 09660 09687 09597 09698 09789
R UREA METABOLISM
1829* 01830* 02523 02549 03870 04064 04546 04620
4676* 04794* 04812 04977* 05810* 07700* 08971
R VITAMIN METABOLISM
5815* 07774

LIVER ZINC METABOLISM
01640* 02474* 02630* 09709
LIVER,AGE EFFECTS ON
00647 01584 01839 02635* 02673 03453 03473 03829
03832 03840 03848 03861 03862 03869 04797* 04806*
04820 04821 04847* 05450* 05478 05481 05692* 05694*
06526 06592 06777* 06781* 06806 07137* 07499* 07852*
08970 09007
LIVER,ALCOHOL EFFECTS ON
00171 00606* 00626 00631 00648 00668 00686* 00693
00738 00739 00914 00947* 01055 01534* 01542* 01552*
01576 01588 01599* 01641* 02470* 02471* 02473* 02536
02539 02577* 02579* 02581* 02585 02586 02587 02589
02590 02627* 02628* 02629* 02630* 02633* 02634* 02751*
03387* 03441 03476 03478 03491 03493 03502 03503
03504 03515 03591* 03607 03619 03836 04434* 04452*
04453* 04523* 04528* 04534 04536 04607* 04798* 04801*
04956 05432* 05447* 05448* 05449* 05457* 05472 05528*
05529* 05531* 05535* 05547* 05550* 05556 05559 05812*
05924 05925 06490 06501 06518 06519 06520 06521
06522 06523 06536* 06546 06585 06589 06591 06810
07016* 07420* 07434 07439 07443* 07451* 07479 07480
07587 07709* 07748* 07749* 07804 07808 08447 08530
08540* 08565 08573 08579 08582 08589 08955 08957
08975 08987 08989 09570 09578 09579 09580 09581
09582 09621 09626 09629 09631 09649 09653 09709
09725
LIVER,AMEBIASIS EFFECTS ON
00203 00206 01003 02479* 02530 02560 02891* 03372*
03375* 03393 03417 03897* 03917* 04970* 04971* 04973*
05443* 05469 05477 05498 05570* 05905 06458* 06874*
07351* 07395 08456 08562* 08570 08571 08572 08573
08582 08583 08584 08723* 08750 08756 08814 09983
LIVER,CLINICAL STUDIES OF
00093 00159 00208 00597* 00599* 00608* 00612 00613
00614 00615 00616 00617 00618 00619 00625 00630
00640 00657 00658 00659 00661 00664 00669 00691
00742 01592 01594 01595 02527 02535 02538 02544
02584
LIVER,DISEASES ASSOCIATED WITH
00610 00616 00618 00622 00630 00645 00661 00664
00668 00669 00704 00716 00728 00770 00774 01610
01678 02486* 02559 02612 03458 03468 03587* 07969
08534 09552 09644
LIVER,DRUG EFFECTS ON
00633 02552 02591 03359* 03394 03481* 03492 03496
03497 03498 03505 03585* 04439* 04444* 04471 04486
04494 04524* 04527* 04531 04694* 04697 04803* 04809*
04817 04818 04848* 05533* 05765* 05808* 05817* 05820*
05869* 06537* 06538* 06540 06626* 06769* 06780* 06799*
06805 06812 06818 06821 06822 06823 06853* 07401
07425 07426 07438 07702* 07705* 07713* 07723* 07728*
07737* 07748* 07751* 07757 07758 07760 07764 07771
07778 07782 07797 07798 07799 07807 07808 07864*
08450 08489 08522 08549* 08560* 08574 08586 08587
08591 08594* 08641 08652 08967 08970 08973 08978
08987 08994 08996 09000 09004 09504 09508 09540
09543 09624 09636 09648 09651 09668 09671 09712
09765 09918
LIVER,DRUG TREATMENT OF
03361* 03362* 03382* 03405 03406 03407 03408 03539
03540 03541 03543 03544 03614 03897* 03917* 03951
03956 04447* 04489 04506 04568* 04584 04586 04592
04925 05461 05469 05477 05576* 05613 06100 06247*
06476 06491 06495 06506 06511 06517 06560 06562
06567 06576 06582* 06613 06965 07351* 07367 07404
07408 07471* 07478 07479 07752* 07808 08426* 08446
08456 08570 08571 08572 08573 08585 08606 08641
08652 08723* 08754 08756 09504 09508 09516 09519
09540 09636 09637 09671 09680 09699 09719 09736
09765
LIVER,ENZYME TREATMENT OF
09645
LIVER,EXPERIMENTAL STUDIES OF
00087* 01609 02527 02558 02573 02582* 02583 02589
LIVER,FATTY
00595* 00606* 00631 00648 00661 00678 00728 00729*
00755* 00947* 01015 01080 01104 01534* 01536* 01542*
01543* 01544* 01552* 01555 01556 01562 01576 01588
01599* 01606 01636* 01675 01730* 02402 02470* 02536
02539 02578* 02579* 02590 02639 03356* 03361* 03362*
03374* 03389 03434 03462 03468 03485* 03492 03495
03497 03503 03507 03517 03520* 03598* 03813* 03822*
03826 03836 03867 03954 04114 04434* 04443* 04443*
04452* 04494 04508 04513 04516* 04517* 04518* 04523*
04534 04535 04656 04683* 04793* 04902 04964 05241
05447* 05448* 05455* 05463 05471 05476 05500 05501
05520 05528* 05529* 05531* 05532* 05537* 05550* 05551*
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06494 06501 06517 06520 06521 06536* 06541 06543
06549 06623* 06692 06704* 06797* 06810 06815 06821
06822 06823 06824 06855* 06868* 07021* 07330* 07332*
07346* 07373 07391 07401 07420* 07424 07480 07705*
07729* 07749* 07751* 07779 07789 08385 08427* 08447
08455 08476 08501 08538 08540* 08548* 08589 08637*
08639 08728* 08955 08957 08987 08989 08993 09000

LIVER,FATTY
CONTINUED
09041 09582 09595 09621 09626 09631 09805
LIVER,HORMONAL CONTROL OF
00099 00621 00904 00913 00915 01606 01836 01840
01849 02547 02699* 02845 02861 02862 02867 02869
03469 03698* 03808* 03816* 03819* 03824* 03874 04471
04496 04514 04697 04797* 04819 04820 04822 04823
04826 04926 05450* 05468 05476 05526* 05562 05567
05573* 05700* 05807* 05808* 05810* 05815* 05818* 05826
06786* 06791* 06794* 06798* 06800* 06804 06817 06820
07377 07381 07418* 07427 07439 07447* 07697* 07698*
07708* 07720* 07725* 07731* 07733* 07737* 07753 07756
07769 07788 07798 07801 08556* 08564 08970 08978
08982 08984 09613
LIVER,NERVOUS CONTROL OF
01843 02869 03367* 03833 04807* 05807* 06454* 06457*
06816 07017* 07348* 07378 07392 09506 09781
LIVER,TOXIC EFFECTS ON
00010 00014* 00603* 00606* 00629 00631 00654 00683*
00684* 00685* 00686* 00688 00692 00693 00694 00702
00715 00906 00927* 00934* 01079 01179* 01539* 01540*
01541* 01556 01558 01559 01588 01601 01603 01604
01605 01606 01608 01633* 01641* 01720* 01833* 01863*
02492 02498 02545 02549 02576* 02578* 02580* 02594
02595 02596 02599* 02639 02875 02877 03368* 03374*
03383* 03389 03432 03433 03435 03440 03443 03460
03475 03479* 03480* 03482* 03483* 03484* 03489 03490
03491 03492 03496 03497 03498 03499 03501 03505
03507 03511 03512 03513 03514 03516 03521* 03549
03585* 03599* 03602 03653 03716 03719 03813* 03814*
03815* 03821* 03834 03844 03855 03874 03954 04009
04012 04094 04446* 04469 04473 04486 04506 04514
04515 04518* 04519* 04520* 04521* 04522* 04523* 04524*
04527* 04528* 04529* 04531 04532 04533 04534 04535
04536 04537 04538 04539 04540 04541 04542 04543
04544 04545 04546 04547 04548 04549 04550 04552
04553 04554 04555 04556 04582 04616 04633* 04705
04719* 04794* 04796* 04834 04845* 04848* 04892 04954
05077 05437* 05442* 05447* 05449* 05460 05465 05481
05500 05527* 05528* 05532* 05533* 05534* 05537* 05538*
05539* 05540* 05541* 05542* 05543* 05544* 05545* 05546*
05547* 05548* 05549* 05550* 05552* 05553* 05554 05555
05557 05558 05561 05562 05563 05580 05596* 05704
05812* 05813* 05820* 05824 05827 06247* 06459* 06460*
06471 06489 06490 06520 06521 06522 06523 06525
06533* 06534* 06535* 06536* 06538* 06540 06541 06542
06543 06544 06546 06548 06549 06550 06589 06692
06781* 06784* 06787* 06792* 06797* 06803 06804 06811
06868* 06884 06933 07187* 07380 07385 07419* 07420*
07421* 07423* 07424 07426 07428 07430 07431 07432
07433 07434 07437 07438 07440 07441 07587 07681*
07706* 07716* 07721* 07722* 07725* 07736* 07737* 07748*
07749* 07752* 07755 07758 07771 07804 07807 07808
07864* 07870 08450 08454 08492 08539* 08540* 08544*
08547* 08550* 08552* 08553* 08554* 08555* 08556* 08559*
08560* 08561* 08563 08564 08574 08575 08576 08577
08579 08587 08588 08591 08616 08654 08956 08958
08975 08976 08977 08979 08987 09000 09004 09102
09533 09536 09537 09543 09588 09589 09601 09624
09626 09628 09641 09642 09643 09646 09650 09654
09660 09662 09704 09731 09972
LIVER,VITAMIN B12 IN
02739 03851 06791*
LUPOID HEPATITIS
00600* 00927* 01553 01571 01720* 04512 04591 04592
04593 06461* 09686
LYMPH
00028* 00740 00818* 01992 02784* 03618 03642 03767*
03923* 04682 04729* 04738 04785 04939 04940 05068
05069 05161 05383* 05440* 05488 05521 05620 05627
05698* 05989* 06457* 06481 06530 06679* 06682 06683
06684 06698* 06705* 06884 06885 06897 06898 07088
07089 07090 07204 07296 07306* 07313 07352 07356
07357 07358 07359 07360 07388 07465* 07472 07473
07474 07475 07558 07635* 07811* 08111 08415 08452
08459 08515 08516 08633* 08634* 08719* 08760 08920
09521 09724
LYMPH NODES
05705 05854* 06486 06660 06996 08144 08312 09174
09201
LYMPH NODES,MESENTERY
00198* 02973* 04681 04887 04933 05015 05064 05304
05989* 06164 06322 08808 08843
LYMPHADENITIS
07449* 09099
LYMPHADENITIS,MESENTERY
09901
LYMPHOGRAPHY
01545* 04945 05002 05854* 05876* 05964 06322 06963
08920
LYMPHOGRAPHY IN CANCER DIAGNOSIS
04933 05906 08096
LYMPHOGRAPHY,ABDOMEN
06899
LYMPHOGRAPHY,GASTROINTESTINAL
01992 04933 06899
LYMPHOGRAPHY,LIVER
01545* 04934 04955 05611* 05969
LYMPHOGRAPHY,PANCREAS
02463 03952
LYMPHOGRAPHY,SMALL INTESTINE
05963 06898
LYMPHOMA
00470 01928* 03029 04356 06009 06035 06223 062
08243 08336 09216
LYMPHOSARCOMA
00170 01029* 01176* 03123 03168 04065 05015 060
08075 08093 08147 08246 08302 09099 09174

MAGNESIUM
04669* 05289* 06441 06802 07787 08928 09019 090
MAGNESIUM ABSORPTION
01037* 05076 05207 07642* 08942
MAGNESIUM METABOLISM,LIVER
01842 06595
MALABSORPTION
SEE ALSO SMALL INTESTINE
00302* 00303* 00320 00345* 00438 00439 01290 017
01732* 01868* 01923* 01953 02051* 02132 02279* 022
02281* 02282* 02283* 02287 02290 02291 02292 022
02297 02391* 02392* 02393* 02603* 02759* 02988* 030
03029 03061 03150* 03152* 03226* 03227* 03228* 032
03236* 03238 03246 03301* 03627* 03629* 03720* 037
03912* 04029* 04044* 04079 04097 04309* 04325 043
04382* 05179* 05181* 05202 05286* 05294 05303 053
05364 05386* 05392 05733 05985* 05986* 06039 060
06220* 06508 07090 07145 07446* 07576 07589 076
07621 07902 08121 08159 08226* 08495 08853 093
09329 09334 09859 09920
MALABSORPTION DIAGNOSIS
00062* 00115* 00126* 00129* 00428* 00923* 01251* 018
02280* 02291 03986 04002 04003 04124 04307* 043
04315 04318 04730* 04854* 04880 04903 04940 049
04950 04951 04959 05030 05288* 05298 05299 053
05841* 05861* 05864* 05886 05887 05910 05973 068
06851* 06863* 06880* 06903 07197 07838* 08232* 082
08240 08242 09082 09335 09338
MALABSORPTION DIAGNOSIS,BIOPSY IN
02281* 04314 08247
MALABSORPTION DIAGNOSIS,D XYLOSE IN
00926* 04000 04314 04959 05309 05867* 06836* 082
08247 09074 09083 09323 09326 09327
MALABSORPTION DIAGNOSIS,TOLERANCE TESTS IN
00129* 00153 00426* 00428* 00435* 02281* 02913* 032
04000 04314 04442* 04880 06836* 08247 09323
MALABSORPTION EPIDEMIOLOGY
01055 02748* 08229*
MALABSORPTION ETIOLOGY
00186* 00436 01112 01375 01494 02282* 02289 022
04062 04092 04101 04310* 04314 04317 04318 043
04323 04714* 05031 05065 05072 05184* 05207 052
05290* 05292 05293 05298 05299 05301 05302 053
06145* 06216* 06322 06324 06325 06406 07027* 070
07190* 07191* 07197 07198 07199 07200 07202 072
07204 07290 08116* 08129 08225* 08228* 08232* 082
08236 08241 08242 08244 09082 09176 09327 093
MALABSORPTION IMMUNOLOGY
05290*
MALABSORPTION IN CHILDREN
00354* 00426* 02286* 02296 04000 04312 04322 052
05299 05733 06669* 07112 07200 08112* 08227* 082
08240 08242 08245 08426* 09337 09339
MALABSORPTION PATHOPHYSIOLOGY
00115* 00126* 00223 00354* 00426* 00429* 00431* 004
00434* 00773 00923* 00925* 00971 01048* 01113 011
01248* 01254* 01260* 01372* 01373* 01374* 01376 015
09338 09954
MALABSORPTION TREATMENT
00354* 00425* 00425* 01374* 02287 03240* 03241* 042
04310* 04314 04318 05030 05184* 05206 05289* 052
05296 05382* 05733 06153* 06325 06863* 07188* 071
07196 07197 08237 08247 08820 09239 09323 093
09339
MALABSORPTION TREATMENT,ANTIBIOTICS IN
03226* 03246* 08248
MALABSORPTION TREATMENT,DIETARY
01372* 01373* 01376 02286* 02911* 03231* 04321 052
05308 08239 08245 09326 09328
MALABSORPTION TREATMENT,FOLIC ACID IN
04301* 05309
MALABSORPTION TREATMENT,VITAMIN B12 IN
04201 08746*
MALABSORPTION,AGE FACTORS IN
05719*
MALABSORPTION,ANEMIA IN
00430* 00439 01856* 02291 03235* 03240* 05206 053
05827* 07201 08248 08746* 09176

ORPTION,BIOCHEMICAL DIAGNOSIS OF
52* 03239* 04314 05867* 07194*
ORPTION,BLIND LOOP-INDUCED SECONDARY
45* 02664* 03981 04124 04242 04729* 06219* 06844*
38* 08228*
ORPTION,CARBOHYDRATE
23*
ORPTION,DISEASES ASSOCIATED WITH
54* 00429* 01177* 02281* 04029* 04316 04321 04402
03 04404 05289* 05293 05305 05306 05864* 05867*
24 07655 08103 08243
ORPTION,DIVERTICULITIS-INDUCED SECONDARY
39
ORPTION,DRUG-INDUCED SECONDARY
13 02090 02138 03232* 03234* 03237* 03980 04091
05* 04306* 05285* 09329
ORPTION,GASTRECTOMY-INDUCED
34* 01191 02281* 02294 03104* 04178* 04204 04208
66* 04283 04730* 05144* 05145* 05867* 06029 06153*
36* 06863* 07177 07197 08049 08050 08058 08077
85 09176 09300
ORPTION,GASTRECTOMY-INDUCED SECONDARY
62 04201 05867* 08041* 09191
ORPTION,INTESTINAL RESECTION-INDUCED SECONDARY
34* 01403* 03246* 04062 04249 04730* 05206 05867*
29 06844* 07183* 07193* 08077 08112* 08133 08230*
ORPTION,MICROORGANISMS IN
96* 02289 03236* 03246* 08238 08248 08881
ORPTION,PARASITIC DISEASE-INDUCED SECONDARY
23* 04024* 04236 04989* 05004 06072 08718* 08889
23
ORPTION,PRIMARY
86* 00354* 00433* 00438 01177* 01251* 01255* 01373*
74* 01451 02274 02280* 02281* 02290 02292 02293
24* 02911* 02913* 02991* 03049 03051 03229* 03230*
31* 03235* 03242* 03243* 03248 03989 04002 04230*
11 04312 04313 04314 04315 04316 04317 04318
20 04324 04941 05070 05284* 05287* 05290* 05291*
93 05295 05297 05299 05305 05306 05307 06323
36* 06844* 06851* 06863* 07189* 07192* 07194* 07196
98 07625* 07838* 08158 08229* 08247 08248 08375*
74 09082 09083 09320 09324 09325 09326 09327
31 09332 09333 09336 09337 09338 09339
ORPTION,SECONDARY
34* 00437 00923* 01082 02279* 02913* 03240* 04308*
15 05305 06863* 07194* 07309* 08192 09429
ORPTION,VITAMIN DEFICIENCIES IN
29* 00427* 00543 04046* 04091 04201 04204 04321
41 05144* 05145* 05179* 05301 05309 05867* 06808
90* 07192* 07194* 07201 08242 08248 08377
ANT ABDOMEN NEOPLASMS
59 07918
ANT ANUS NEOPLASMS
76 01377* 01421 02304* 02342 08297 08345
ANT BILIARY TRACT NEOPLASMS
80 00784 01636* 01654* 01658* 01682 01685 01687
52* 01942 02665* 02692 04657 06030 08675 09527
42 09772 09796 09801
ANT ESOPHAGUS NEOPLASMS
78 00279 00963 00985 01033* 01136* 01137* 01147
52 01160 01969 02008* 02035 02042 02113 02885*
09 03015 03072* 03079* 04126* 04145 04969 05087*
17 05698* 06030 06108* 06109* 07003 07999 08015
28 08719* 08821 09135 09149
ANT GALLBLADDER NEOPLASMS
57* 00778 00784 02685 02986* 06617* 06621* 06622*
27* 06639 07242 08711 09772
ANT GASTROINTESTINAL NEOPLASMS
24 00227 00937* 00948* 00963 00969 00983 01033*
81 01256* 01928* 01943 01967 01987 02292 03023
04* 03923* 04050* 05067 06001* 06009 06030 06055
77 06913 07547* 07559 08719* 08821 09813 09892
ANT LARGE INTESTINE NEOPLASMS
64 00474 00504 00508 00530 01029* 01083 01096
78* 01391* 01392* 01414 01416 01418 01420 01432
57 02183 02320* 02348 02355 02359 02362 03254*
60* 03262* 04065 04333* 04334* 04350 04352 05319*
59 06027 06326* 06330* 06347 06363 06370 07068
21 07226 07232 07234 07240 07242 07260 08253*
65* 08305 08355 09391 09415 09889
ANT LIVER NEOPLASMS
48 00205 00650 01556 01557 02480* 02482* 02502
05 02506 02513 02561 02565 03408 03463 03904*
15 05437* 05874* 06456* 06464* 06837* 06876* 06894
38* 07343* 07429 08428* 08433* 08460 08469 08498
20 08645 08719* 09125 09493 09499 09501 09516
62 09656
ANT MESENTERY NEOPLASMS
75
ANT PANCREAS NEOPLASMS
99 01501 01504 01508 02414* 02419 02420 03004
15* 03320 03904* 03918* 04037* 04048* 04386* 04387*
98 05018 05075 06409 07279* 07838* 08045* 08373*
20 08719* 09443 09451 09454 09467

MALIGNANT RECTUM NEOPLASMS
01417 01438 02368 04352 05359 06330* 06350 07206*
08297 09392 09415 09982
MALIGNANT SALIVARY GLAND NEOPLASMS
09831 09931
MALIGNANT SMALL INTESTINE NEOPLASMS
00346* 01029* 01083 01253* 01268 01269 01279 02118*
02132 02145 02184 02986* 03149* 03168 03225 04065
04246 04251 05012 05189 05212 05963 06027 06225
06227 06229 06234 07067 07081* 07086* 07092 07101
07119 07186 08125 08149 08155 08678 08743* 09110
09222 09875 09889
MALIGNANT STOMACH NEOPLASMS
00132* 00332 01096 01169* 01173* 01176* 01185 01190
01196 01201 01226 01228 01233 01335 01998 02052*
02057* 02082 02085 02086 02088 02092 02093 02108
02113 02242 02258 02885* 02886* 02912* 02947 02975*
02986* 03009 03015 03047 03098* 03099* 03105* 03107*
03110* 03111* 03116* 03117* 03123 03131 03133 03140
03141 04065 04191 04225 05173 05863* 05923 06142*
06148* 06150* 06168 06194 06204 07015* 07023* 07029*
07067 07071 07076 08042* 08044* 08056 08058 08075
08076 08093 08096 08106 08620 09056 09170 09174
09193 09212 09213
MALLORY-WEISS SYNDROME
01108 01154 01959 05042 05109 08018 08845 09139
MALNUTRITION
02572 02581* 02828 03075* 03152* 04062 04988* 05501
05985* 05986* 05992* 06033 06071 06220* 06669* 07194*
07198 07199 07276* 07696 07789 08234* 08476 08573
08889 08891 09082 09083 09234 09240 09657 09916
09920 09997
MANGANESE ABSORPTION
07787
MANOMETRY,ESOPHAGUS
00887* 00985 00990 01000 01138* 02006* 02013* 02994*
03070* 04127* 04774*
MARASMUS
04988*
MARKER STUDIES
00044* 00117* 00142 00169 00598* 00621 00670 00856
00951* 00961 01010 01237 01812* 01870* 01873* 01879*
01884 01897 01920 02086 02315* 02350 02413* 02557
02693 02753* 03154* 03301* 03903* 04802* 04861* 04864*
04877 04899 04927 04954 04967 04981* 05079 05202
05440* 05727 05842* 05843* 05844* 05846* 05852* 05951
05955 05961 06171 06264* 06347 06352 06684 06782*
06839* 06881* 06885 06908 06909 06966 07085* 07356
07633* 07825 07832 07851* 07866* 07895 07901 07903
07906 07928 07938 07940 07960 07966 07974 08007
08143 08287 08484 08563 08697 08751 08929 08943
09042 09094 09101 09104 09132 09169 09235 09566
09753 09871 09950 09982 09996
MECKELS DIVERTICULUM
01265 02146 03169 03170 03171 04870 04976* 06890
07109 08124 08131 08147 08153 08266* 08729* 08743*
08823 08909 09231 09837
MEDICATIONS IN TREATMENT OF PEPTIC ULCERS
00380* 00391* 00392* 00394* 00404 00409 00412 01296*
01300* 01318* 01319* 01320*
MEGABOWEL
01389* 02313* 02316* 02321* 05210 06221 07259
MEGACOLON
00186* 00455* 00471 00511 01389* 02313* 02316* 02321*
03252* 03289 04329* 04359 04370* 05311* 05324 05331
05336 06343 06372 07211* 07219 07224 07236 07246
07863* 08249* 08250* 08269* 08278 08335 08369 08371
08792 09040 09146 09368 09375 09405 09913
MEGAESOPHAGUS
06111* 06136 07992* 08023
MELANOSIS COLI
02328* 08338
MELENA
00259 01994 02218* 08130 08154 08161 08807 08909
08912 09479 09850 09906
MENETRIERS DISEASE
01242 03127 03940 06192 06197 07045 07046 07872
08085 08108 08888
MESENTERY
00219 00245 00372 01518 09228
MESENTERY ANGIOGRAPHY
00069* 00135 00219 02944 02997* 04967 07286 07606
07903 07926 08130 08738* 08850 09420 09457 09517
MESENTERY ANOMALIES
08120*
MESENTERY CIRCULATION
00791 00792 00818* 00976 01276 01277 02640 02973*
02997* 03148* 03167 03171 03936 04069 04252 04353
05562 05670* 05844* 06666* 06681 06834 06888 07024*
07115 07264* 07550* 07555 07606 07922 08130 08717*
08738* 08867 09091 09828 09857
MESENTERY CYSTS
06066 08818 08843

MESENTERY DEVELOPMENT
09822
MESENTERY DIAGNOSIS
00135 00219 02944 02997* 04967 07606 07903 07926
08130 08154 08738* 08850 09457 09815 09822 09857
MESENTERY DISEASES
08312
MESENTERY IN CHILDREN
08120*
MESENTERY INJURIES
01039*
MESENTERY LYMPH NODES
00198* 02973* 04681 04887 04933 05015 05064 05304
05989* 06164 06322 08808 08843
MESENTERY LYMPHADENITIS
09901
MESENTERY NEOPLASMS
08154
MESENTERY NEOPLASMS,BENIGN
04030* 06066 08154 09888
MESENTERY NEOPLASMS,MALIGNANT
09875
MESENTERY RADIOLOGY
00135 00219 01927* 02944 02997* 04967 07606 07903
07926 08130 08154 08738* 08850 09457 09517 09815
09822
MESENTERY SURGERY
00219 01246* 01276 01394* 01927* 06066 08878 09815
09822 09875 09888
MESENTERY VASCULAR DISEASES
00973 01028* 01248* 01268 01927* 01929* 01953 02997*
04096 04240 04849* 04938 04995 05040 05063 05064
05065 05201 05518 06023 06386 06886 06896 07084*
07115 07553 07554 07555 07556 07887 08154 08738*
08763 08831 08850 08853 08911 09091 09232 09428
09493 09857 09888 09922
MESENTERY,DISEASES ASSOCIATED WITH
01291
METABOLIC BALANCE STUDIES
05846*
METABOLIC CHANGES IN CIRRHOSIS
00096 00732* 00745 00773 02490 02629* 02636* 02751*
03613 03981 04060 04434* 04495 04604* 04607* 04619
04719* 05444* 05480 05592* 05593* 05594* 05599* 05601*
05604* 05609* 05616 05881* 06595 06787* 07337* 07467*
07722* 07761 07862* 08475 08483 08485 08631* 08636*
08637* 08649 08991 09491 09709 09716 09722 09725
09867
METABOLIC CHANGES IN GASTRITIS
07019*
METABOLIC CHANGES IN HEPATITIS
02597* 03524* 03573 03574 03575 03580 03581 04495
04561* 05444* 07337* 07448* 07805 07963 08442* 08593*
08597* 08598* 08601* 08617 08618 08623 08626 09628
09663 09693
METABOLIC CHANGES IN LIVER DISEASES
09555
METABOLIC CHANGES IN PANCREATITIS
07299* 07309* 08375*
METABOLIC CHANGES IN PEPTIC ULCERS
00103* 00144 00298* 01315* 01932* 02260 06743* 07016*
07019* 07135* 07274* 08206 09263
METABOLIC CHANGES IN ULCERATIVE COLITIS
00531* 02365 02391* 05368 09429
METABOLIC CHANGES IN VIRAL HEPATITIS
09491 09668 09669
METABOLIC CHANGES,GASTRECTOMY-INDUCED
01049 01996 02089 02203* 03104* 04166* 06155* 06836*
08043* 08045* 08058 08067
METABOLIC EFFECTS OF MICROORGANISMS
00369 00790* 02790* 04671* 04990* 05180* 07399 07791
07825 07833 07946 08067 08238 08375* 08575 08664*
08841 08881 08893 09853
METABOLIC TOXINS
02637* 03668* 04678 05077
METABOLIC TOXINS OF MICROORGANISMS
04108 05185* 06214* 06533* 06803 06804 06831 07082*
07542* 07748* 07755 08547* 08734*
METABOLISM IN CIRRHOSIS,ELECTROLYTE
03600* 03617 04600* 05604* 05613 05881* 06580* 07471*
07478 08634*
METABOLISM IN CIRRHOSIS,WATER
02512 02658 03617 05609* 05613 08634*
METABOLISM IN GASTROINTESTINAL TRACT,ELECTROLYTE
00444* 01049 01096 01395* 08058
METABOLISM IN PANCREATITIS,ELECTROLYTE
07309*
METABOLISM,ALCOHOL
07827
METABOLISM,AMINO ACID
00085* 00100 00601* 01129 02755* 02841* 02843 03735
03822* 03824* 03833 03870 03876 03933 04234 04447*
04491 04495 04607* 04676* 04798* 04829 05288* 05362*
05592* 05818* 05828 06166 06401* 06711 06807 07061
07672 07744* 07767 07777 07797 07799 07899 08442*
08557* 08576 08593* 08744* 08903 08963 08981
METABOLISM,BILE
00098 00168 00790* 00878* 00896* 00964 01795 01826*
02840* 02857 03060 03820* 03881 04652 05479 05805*
06783* 06785* 07376 07791 07792 07793 07794
METABOLISM,BILIRUBIN
07742* 07743*
METABOLISM,CALCIUM
08885 09237
METABOLISM,CANCER
03463 03875 03876 03877 03878 05865* 06788* 07019*
08903
METABOLISM,CARBOHYDRATE
00432* 01828* 01831* 01913 01939* 02129* 02712 03100*
03116* 03613 03668* 03673* 03675* 03745 04079 04170*
04304 04388* 04393 04397 04434* 04608* 04680 04737
04801* 04826 04829 04878 04988* 05407 05410 05592*
05593* 05605* 05654 05780 06004* 06717 06810 06826*
07132* 07191* 07277* 07371 07629* 07668* 07781 08043*
08947 08986 09333 09444 09668
METABOLISM,CHLORIDE
02817 06208 07747*
METABOLISM,CHOLESTEROL
06827* 07791 07811* 08688
METABOLISM,COBALT
06791* 09861
METABOLISM,COPPER
07467*
METABOLISM,DRUG
06825*
METABOLISM,ELECTROLYTE
02126* 02290 02363 02521 03041 03042 03260* 03669*
03955 04070 04180* 04453* 04863* 05368 05410 05731
07065 07124 07371 07543* 07633* 07636* 07637* 07638*
07688 07747* 08055 08516 08915 08939 09316 09701
09738
METABOLISM,GLUCOSE
05811* 06004* 07277*
METABOLISM,IRON
04678 05681* 08068
METABOLISM,LIPID
01738* 02087 02636* 02700* 02710 02711 02728 02882
03573 03710* 04058 04061 04080 04294 04382* 04673*
04949 05077 05593* 05673* 05841* 06003* 06007* 06406
06735* 06802 06826* 06827* 07188* 07629* 07634* 07640*
07643* 07688 07727* 07752* 07781 07786 07803 08228*
08668 08686 08688 08830 08978 09020 09127 09332
09867
METABOLISM,NUCLEIC ACID
00013* 00801 00802 00879* 00909 01031* 01179* 01707*
02718 02730* 02736* 02846 02975* 04712* 04905 05737*
06218* 06735* 07580 07669 07670 07813* 07815*
METABOLISM,PHOSPHOLIPID
00063* 01513* 02501 03641 04458* 05813* 06774
METABOLISM,PROTEIN
04064 04081 04826 05166 05841* 06315* 07626* 07696
09663 09811
METABOLISM,TRACE ELEMENT AND MINERAL
05233* 05289* 05456* 05681* 06307 07281 07467* 07787
08577 09611 09861
METABOLISM,VITAMIN
05454*
METABOLISM,ZINC
07281
METABOLITES,BILE
00679 01832* 03929 08698
METASTATIC CANCER
00114* 00609 00624 00929* 00937* 00941* 01033* 01170*
01235 01282 01396* 01533* 01583 01861* 01862* 01863*
01928* 02041 02061 02096 02136 02155 02340 02342
02482* 02524 02890* 02916* 02958 03022 03133 03309*
03365* 03382* 03414 03901* 03942 04013 04022* 04048*
04146 04151 04196 04206 04362 04485 04945 05018
05044 05047 05391 05698* 05921 05969 06156* 06164
06171 06204 06234 06347 06359 06363 06464* 06480
06493 06617* 06621* 06788* 06837* 06864* 06963 06996
07007* 07029* 07050 07067 07092 07209* 07338* 07342*
07343* 07559 07869 07879 07950 08047 08070 08071
08111 08257* 08261* 08302 08320 08384 08392 08428*
08487 08498 08505 08514 08720* 08730* 08862 08876
08877 09033 09144 09173 09353 09364 09435 09437
09458 09502 09516 09577 09590 09717
MICROORGANISMS
SEE ALSO AMEBIASIS
SEE ALSO CHOLERA
SEE ALSO GNOTOBIOSIS
SEE ALSO LEPTOSPIROSIS
SEE ALSO SALMONELLOSIS
SEE ALSO SHIGELLOSIS
00452* 01394* 01938* 01938* 02555 02590 02694 03017
03054 03329 03487* 03923* 04602* 04627* 04800* 05385
07292 07909 07971 08531 08904 09331 09871
MICROORGANISMS IN CIRRHOSIS
00741 08646

CROORGANISMS IN GASTRECTOMY COMPLICATIONS
00312*
CROORGANISMS IN HEPATITIS
04569*
CROORGANISMS IN MALABSORPTION
00196* 02289 03236* 03246* 08238 08248 08881
CROORGANISMS IN ULCERATIVE COLITIS
01367* 01466* 02391* 02398 05363* 06377* 06381* 07262*
09423
CROORGANISMS,ABDOMEN
09833
CROORGANISMS,BILE
01648* 01649* 01649* 01662* 02660* 02664* 02667* 03626*
03637 04033* 04111 05639* 05814* 06649 06955 07399
07449* 08609 08656* 09765
CROORGANISMS,ESOPHAGUS
03076* 09140
CROCRGANISMS,GALLBLADDER
03626* 08778 09765
CROORGANISMS,GASTROINTESTINAL
00003* 00005* 00158 00177 00196* 00222 00795 00896*
00946* 01095 01115 01367* 01795 01811* 01883 01922*
01938* 01980 01981 01983 01988 02705* 02708 02727
02984* 02985* 03040 03045 03150* 03882* 03892* 03905*
03974 04014 04035* 04036* 04069 04074 04106 04108
04111 04241 04259* 04411* 04671* 04679 04862* 04887
04913 05006 05019 05035 05077 05526* 05598* 05713*
05780 05838* 05986* 05992* 05996* 05998* 06057 06060
06825* 06829 06830 06831 06832 06907 06915 06916
06952 06953 06954 07027* 07216* 07253 07262* 07544*
07548* 07566 07640* 07791 07830 07833 07859* 07909
07911 07951 08067 08145 08228* 08429* 08664* 08726*
08732* 08734* 08737* 08740* 08747* 08758 08778 08796
08798 08800 08804 08838 08841 08847 08849 08852
08881 08884 08888 08898 08927 09230 09810 09817
09819 09863 09871 09872 09881 09885 09938 09939
09940 09941 09942 09943 09944 09945 09946 09977
09978 09979
CROORGANISMS,LARGE INTESTINE
00369 00790* 00933* 01393* 01694* 01735* 02883* 03892*
04336* 04338 04345 04987* 04990* 05007 05363* 05605*
06147* 06381* 06390 06825* 07210* 07617 07820 08344
08356 08360* 08721* 08898 08904 09024 09025 09937
09938 09939 09940 09941 09942 09943 09944 09945
09946 09977 09983
CROORGANISMS,LIVER
03951 07363 07400 07431 08504 09507 09765 09981
CROORGANISMS,METABOLIC EFFECTS OF
00369 00790* 02790* 04671* 04990* 05180* 07399 07791
07825 07833 07946 08067 08238 08375* 08575 08664*
08841 08881 08893 09853
CROORGANISMS,METABOLIC TOXINS OF
04108 05185* 06214* 06533* 06803 06804 06831 07082*
07542* 07748* 07755 08547* 08734*
CROORGANISMS,NORMAL
00003* 00005* 01694* 01695* 01696* 01735* 01795 01811*
CROORGANISMS,RECTUM
01694* 08338
CROORGANISMS,SMALL INTESTINE
00196* 00348* 00354* 00369 00972 01695* 01696* 01735*
02138 02317* 02664* 03236* 03991 04307* 04913 04977*
04987* 04990* 05006 05184* 05887 06053 06434 06477
07825 08233* 08898 08904 09024 09025 09819 09880
09926 09937 09938 09939 09940 09941 09942 09943
09944 09945 09946 09983
CROORGANISMS,STOMACH
00196* 00369 05134* 06150*
CROSCOPY,ELECTRON
00729* 00811 01015 01287 01451 01513* 01583 01584
01706* 02282* 02576* 02577* 02732* 02988* 03478 03517
03518* 03592*
LK INTOLERANCE
00426* 00923* 01374* 01868* 02051* 02391* 02392* 02393*
03228* 03238* 04325 04880 05293 05364 06145* 06851*
07589 08244 09425 09859 09964
NERAL ABSORPTION
EE ALSO TRACE ELEMENT AND MINERAL ABSORPTION
01732*
NERAL ABSORPTION,TRACE ELEMENT AND
00019 00022* 00026* 00030 00031 00032 00039 00311*
00396* 00425* 00439 00773 00893* 00894* 00978 01037*
01192 01315* 01530* 01640* 01719* 01720* 01726* 01728*
01730* 01732* 01737* 01739 01744 02260 02291 02629*
02738 02750* 02751* 02752* 02753* 02754* 02756* 02802*
02920* 03017 03077* 03234* 03240* 03241* 03301* 03500
03588* 03592* 03679 03723* 03724* 03726* 03733* 03734*
03736 03739 03742 03986 04219 04240 04309* 04310*
04384* 04436* 04602* 04717* 04719* 04724* 04725* 04726*
04727* 04731* 04732 04733 04950 05076 05127* 05207
05289* 05291* 05444* 05629 05729 05733 05846* 06029
06704* 06709 06712 06713 06714 06715 06716 07197
07632* 07641* 07642* 07645 08036* 08110 08236 08525
08531 08937 08942 08951 08988 09106 09330 09534
09709 09845
MINERAL DEFICIENCIES,TRACE ELEMENT AND
01829* 05076 05289* 06441
MINERAL EXCRETION,TRACE ELEMENT AND
03676* 03915*
MINERAL METABOLISM,TRACE ELEMENT AND
05233* 05289* 05456* 05681* 06307 07281 07467* 07787
08577 09611 09861
MITOSIS
05692* 05817* 06218* 06692 06790* 06801 07419* 07706*
07723* 07812* 09642
MOTILITY
SEE ALSO SMOOTH MUSCLE
00785* 02894* 04779* 06702*
MOTILITY MEASUREMENT TECHNIQUES
00068* 00069* 00071* 00075* 00103* 00104* 00123* 00186*
00353* 01172* 01410* 01798* 01800* 01801* 01811* 01815
01816 01817 01855* 01859* 01882 02830* 02833 02834
02972* 03258* 03791* 03793* 03795* 03796* 03798 03899*
04769* 04770* 04774* 04779* 04784 04787 04788 05124
05781* 05782* 05783* 05791* 05801 05936 06730 06731
07657* 07850* 07894 08662*
MOTILITY PHARMACOLOGY
00068* 00076* 00127* 00186* 00353* 00883* 00888 01081
01172*
MOTILITY PRESSURE STUDIES
SEE ALSO ANUS MOTILITY
SEE ALSO BILIARY TRACT MOTILITY
SEE ALSO ESOPHAGUS MOTILITY
SEE ALSO GALLBLADDER MOTILITY
SEE ALSO GASTROINTESTINAL MOTILITY
SEE ALSO LARGE INTESTINE MOTILITY
SEE ALSO PANCREAS MOTILITY
SEE ALSO SMALL INTESTINE MOTILITY
SEE ALSO STOMACH MOTILITY
00009* 00069* 00071* 00072* 00073* 00074* 00229 00263*
00785* 00883* 01172* 01381* 01410* 01786 01796* 01798*
01800* 01801* 01802* 01812* 01813 01816 01817 01821
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04164 04771* 04774* 05079 05080* 05085* 05086* 05781*
05782* 05788* 05789* 05791* 05795* 05797 05936 06110*
06190 06872* 06967* 07001 07498* 07657* 07659* 07661*
07662* 07863* 07986* 08340 09062 09122
MOTILITY,ANUS
01381* 01799* 01804* 02333 03799 04767* 04788 04789
05316*
MOTILITY,BILIARY TRACT
00076* 01104 01353 01812* 02894* 03412 03639 04085
04086 04668 05648* 05782* 05798 05936 06634 06720*
06721* 06878* 07498* 07514 07660* 08051 08443* 08662*
09062
MOTILITY,DRUG EFFECTS ON
03796* 03797* 03800 04777* 04779* 04780* 04786 04884
05648* 05782* 05784* 05785* 05786* 05789* 05790* 05792*
05797 05798 05802 05803 05885* 05933 05956 05967
06080 06107 06620* 06702* 06719* 06721* 06726* 06731
06906 06912 07656* 07657* 07658* 07659* 07661* 07662*
09050 09054 09393 09816 09950
MOTILITY,DRUG TREATMENT OF
01797* 01804* 01867* 08267*
MOTILITY,DUODENUM
05797 05799 05800 05801 05930 06190 06857* 06872*
07545* 09054 09936
MOTILITY,ESOPHAGUS
00070* 00074* 00078* 00079* 00263* 00884* 00887* 00940*
00985 00990 01000 01138* 01139* 01141* 01142* 01155
01157 01158 01800* 01806* 01819 01925* 02013* 02020
02024 02831 02832 03070* 03074* 03076* 03078* 03797*
03899* 04101 04127* 04133 04134 04136 04138 04164
04263* 04784 04786 04969 05080* 05085* 05086* 05090
05095 05101 05124 05126 05785* 05957 06052 06140
06696 06724* 06732 06967* 06971* 06974* 07001 07986*
08023 08025 09147
MOTILITY,GALLBLADDER
00076* 01796* 02663* 02894* 04085 04557* 04776* 04884
05152 05153 05644* 05648* 05660 05788* 05790* 05792*
05796* 05798 05802 05936 05938 05956 05961 06529
06634 06664 06718* 06721* 06933 06957 07529 07540
07661* 07824 07894 08045* 08051 08677
MOTILITY,GASTROINTESTINAL
00068* 00069* 00074* 00080 00123* 00127* 00187* 00193*
00229 00788* 00883* 01725* 01808* 01809* 01811* 01815
01816 01818 01893 02117* 02151 02834 02835 02836
02990* 03757* 03793* 03798 04092 04097 04101 04110
04263* 04779* 04786 04787 04993 05079 05175 05784*
05792* 05803 05847* 05933 06013 06031 06039 06058
06080 06101 06106 06264* 06412 06689 06702* 06719*
06730 07656* 07823 07901 08767 08772 08847 08858
09023 09093 09291 09825 09839 09868 09873
MOTILITY,HORMONAL CONTROL OF
00883* 05786* 05797 05803 08677 08877
MOTILITY,LARGE INTESTINE
00071* 00074* 00079* 00186* 00889 00984 01381* 01402*
01410* 01798* 01801* 01802* 01805* 01808* 01809* 01810*
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03304 03793* 03799 03805 03888* 04086 04373* 04707
04770* 04775* 04777* 04789 05058 05059 05062 05208

MOTILITY,LARGE INTESTINE
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05349 05352 05354 05783* 05788* 05794* 06101 06302
06327* 06719* 06725* 07589 08276 08306 08328 08335
08342 08356 08371 08851 09075 09229 09396 09397
09950
MOTILITY,NERVOUS CONTROL OF
03800 03801
MOTILITY,PANCREAS
04760
MOTILITY,PHYSIOLOGY OF
00073* 00074* 00080 00229 00797 01808* 01813 03794*
MOTILITY,RECTUM
00186* 02333 03799 04768* 04777* 04787 04788 05058
05059 05062 06373 07214* 09075
MOTILITY,SMALL INTESTINE
00007* 00008* 00075* 00077* 00079* 00186* 00353* 00785*
00787* 00881* 00885* 00889 00921* 01796* 01797* 01800*
01803* 01808* 01809* 01813 01814 01820 01821 01867*
02125* 02138 02290 02829* 02911* 03194* 03782 03791*
03792* 03795* 03798 03804 03891* 03898* 04086 04203
04249 04772* 04780* 04781* 04782 04783 04785 04786
04790 04849* 04878 04986* 05196 05198 05205 05207
05210 05781* 05786* 05787* 05789* 05791* 05793* 05794*
05795* 05799 05800 05801 06217* 06302 06687 06722*
06723* 06727 06731 06733 06872* 07060 07078* 07106
07122 07123 07162 07191* 07299* 07657* 07658* 07660*
07691* 07959 08118* 08129 08906 08943 08946 09054
09229 09245
MOTILITY,STOMACH
00046* 00068* 00072* 00073* 00079* 00103* 00104* 00186*
00309* 00786* 00886* 00887* 00888 00890 01048* 01171*
01172* 01176* 01316* 01359 01704* 01796* 01797* 01800*
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03800 03801 03802 03803 04086 04132 04170* 04766*
04769* 04772* 04773* 04869 05156 05679 05771 05787*
05797 05883* 05946 06052 06122 06154* 06201 06236*
06244* 06245* 06282 06726* 06728 06729 06733 06767
06883* 06912 07038 07062 07063 07069 07132* 07589
07658* 07659* 07662* 07679* 07850* 08048 08110 08187
08805 09175 09215 09936
MUCOPOLYSACCHARIDES
00763 00880 01487* 02158 03435 03684 03695* 03771
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05980* 06371 06402* 07138* 07665* 07832 08497 08848
09292 09302
MUCOPROTEINS
02686 03771 03778 03780 03781 03966 03968 05762*
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MUCOSA,ANUS
07605
MUCOSA,BILIARY TRACT
05703 07596* 07673 07711* 07712* 07715* 07717* 07719*
MUCOSA,DUODENUM
02248 03707* 03717 04231* 04271* 04698 04699 04778*
05681* 05685* 07080* 07448* 08101 08200 09202 09256
09273
MUCOSA,ESOPHAGUS
01705 02013* 02807 03074* 03980 04133 05044 05095
05121 05122 05123 05126 06043 06118 06121 06133
06676* 06696 06945 06971* 06975* 06978 07002 07610
07673 07889 09129 09130 09140 09166
MUCOSA,GALLBLADDER
01650* 01666* 01688 01824* 02669* 03743 05643* 05645*
05652 05703 06622* 06671* 07489* 08677 08680 08684
08697 09739 09795 09806
MUCOSA,GASTROINTESTINAL
00003* 00005* 00019 00021* 00827 01045* 01115 01705
01725* 01905 02130* 02295 02720 02725 02738 02747
02762* 02764 02766 02767 02974* 03703* 03722* 03729*
03731* 03738 04046* 04067 04095 04115 04685* 04691*
04738 04983* 04989* 04994 05020 05044 05045 05048
05066 05364 05697* 05705 05728 05865* 05973 06015
06025 06689 06702* 06735* 06792* 07044 07148 07631*
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08740* 08791 08848 09121 09835 09935
MUCOSA,LARGE INTESTINE
00310* 00473 01027* 01080 01144 01378* 01451 01478
01706* 01707* 01802* 01910 02302* 02308* 02318* 02319*
02320* 02343 02358 02384* 02388* 02390* 02745 02777
02935 02983* 03253* 03254* 03304 03669* 03740 03778
03780 03781 03888* 04330* 04335* 04343 04345 04360
04368 04703 04970* 05352 05354 05359 05360* 05362*
05363* 05367 05376 05696* 05697* 05726 05980* 06318
06326* 06336 06337 06356 06376* 06378* 06379* 06386
06389 06390 06394 06675* 06678* 06688 06694 07183*
07210* 07216* 07222 07268 07269 07270 07636* 07637*
07638* 07673 08160 08255* 08256* 08259* 08299 08328
08781 08934 09025 09394 09410 09412 09920
MUCOSA,ORAL
00174 01957 04905 08900 09129
MUCOSA,RECTUM
00443* 00468 01082 01404* 01405* 01406* 01473* 01478
01900 02361 02732* 03255* 03257* 04330* 05344 05688*
06336 06341 06350 06353 06371 06381* 06396 06862*
07272 07944 08226* 08259* 08365 08370 08932 09365
09386 09657
MUCOSA,SMALL INTESTINE
00115* 00198* 00310* 00347* 00817* 00826 00922* 00923*
01027* 01048* 01080 01082 01177* 01255* 01260* 01264
01273 01287 01289 01373* 01374* 01376 01619* 01696*
01705 01708* 01721* 01722* 01723* 01724* 01741 01745
01760* 01910 01923* 01935* 01937* 02116* 02121* 02126*
02134 02139 02165 02166 02175 02274 02279* 02281*
02282* 02287 02290 02324* 02352 02391* 02392* 02393*
02702* 02716 02728 02731* 02733* 02765 02777 02807
02824 02973* 02983* 03002* 03148* 03151* 03152* 03153*
03228* 03231* 03235* 03238* 03239* 03248 03670* 03671*
03673* 03674* 03680 03682 03686 03687 03703* 03710*
03714* 03718 03728* 03729* 03735 03737 03745 03777
03782 03871 03891* 03930 03986 03989 04003 04024*
04051* 04092 04226* 04234 04236 04238 04239 04252
04253 04310* 04311 04312 04313 04315 04319 04323
04326 04671* 04686* 04703 04704 04709 04716* 04733
04736 04737 04782 04783 04790 04868 04987* 05160
05179* 05182* 05183* 05184* 05185* 05188* 05196 05198
05199 05202 05203 05207 05219* 05226* 05228* 05285*
05294 05295 05297 05300 05303 05361* 05386* 05670*
05691* 05696* 05697* 05709* 05712* 05714* 05726 05778
05853* 05886 05980* 06013 06147* 06215* 06216* 06220*
06313 06323 06325 06669* 06686 06691 06692 06694
06700* 06707* 06717 06834 06836* 06881* 07024* 07079*
07080* 07082* 07085* 07112 07116 07122 07123 07162
07182* 07183* 07189* 07192* 07196 07200 07592* 07593*
07594* 07597* 07609 07611 07620 07621 07625* 07626*
07627* 07630* 07634* 07653 07673 07812* 07813* 07819
07856* 07871 07966 08063 08116* 08119* 08134 08135
08136 08142 08160 08223 08225* 08226* 08231* 08232*
08234* 08236 08901 08917 08918 08934 08935 08936
08938 09018 09020 09021 09025 09037 09084 09110
09225 09231 09234 09240 09244 09322 09323 09330
09332 09333 09920
MUCOSA,STOMACH
00001* 00004* 00021* 00061 00105* 00289* 00295* 00298*
00301* 00304* 00305* 00310* 00315 00339 00378* 00384*
00789* 00808 00810 00834* 00835* 00840* 00844* 00845*
00862 00876 00975 01027* 01048* 01051 01080 01082
01166* 01171* 01173* 01176* 01177* 01179* 01180* 01181*
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01702* 01705 01711* 01712* 01716 01727* 01760* 01762*
01781 01784 01787 01924* 02045* 02046* 02057* 02059
02078 02090 02196* 02705* 02709 02741 02770 02779*
02783* 02798* 02799* 02800* 02801* 02806 02807 02813
02971 02992* 03099* 03103* 03112* 03119 03122 03136
03693 03694* 03695* 03697* 03701* 03705* 03708* 03712*
03713* 03715 03732* 03747* 03778 03779 03884* 03890*
03927 03985 04100 04168* 04179* 04192 04213 04214
04216 04220 04290 04300 04310* 04674* 04698 04699
04702 04706 04708 04747* 04757 04761 04764 04765
04876 04958 05005 05133* 05135* 05136* 05142* 05146*
05147* 05154 05155 05157 05166 05168 05169 05172
05215* 05223* 05274 05276 05673* 05683* 05695* 05696*
05697* 05702 05736* 05744* 05745* 05755* 05768 05778
05923 05981* 06013 06016 06043 06069 06146* 06161*
06169 06179 06180 06182 06192 06197 06200 06205
06208 06209 06672* 06673* 06738* 06740* 06743* 06744*
06755 06759 06764 06768 06769* 07009* 07013* 07016*
07017* 07025* 07030* 07031* 07045 07047 07049 07055
07069 07072 07584 07604 07613 07665* 07674* 07676*
07677* 07683 07845* 07872 07885 07889 07896 07916
08044* 08068 08069 08085 08091 08108 08168* 08175*
08190 08207 08762 08780 08931 08947 08951 08952
08953 09097 09109 09114 09179 09180 09181 09183
09194 09195 09197 09198 09199 09205 09206 09214
09215 09302 09573 09737 09935
MUCOSAL INJURIES,STOMACH
00301* 00310* 00819* 01185 01231 02048* 02112 02216*
02982* 03107* 03108* 03120 04168* 05140* 05157 05266
06158* 06159* 06160* 07127* 07994* 08033* 08040* 08081
08168*
MUCOVISCIDOSIS
00433* 00547 00548 00553 00968 01109 01110 01493*
01495 01497 02408* 02410* 02411* 02441 03311* 03313*
03966 03968 04384* 04385* 04389* 04397 04402 04403
04404 04919 05179* 05381* 05385* 05386* 05397 06402*
06405 06863* 07091 07111 07282 07292 07937 07939
08164 08372* 08378 08385 08386 08387 08390 08393
09245 09447 09453 09464 09465
MUCUS
00047* 00378* 00878* 01688 04397 05332 05652 05683*
05750* 05751* 07231 07489* 08387 08677
MUCUS SECRETION,STOMACH
00047* 00398* 00858 00859 00862 01794* 02783* 03694*
03695* 03705* 03757* 03769* 03771 03775 03779 04216
06741* 06750* 06934 07037 07125* 07688

USCLE MORPHOLOGY,SMOOTH
01818
USCLE PHARMACOLOGY,SMOOTH
00007* 00008* 00075* 00881* 00882* 01804* 01809* 01814
01815 01819 02138 02207* 02832 03797* 04116 04707
05790* 05792* 06721* 06727 07264* 07663 07818 08946
09019 09873 09926
USCLE PHYSIOLOGY,SMOOTH
00001* 00007* 00008* 00070* 00071* 00074* 00079* 00881*
00884* 00885* 01381* 01429 01814 01815 01818 01819
02102 02334 03793* 03797* 04116 04117 04760 04780*
04781* 04790 04849* 05095 05706 05778 05781* 05784*
05785* 05790* 05799 05800 06037 06039 06058 06687
06689 06696 06725* 06731 07660* 07663 07818 07986*
08767 08858 08917 08928 09019 09418
USCLE,SMOOTH
SEE ALSO MOTILITY
01821 05128* 05179* 07415 07602 07612
YOMA
01204 03141 06224 09178
YOSARCOMA
00373

AUSEA
01972 05787* 05788*
ECROSIS
01539* 02428 02580* 03336* 03374* 03440 03496 03511
03972 04231* 04252 04353 04406 04435* 04520* 04531
04533 04540 04548 04552 04556 04578 04683* 04849*
05183* 05337 05383* 05415 05422* 05462 05526* 05527*
05543* 05546* 05575* 06428* 06549 06583* 06589 07179
07324 07440 07469* 07513 07556 07706* 08030 08163
08379 08414 08415 08417 08476 08502 08584 08757
08789 09400 09473 09566 09568 09612 09643 09727
09788 09789
ECROTICANS,ENTEROCOLITIS
02308* 02358 03271 03275 04027* 04367 04972* 05183*
05337 08728*
EONATAL JAUNDICE
00636 00637 02666* 03902* 04041* 04439* 04462 04463
04501 04563* 04564* 04572 04574 05595* 06462* 06479
06492 06505 06552 07369 07371 07742* 07743* 07809
07841* 07928 08453 08455 08462 08466 08508 08511
09600 09605 09620
EOPLASMS,ABDOMEN
00937* 01444 02004 02933 02972* 03904* 04056 04937
04991 05920 05964 06046 06068 07239 07558 07568
07952
EOPLASMS,ANUS
02345 09387
EOPLASMS,APPENDIX
08285
EOPLASMS,BENIGN ABDOMEN
07559
EOPLASMS,BENIGN BILIARY TRACT
01690 07495* 07977
EOPLASMS,BENIGN DUODENUM
08146
EOPLASMS,BENIGN ESOPHAGUS
00281 00985 01147 01153 01162 02028 02039 04145
04148 04154 04969 05117 06117 06134 06138 06981
07989* 08008 08012 09134
EOPLASMS,BENIGN GALLBLADDER
02671 03646 07495* 08680 08681 08703 08709
EOPLASMS,BENIGN GASTROINTESTINAL
00224 00948* 01077 04042* 04068 06015 08748 08817
08824 08912 09823
EOPLASMS,BENIGN LARGE INTESTINE
00441* 01083 01437 02153 02305* 02343 02361 03254*
04334* 04340 04352 04354 04356 04361 04366 05060
05319* 06330* 06334 06337 06357 07223 07229 07230
07231 07234 07237 07242 08259* 08281 08308 08323
08336 08347 09395
EOPLASMS,BENIGN LIVER
01580 02502 02506 03371* 03939 04476 06464* 06894
07378 08469 08493 08501 08512 08520 09516 09520
09551 09566 09619
EOPLASMS,BENIGN MESENTERY
04030* 06066 08154 09888
EOPLASMS,BENIGN PANCREAS
00564 01507 01508 02415 04386* 05014 08373* 08748
09233
EOPLASMS,BENIGN RECTUM
00443* 00470 04352 06330* 08273 08288 08336 08352
09199
EOPLASMS,BENIGN SALIVARY GLAND
02910* 09831 09931
EOPLASMS,BENIGN SMALL INTESTINE
00165 00346* 00361 00370 00418 01051 01083 01262
01263 01270 01272 01282 01370 02118* 02123* 02139
02154 03149* 03165 04250 04251 05060 05194 05209
05212 06233 06929 07086* 08101 08122 08130 08131
08151 08161 08743* 09823 09905
NEOPLASMS,BENIGN STOMACH
00132* 00292* 00325 00332 00333 00341 01174* 01182*
01233 01234 02047* 02059 02104 02234 03126 03130
03134 03135 04191 04193 04199 05151 06165 06169
06194 07022* 08046 08053 08057 08084 08101 08104
08106 09178 09193 09199 09203 09208 09216 09823
09905
NEOPLASMS,BILIARY TRACT
00108* 00777 01664* 02674 02696 04632* 04660 07265
07337* 07878 07978 08506 08702 08704 08708 09743
09747 09752
NEOPLASMS,ESOPHAGUS
00147 00276 02022 02037 02038 03078* 06118
NEOPLASMS,GALLBLADDER
05640* 08686 09743
NEOPLASMS,GASTROINTESTINAL
00089* 00200* 00945* 01042* 01055 01430 01487* 01918
01993 02000 02004 02997* 03006 03023 03062 05023
05042 05047 06104 06368 09866
NEOPLASMS,LARGE INTESTINE
00450* 00469 00503 00509 00956 01380* 02356 02372
03067 03269 03281 05332 05357 06026 06171 06368
06889 07256 08285 08315 08316 08324 08337 09346
09352 09389 09394 09419 09893
NEOPLASMS,LIVER
00632 00677 02966 03399 04901 05491 05609* 05898
05965 06478 07441 07738* 07778 07942 08440* 08491
08506 08552* 08995 09013 09500 09614
NEOPLASMS,MALIGNANT ABDOMEN
07559 07918
NEOPLASMS,MALIGNANT ANUS
00476 01377* 01421 02304* 02342 08297 08345
NEOPLASMS,MALIGNANT BILIARY TRACT
00780 00784 01636* 01654* 01658* 01682 01685 01687
01852* 01942 02665* 02692 04657 06030 08675 09527
09542 09772 09796 09801
NEOPLASMS,MALIGNANT ESOPHAGUS
00278 00279 00963 00985 01033* 01136* 01137* 01144
01152 01160 01969 02008* 02035 02042 02113 02885*
03009 03015 03072* 03079* 04126* 04145 04969 05087*
05117 05698* 06030 06108* 06109* 07003 07999 08015
08028 08719* 08821 09135 09149
NEOPLASMS,MALIGNANT GALLBLADDER
00757* 00778 00784 02685 02986* 06617* 06621* 06622*
06627* 06639 07242 08711 09772
NEOPLASMS,MALIGNANT GASTROINTESTINAL
00224 00227 00937* 00948* 00963 00969 00983 01033*
01081 01256* 01928* 01943 01967 01987 02292 03023
03904* 03923* 04050* 05067 06001* 06009 06030 06055
06077 06913 07547* 07559 08719* 08821 09813 09892
NEOPLASMS,MALIGNANT LARGE INTESTINE
00464 00474 00504 00508 00530 01029* 01083 01096
01378* 01391* 01392* 01414 01416 01418 01420 01432
01437 02183 02320* 02348 02355 02359 02362 03254*
03260* 03262* 04065 04333* 04334* 04350 04352 05319*
05359 06027 06326* 06330* 06347 06363 06370 07068
07221 07226 07232 07234 07240 07242 07260 08253*
08265* 08305 08355 09391 09415 09889
NEOPLASMS,MALIGNANT LIVER
00148 00205 00650 01556 01557 02480* 02482* 02502
02505 02506 02513 02561 02565 03408 03463 03904*
04515 05437* 05874* 06456* 06464* 06837* 06876* 06894
07338* 07343* 07429 08428* 08433* 08460 08469 08498
08620 08645 08719* 09125 09493 09499 09501 09516
09562 09656
NEOPLASMS,MALIGNANT MESENTERY
09875
NEOPLASMS,MALIGNANT PANCREAS
01499 01501 01504 01508 02414* 02419 02420 03004
03315* 03320 03904* 03918* 04037* 04048* 04386* 04387*
04398 05018 05075 06409 07279* 07838* 08045* 08373*
08620 08719* 09443 09451 09454 09467
NEOPLASMS,MALIGNANT RECTUM
01417 01438 02368 04352 05359 06330* 06350 07206*
08297 09392 09415 09982
NEOPLASMS,MALIGNANT SALIVARY GLAND
09831 09931
NEOPLASMS,MALIGNANT SMALL INTESTINE
00346* 01029* 01083 01253* 01268 01269 01279 02118*
02132 02145 02184 02986* 03149* 03168 03225 04065
04246 04251 05012 05189 05212 05963 06027 06225
06227 06229 06234 07067 07081* 07086* 07092 07101
07119 07186 08125 08149 08155 08678 08743* 09110
09222 09875 09889
NEOPLASMS,MALIGNANT STOMACH
00132* 00332 01096 01169* 01173* 01176* 01185 01190
01196 01201 01226 01228 01233 01335 01998 02052*
02057* 02082 02085 02086 02088 02092 02093 02108
02113 02242 02258 02885* 02886* 02912* 02947 02975*
02986* 03009 03015 03047 03098* 03099* 03105* 03107*
03110* 03111* 03116* 03117* 03123 03131 03133 03140
03141 04065 04191 04225 05173 05863* 05923 06142*
06148* 06150* 06168 06194 06204 07015* 07023* 07029*
07067 07071 07076 08042* 08044* 08056 08058 08075
08076 08093 08096 08106 08620 09056 09170 09174
09193 09212 09213

NEOPLASMS,MESENTERY
08154
NEOPLASMS,PANCREAS
00108* 00195* 00385* 00539* 00541* 00556 00560 00566
00570 01021* 01487* 02425 02439 03314* 04039* 04049*
05031 05034 05037 05078 05949 05953 06000* 06014
06104 06408 06875* 06910 07581 07582 07583 07584
07865* 07880 07954 07958 08375* 08383 08384 08720*
08730* 08768 08769 08779 08780 08824 08826 08856
08871 09079 09448 09458 09809 09824 09865
NEOPLASMS,RECTUM
00440* 01380* 01409* 01439 04331* 05314* 06368
NEOPLASMS,SALIVARY GLAND
00185* 04119 07927 07967 09932
NEOPLASMS,SMALL INTESTINE
00355 00373 00374 01265 01503 02119* 02120* 03163
03175 05186* 06026 06223 06224 06889 08137 08148
08243 08762 09084 09893
NEOPLASMS,STOMACH
00130* 00261 00293* 00300* 00322 00329 00330 00334
00337 00956 01204 01985 02104 02249 03104* 03121
05128* 05903 05994* 07011* 07032 07057 07846* 07929
08080 08096 08762 09098 09185 09194 09206
NEOPLASMS,VILLOUS GASTROINTESTINAL
00461 00524
NEOPLASMS,VILLOUS LARGE INTESTINE
00446* 01049 01144 02320* 02325* 02337 04334* 05319*
06330* 06357 06949 07242 08317 09399
NEOPLASMS,VILLOUS RECTUM
00440* 00443* 00444* 00446* 00457 00525 02337 02363
05319* 05351 06341 09199
NEOPLASMS,VILLOUS SMALL INTESTINE
04042*
NEOPLASMS,VILLOUS STOMACH
09199
NERVOUS CONTROL OF ANUS
01799* 01804* 04767* 04768*
NERVOUS CONTROL OF BILIARY TRACT
00086* 00772 00839* 00867 01851 02894* 04626* 05793*
06649 07533
NERVOUS CONTROL OF ESOPHAGUS
00887* 01149 01155 01161 01806* 01819 04127* 04136
05086* 05126 05957 06052 06724* 06732 06967* 07005
07992*
NERVOUS CONTROL OF GALLBLADDER
00086* 00839* 00867 01796* 02663* 04085 04626* 04642
05788* 06718* 07145 07540 07616 08443* 09806
NERVOUS CONTROL OF GASTROINTESTINAL TRACT
00081 00127* 00197* 01081 01815 02151 02187 02894*
02895* 02918* 03016 03757* 03792* 04040* 04097 04766*
04924 05250 05677 05678 05732 05784* 05847* 06058
06157* 07579 07616 08767 08858 09291 09812 09962
09966
NERVOUS CONTROL OF LARGE INTESTINE
00889 02302* 02316* 02321* 02323* 02333 02334 02894*
02895* 03805 04329* 04363 04707 05688* 05706 05788*
05794* 06390 06680* 06719* 07663 07863* 08249* 08264*
08267* 08270 08335 08342 09040 09229 09397 09407
09412 09913
NERVOUS CONTROL OF LIVER
01843 02869 03367* 03833 04807* 05807* 06454* 06457*
06816 07017* 07348* 07378 07392 09506 09781
NERVOUS CONTROL OF MOTILITY
03800 03801
NERVOUS CONTROL OF PANCREAS
00193* 00559 00836* 00855 00860 00867 01484* 01505
01523 01751* 01759* 01763* 02407* 03333* 03757* 03783
04407 04427 04692* 04744* 04760 06425* 06774 06775
07145 07305* 09476
NERVOUS CONTROL OF RECTUM
08364
NERVOUS CONTROL OF SALIVARY GLANDS
01766* 01772* 03783 07671
NERVOUS CONTROL OF SMALL INTESTINE
00885* 00889 01813 01814 02894* 02895* 03148* 03792*
03804 04781* 04790 05294 05787* 05794* 06243* 06670*
06722* 06723* 07545* 07602 07612 09229 09913
NERVOUS CONTROL OF STOMACH
00861 00886* 00888 00890 02103 02196* 02789* 02894*
03144 03794* 04132 04769* 04773* 05057 05169 05276
05668* 05679 05787* 05883* 06052 06122 06154* 06191
06201 06206 06243* 06297 06298 06726* 07012* 07017*
07049 07145 07659* 07662* 08110 08187 08193 08805
08950
NERVOUS CONTROL OF STOMACH SECRETION
00043* 00054 00059 00299* 00307* 00324 00831* 00841*
00842* 00843* 00848* 00852* 00856 00857 00859 00862
00867 00868 00871 00872 00873 00874 01358 01359
01362 01749* 01759* 01778* 01794* 02212* 02235 02779*
02779* 02785* 02788* 02789* 02792* 02793* 02794* 02795*
02799* 02806 02809* 02818 02819 02909* 03180* 03705*
03754* 03755* 03757* 03758* 03760* 03776 03784 03978
04086 04256* 04259* 04742* 04748* 04751* 04756 04758
05229* 05250 05258 05679 05748* 05753* 05772 06252*
06258* 06259* 06289 06292 06295 06667* 06738* 06741*
07039 07154 07275* 07604 07675* 07684 08165* 08168*
NERVOUS CONTROL OF STOMACH SECRETION
CONTINUED
08172* 08188 08203
NERVOUS SYSTEM,AUTONOMIC
06303 06454* 06506 06733 07005 07039 07049 07224
07305* 07348* 07392 07458 07602 07612 07760 08216
09093 09844
NERVOUS SYSTEM,CENTRAL
SEE ALSO NEUROENDOCRINE CONTROL
00038 00559 00676 01149 01175* 01189 01759* 01970
02448 02473* 02799* 02809* 02979* 03198 03592* [illegible]
03754* 03758* 03792* 03833 04015 04129* 04321 04381*
04491 04605* 04620 04622 05145* 05404 05426* 05489
05514 05547* 05618 05656 05678 05679 05732 06084
06157* 06206 06243* 06261* 06445 06475 06476 [illegible]
06649 06741* 07017* 07039 07170 07344* 07392 07458
07466* 07572 07662* 07950 08080 08179 08203 [illegible]
08411 08453 08455 08461 08479 08496 08499 [illegible]
08614 08638* 08728* 08870 09264 09639 09640 09658
09710 09812 09842 09844 09917 09985 09989
NEURINOMA
06929 08053 08273 08748 09134
NEUROENDOCRINE CONTROL
00385*
NEUROHUMORAL AGENTS
00002* 00050* 00072* 00127* 00799 00882* 02267 02431
03675* 03762* 03804 03833 04622 04749* 04780* 04807*
05668* 05706 05760* 05776 05784* 05785* 05790* 05797
05807* 05826 06146* 06670* 06722* 06738* 06759 06804
06826* 07049 07264* 07412 07658* 07659* 07702* 07897
08136
NEUROMATOSIS
02341
NEUROMUSCULAR DISEASES
00429* 06058 08335
NONSPECIFIC SMALL INTESTINE ULCERS
01080 01283 01311* 02252 02914* 03151* 03162 03172
03173 04245 05219* 07114 07141 08131 09255
NONSPECIFIC ULCERS
01123 01257* 01442 02326* 02335 06391
NONSPECIFIC ULCERS,SIMPLE
SEE NONSPECIFIC ULCERS
NOREPINEPHRINE
00799 03833 05826 06738* 06759 07702*
NORMAL MICROORGANISMS
00003* 00005* 01694* 01695* 01696* 01735* 01795 01811*
NUCLEIC ACID METABOLISM
00013* 00801 00802 00879* 00909 01031* 01179* 01707*
02718 02730* 02736* 02846 02975* 04712* 04905 05737*
06218* 06735* 07580 07669 07670 07813* 07815*
NUCLEIC ACID METABOLISM,LIVER
00906 01547* 01563 01835 02609 02846 02869 02871
02873 02876 02879 02881 03358* 03439 03442 03489
03496 03497 03512 03550 03585* 03592* 03709* 03815*
03818* 03825* 03841 03843 03844 03845 03847 03848
03851 03864 03865 03866 03869 03874 03875 03879
04469 04508 04510 04518* 04522* 04555 04794* 04796*
05704 05806* 05816* 05817* 06535* 06794* 06805 06806
06807 06823 07419* 07421* 07455* 07456 07703* 07706*
07721* 07724* 07733* 07740* 07766 07795 08559* 08560*
08962 08985 09687
NUTRITION
SEE ALSO ABSORPTION
SEE ALSO ANOREXIA NERVOSA
SEE ALSO KWASHIORKOR
SEE ALSO MALABSORPTION
SEE ALSO OBESITY
SEE ALSO VITAMINS
00302* 00396* 00664 00984 01026* 01037* 01250* 01638*
01666* 01728* 01760* 01857* 01926* 02197* 02205* 02262
02283* 02286* 02626* 02729* 02755* 02838* 02839* 02841*
02978* 03097* 03098* 03330* 03469 03538 03556 03614
03812* 03955 04015 04509 04517* 04535 04595* 04605*
04725* 04815 04939 05000 05238 05286* 05308 05364
05369 05461 05463 05471 05578 05598* 05613 05632*
05737* 05997* 06007* 06040 06041 06069 06093 06211
06216* 06276 06476 06491 06567 06576 06613 06641
07034 07094 07195 07288 07523 07563 07640* 07642*
07761 08132 08192 08403 08607 08741* 08758 08820
08830 08891 09195 09237 09266 09328 09880 09997

OBESITY
01292 02677 03036 04513 05471 05774 06041 06494
07000 07803
OBSTRUCTION,ANUS
04351 07245
OBSTRUCTION,BILIARY TRACT
00051* 00768 00770 00774 00775 01254* 01575 01656*
01657* 01684 01685 01691 01692 01901 02450 02664*
02676 02704* 03597* 03624* 03626* 03629* 03631* 03642
03811* 04043* 04067 04233* 04495 04650 04667 04778*
04813 04837* 04885 05064 05459 05502 05504 05546*
05636* 05638* 05646* 05649* 05650* 05664 05665 05862*
05955 06465* 06466* 06632 06633 06644 06645 07291
07337* 07356 07357 07358 07363 07366 07490* 07504
07524 07526 07536 07539 07879 07914 07938 07947

STRUCTION,BILIARY TRACT
ONTINUED
08152 08156 08387 08423* 08462 08463 08464 08507
08522 08640 08665* 08698 08708 08710 09100 09124
09490 09498 09542 09556 09594 09603 09617 09730
09745 09748 09751 09754 09755 09763 09766 09767
09768 09789 09799
STRUCTION,DUODENUM
06849* 08382 09241
STRUCTION,ESOPHAGUS
01141* 01145 01151 01156 02011* 02043 03009 03069*
03070* 04141 04145 04155 05080* 05099 05102 05115
05125 06120 06123 06127 06137 06140 06141 06974*
08006 08011 08012 09138 09140
STRUCTION,GALLBLADDER
01676 03650 04653 05665 07257 08674 08681
STRUCTION,GASTROINTESTINAL
00200* 00220 00231 00362 00764 00795 01028* 01042*
01044* 01056 01057 01060 01103 01107 01109 01125
01392* 01401* 01430 01495 01655* 01927* 01928* 01950
01967 02000 02117* 02151 02199* 02290 02708 02974*
02977* 03003 03012 03040 03115* 03266 03970 03973
04070 04077 04078 04094 04103 04113 04121 04222
04228* 04299 04976* 04993 04996 05000 05001 05003
05023 05032 05041 05044 05066 05321 06015 06019
06051 06056 06098 06106 06181 06196 06689 06991
07111 07537 07542* 07566 08128 08272 08717* 08734*
08751 08763 08767 08770 08772 08792 08832 08841
08846 08895 09235 09866 09886 09900
STRUCTION,LARGE INTESTINE
00120* 00472 00503 00508 01091 01124 01125 01387*
01395* 01413 01415 01434 01435 01452 01454 01457
01493* 02300* 02327* 02334 02356 02378 03037 03038*
03155 03170 03280 03297* 03997 04101 04107 04328*
04335* 04339 04351 04993 04996 05278 05323 05352
05353 06026 06063 06090 06343 06351 06355 06365
06372 07183* 07241 07244 07256 07565 07579 07863*
08164 08249* 08287 08335 08341 08347 08350 08784
08916 09104 09122 09229 09347 09375 09384 09385
09391 09406 09841 09847 09866 09890 09895 09933
09985
STRUCTION,PANCREAS
03319 04098 04233* 04431 05009 05014 05064 05401*
05904 06232 06427* 06431 06434 07325 07326 09455
09462
STRUCTION,PYLORIC
00286 00572 00786* 00832* 00835* 00868 01049 01172*
01300* 01303* 01747* 01950 02799* 04123 04132 04169*
04180* 04200 04205 04207 04211 04212 04217 04277
05163 05172 05174 05175 05221* 05939 06122 06127
06143* 06172 06187 06288 06849* 07012* 07043 07058
07059 07137* 07166 07846* 08048 08079 08088 08174*
08187 08203 08837 09169 09178 09256
STRUCTION,RECTUM
01438 03280 09985
STRUCTION,SMALL INTESTINE
00245 00352* 00372 01091 01124 01125 01171* 01246*
01247* 01249* 01254* 01263 01277 01278 01279 01290
01291 01294 01876* 01878* 01951 02114* 02139 02140
02143 02187 02190 02346 02981* 03168 03171 03172
03176 03236* 03318 03336* 04043* 04054 04107 04124
04232* 04235 04243 04247 04254 04322 04661 04666
04667 04772* 04877 04986* 04993 04996 05009 05055
05137* 05150* 05186* 05187* 05189 05193 05195 05197
05200 05205 05209 05219* 06036 06063 06090 06223
06226 06228 06232 06415 06625* 07087* 07091 07093
07097 07098 07099 07102 07103 07105 07106 07111
07113 07119 07121 07181* 07183* 07505 07537 07565
07579 08114* 08115* 08118* 08123 08124 08126 08161
08162 08195 06221 08223 08346 08382 08784 08916
09104 09220 09221 09229 09231 09241 09244 09245
09460 09746 09759 09761 09786 09841 09847 09866
09890 09895 09986
BSTRUCTION,STOMACH
01231 04043* 05157 05171 05172 05946 06172 07043
07069 07074 08712 09098 09272 09866
BSTRUCTIVE JAUNDICE
00171 00603* 00670 00782 00996 01113 01247* 01539*
01576 01577 01603 01656* 01658* 01684 01690 01863*
02492 02890* 02916* 02976* 03003 03356* 03555 03631*
03653 03827 03834 03893* 03894* 03902* 03914* 03954
03963 04009 04067 04443* 04445* 04463 04465 04471
04496 04507 04527* 04538 04542 04543 04550 04564*
04647 04650 04657 04839* 04845* 04886 04905 05485
05487 05515 05521 05573* 05646* 05822* 05837* 05870*
05881* 05960 06087 06418* 06449* 06465* 06466* 06483
06505 06590 06621* 06645 06660 06781* 06861* 07358
07360 07385 07387 07415 07417 07426 07449* 07507
07516 07517 07536 07778 07837* 07890 07904 07928
07953 07978 08436* 08507 08656* 08716 09046 09079
09100 09432 09491 09494 09515 09525 09558 09576
09773 09784
DDI,SPHINCTER OF
SEE ALSO BILIARY TRACT

ODDI,SPHINCTER OF
CONTINUED
00076* 00351* 00766 00839* 01022* 01651* 01658* 01684
01796* 02668* 02919* 03177 04085 04086 04419 04484
04658 04778* 05409 05566 05636* 05644* 05647* 05658
05782* 05792* 05793* 05798 06423* 06441 06620* 06624*
06630* 06661 06662 06689 06720* 06914 07327 07498*
07501* 07533 07660* 07878 07879 07977 08051 08659*
08685 08686 08696 08698 08710 09431 09773 09775
09779 09782
OMENTUM
00475 00512 01088 07056 09049
OMPHALOCELES
01040*
ORAL MUCOSA
00174 01957 04905 08900 09129
OXYGEN
01762* 02798* 02816 03444 03445 03451 05528* 05670*
06453* 06472 06565 06728 07344* 07347* 07361 07403
07424 07542* 08554* 08906 08954 08989

PAIN,ABDOMEN
00750* 00953* 01042* 01105 01120 01141* 01283 01953
02115* 02132 02254 02972* 03004 03021 03228* 03244*
03336* 03375* 03937 03944 03973 04052* 04056 04103
04105 04113 04114 04237 04244 04365 04373* 04415
04417 04420 04424 04668 04982* 05054 05055 05178*
05232* 05276 05402* 05871* 06023 06351 06431 06648
06911 06926 07071 07076 07114 07118 07121 07192*
07226 07231 07250 07328 07491* 07541* 07561 07567
07579 07843* 07882 07918 07945 07961 08107 08113*
08195 08198 08303 08304 08305 08399* 08718* 08725*
08733* 08757 08761 08829 08837 08840 09045 09192
09276 09277 09378 09485 09657 09859 09950 09961
09965 09988 09990
PANCREAS
02968 09073
PANCREAS ABSCESSES
01511* 07283 07285 08396* 08414
PANCREAS AMYLASE SECRETION
00579* 00836* 00860 00930* 01489* 01498 01751* 01753*
01762* 01771* 02407* 02409* 02443* 02729* 02782* 02784*
02786* 02805 02970 03330* 03334* 03343* 03711* 03953
03983 04380* 04389* 04424 04427 04921 05391 05765*
05971 06435 06642 06871* 07278* 07293 07311 07690*
07692* 07693* 07695 07853* 07936 07954 07958 07970
08415 09096 09210
PANCREAS ANGIOGRAPHY
00538* 00836* 04893 04967 05904 05911 05949 05953
06409 06840* 06866* 07297 07322 07550* 07847* 07865*
08373* 08382 09052 09057
PANCREAS ANOMALIES
00572 00994 02001 02114* 03318 03319 04391* 05009
05154 06232 06239* 06415 06681 07057 07907 08388
08389 08394 09451
PANCREAS BICARBONATE SECRETION
00930* 01483* 01492* 01751* 01752* 01753* 01759* 01764*
01780 02409* 02797* 02803* 02804 03752* 03753* 04380*
04389* 04750* 04875 05203 05747* 06423* 06772* 06880*
07690* 07692* 07853* 08375*
PANCREAS BIOCHEMISTRY
00044* 00067 00223 00556 00590 00804 00805 00836*
00860 00864 00879* 00895* 00967 01021* 01116 01486*
01492* 01494 01500 01509* 01513* 01514* 01516* 01522
01710* 01730* 01771* 01933* 02416 02417 02430 02449
02453 02454 02729* 02787* 02810* 02825* 02828 02846
02970 03325 03338* 03339* 03340* 03351 03354 03677
03683 03685 03686 03689 03690 03691 03692 03699*
03704* 03711* 03739 03766* 03783 03787 03788 03789
03790 03909* 03965 03969 03971 03974 04382* 04412*
04424 04431 04669* 04675* 04687* 04693* 04734 05203
05382* 05708 05737* 05833 05850* 05932 06400* 06424*
06425* 06770* 06774 06827* 06880* 07276* 07696 07740*
07937 07954 07958 08391 08415 08924 08925 08954
09073 09446
PANCREAS BIOPSY
04390* 04852* 05241 05968 08384
PANCREAS CALCIUM SECRETION
00547 00589 01521 06407 06429*
PANCREAS CALCULI
02435 03324 06407 08388 09079
PANCREAS CANCER
00551 00560 00860 00930* 00988 01033* 01112 01134
01488* 01489* 01491* 01498 01658* 01873* 01889 01890
01942 02412* 02414* 02419 02420 02426 02781* 02927
02975* 03309* 03315* 03320 03321 03328 03333* 03894*
03896* 03904* 03916* 03971 03992 03994 03995 04102
04122 04208 04381* 04386* 04387* 04390* 04395 04396
04400 04405 04851* 04852* 04866 04893 04917 04937
04944 04946 04961 04962 04999 05017 05039 05053
05075 05186* 05384* 05387* 05391 05393 05396 05651
05855* 05911 05953 06077 06398* 06409 06410 06432
06635 06880* 07279* 07286 07287 07291 07297 07585
07838* 07847* 07880 07907 08045* 08374* 08382 08383
08388 08392 08742* 08764 08862 09049 09432 09435
09437 09440 09443 09451 09454 09457 09467 09577

PANCREAS CANCER
CONTINUED
09769
PANCREAS CARBOXYPEPTIDASE SECRETION
00836* 00860 03689 03690 03691 03692 04389*
PANCREAS CHEMICAL COMPOSITION
04669*
PANCREAS CHLORIDE SECRETION
02797* 05203
PANCREAS CHYMOTRYPSIN SECRETION
00058 00119* 00804 00805 00836* 00860 01765* 01771*
02804 04389* 04412* 07838* 07937
PANCREAS CIRCULATION
00538* 01505 02413* 04392* 04692* 04760 04762 05063
05383* 05388 05401* 06070 06420* 06681 06684 06771*
07279* 07280* 07296 07324 07550* 07865*
PANCREAS CYSTS
00524 00561 01496 01504 01510* 01947 02418 02468
03311* 03317 03329 03341* 03350 03975 04386* 04387*
04391* 04419 04420 05390 05395 05402* 06411 06418*
07283 07284 07285 07297 07507 07847* 08379 08380
08381 08385 09431 09439 09441 09442 09448 09449
09459 09460 09461 09470
PANCREAS DEVELOPMENT
02092 04383* 04696 06624*
PANCREAS DIAGNOSIS
00152 00538* 00539* 00544 00552 00557 00565 00567
00568 00574* 00836* 00860 00988 00994 01485* 01488*
01491* 01492* 01499 01504 01507 01780 01873* 01890
01901 02414* 02428 02436 02437 02463 02811 02827*
02898* 02903* 02917* 02926 02927 02934 02946 02960
03311* 03312* 03325 03916* 03971 03975 03992 03994
03995 04001 04386* 04390* 04391* 04396 04398 04400
04405 04406 04843* 04851* 04852* 04858* 04866 04875
04893 04894 04917 04919 04940 04944 04960 04961
04962 04967 05014 05393 05397 05747* 05850* 05855*
05857* 05862* 05878* 05895 05896 05904 05911 05915
05932 05949 05953 05968 05976 06016 06404 06409
06411 06840* 06857* 06863* 06866* 06875* 06880* 06910
06935 06961 07279* 07284 07286 07293 07583 07693*
07838* 07847* 07848* 07853* 07865* 07880 07884 07886
07888 07907 07936 07937 07970 08373* 08374* 08384
08392 08414 08730* 09049 09050 09052 09057 09058
09073 09096 09433 09438 09442 09443 09449 09454
09461 09463 09466 09470
PANCREAS DISEASE ETIOLOGY
06004* 07293 08381
PANCREAS DISEASE TREATMENT
08381 09461
PANCREAS DISEASES
00026* 00434* 00544 00545 00548 00552 00555 00559
00561 00565 00703 01055 01087 01485* 01486* 01491*
01492* 01497 01511* 02428 02437 02903* 03051 03052
03060 03068 03311* 03313* 03974 04402 04403 04404
05070 05392 05397 05398 05857* 05878* 06402* 06407
06416 06508 06840* 07282 07283 07284 07285 07293
07297 07919 08121 08374* 09073 09438 09446 09447
09453 09465 09476 09479 09481 09773
PANCREAS DISEASES,BIOCHEMICAL DIAGNOSIS OF
00107* 00119* 00544 00545 00547 00592 00836* 00860
00930* 01483* 01485* 01489* 01491* 01514* 01519 02416
03325 03909* 07838* 07970
PANCREAS DUCTS
00152 00172 00368 00549 00554 00566 00573 00776
01343 01490* 01717 02438 02810* 04102 04409* 04431
04658 04760 04858* 05401* 06426* 06431 06776 06935
07324 07325 07326 07695 07884 07886 08379 08421
08648 08873 09431 09455 09462
PANCREAS ELASTASE SECRETION
00836* 03338* 03339* 03340* 04412*
PANCREAS ENZYMES
00044* 00067 00223 00540* 00545 00583 00594 00804
00805 00836* 00863 00864 00879* 01021* 01116 01484*
01486* 01489* 01494 01509* 01514* 01516* 01522 01697*
01765* 01771* 01933* 02416 02417 02430 02449 02453
02454 02456 02729* 02787* 02810* 02825* 02828 02987*
03325 03338* 03340* 03588* 03677 03683 03685 03686
03689 03690 03691 03692 03699* 03711* 03739 03787
03788 03789 03790 03965 03969 03971 03974 04382*
04389* 04412* 04424 04431 04669* 04687* 04693* 04722*
04734 04878 04920 04921 04960 04961 05203 05292
05382* 05383* 05395 05415 05522 05737* 05738* 05740*
05779* 05850* 05915 05932 06400* 06412 06424* 06425*
06429* 06434 06435 06440 06528 06568 06770* 06776
06827* 06863* 06924 07190* 07274* 07278* 07288 07290
07295 07296 07301* 07306* 07307* 07308* 07315 07321
07323 07326 07329 07416 07446* 07651 07652 07693*
07696 07754 07836* 07838* 07853* 07914 07937 07958
08377 08395* 08402 08412 08415 08659* 08790 08954
09100 09210 09468 09469 09898
PANCREAS EXTRACTS
03588* 04384* 04722* 05995* 06704*
PANCREAS FISTULAS
00546 01511* 01751* 01753* 01763* 01779* 01949 02432
02827* 03993 04414 07120 07294 07513 07695 09434
09439 09456 09466 09898
PANCREAS HISTOLOGY
02427 02440 02810* 03311* 03339* 03340* 03696* 03699*
03898* 03916* 04388* 04412* 04687* 04692* 05292 05708
06400* 06401* 06428* 06429* 07304* 07328 07665* 08389
08392 08400* 08648 08720* 08780 09073 09455 09462
09473 09480 09487
PANCREAS HORMONES
00541* 01939* 03898* 04393 04684* 05696* 07274*
PANCREAS IMMUNOLOGY
00571 02441 02469 03896* 04388* 05016 06413 09475
PANCREAS IN CHILDREN
00558 01490* 02410* 02411* 02421 03311* 03312* 03318
03319 04384* 04389* 04394 05009 06004* 06415 07091
07276* 07292 08378 08385 08393 09433 09473 09483
PANCREAS INJURIES
02422 02433 02436 03327 04401 05394 05399 06153*
06407 07295 08822 09433 09484 09856
PANCREAS LIPASE SECRETION
00027* 00836* 00860 00930* 01371* 01489* 01759* 02407*
02729* 02784* 02804 03699* 03983 04080 04268* 04389*
04921 05382* 05779* 05850* 05932 06423* 06435 06773*
07278* 07651 07954 08239 08415 09210
PANCREAS LITHIASIS
00550
PANCREAS LYMPHOGRAPHY
02463 03952
PANCREAS METABOLISM
00548 03783 05419 05660 07853* 08418 09210 09481
PANCREAS MORPHOLOGY
00554 01500 01513* 01709* 01715 01717 02413* 02417
03037 03310* 03318 03319 03323 03704* 04399 04696
06401* 06404 06414 06681 06684 06734* 06875* 07288
07305* 07954 09330 09455 09462 09481
PANCREAS MOTILITY
04760
PANCREAS NEOPLASMS
00108* 00195* 00385* 00539* 00541* 00556 00560 00566
00570 01021* 01487* 02425 02439 03314* 04039* 04049*
05031 05034 05037 05078 05949 05953 06000* 06014
06104 06408 06875* 06910 07581 07582 07583 07584
07865* 07880 07954 07958 08375* 08383 08384 08720*
08730* 08768 08769 08779 08780 08824 08826 08856
08871 09079 09448 09458 09809 09824 09865
PANCREAS NEOPLASMS,BENIGN
00564 01507 01508 02415 04386* 05014 08373* 08748
09233
PANCREAS NEOPLASMS,MALIGNANT
01499 01501 01504 01508 02414* 02419 02420 03004
03315* 03320 03904* 03918* 04037* 04048* 04386* 04387*
04398 05018 05075 06409 07279* 07838* 08045* 08373*
08620 08719* 09443 09451 09454 09467
PANCREAS OBSTRUCTION
03319 04098 04233* 04431 05009 05014 05064 05401*
05904 06232 06427* 06431 06434 07325 07326 09455
09462
PANCREAS PATHOLOGY
00552 00553 00556 00564 00566 01498 01513* 02414*
02436 02451 02453 02973* 03068 03310* 03658 04098
04386* 04387* 04394 04396 04398 04406 04684* 05241
05388 05394 05406 05415 06033 06087 06420* 06428*
07281 07283 07289 07304* 07580 07584 07954 07958
07970 08379 08385 08393 08396* 08400* 08414 08648
09073 09438 09442 09446 09448 09449 09459 09473
09477 09479 09480
PANCREAS PERFORATION
01352
PANCREAS PERFUSION
02413* 02431 02437 02804 03898* 05752* 06399*
PANCREAS PHOSPHOLIPASE SECRETION
01517 07308*
PANCREAS PROTEASE SECRETION
00193* 00860 02729* 03785* 04960 05382*
PANCREAS PROTEIN SECRETION
01752* 02782* 02803* 03752* 04684* 04744*
PANCREAS PSEUDOCYSTS
00561 00569 01516*
PANCREAS RADIOLOGY
00538* 00539* 00567 00568 00591 00836* 00967 00988
00994 01021* 01488* 01510* 01519 01889 01890 02415
02463 02898* 02917* 02926 02927 02934 02946 03323
03947 03952 03971 03975 03992 03995 04386* 04387*
04391* 04400 04415 04419 04858* 04893 04894 04917
04944 04962 04967 05396 05854* 05855* 05857* 05878*
05904 05911 05915 05949 05953 05976 05977 06409
06840* 06857* 06866* 06875* 06935 06963 07297 07322
07326 07583 07847* 07848* 07865* 07868* 07880 07884
07886 07907 08373* 08379 08381 08392 08414 09052
09057 09058 09073 09449 09470
PANCREAS RIBONUCLEASE SECRETION
00804 00805 00836* 04684*
PANCREAS SECRETION
00023* 00026* 00052 00188* 00193* 00193* 00540* 00546
00590 00797 00836* 00849* 00855 00860 00863 00864
00866 00867 00869 00875 00877 01092 01494 01514*
01521 01523 01568 01709* 01715 01720* 01752* 01763*
01771* 01779* 01933* 01935* 01939* 01996 02071 02125*

CREAS SECRETION
NTINUED
02279* 02400 02408* 02421 02422 02423 02431 02432
02678 02707 02791* 02803* 02810* 02811 02827* 02970
03004 03323 03331* 03350 03672* 03752* 03753* 03757*
03765* 03766* 03783 03784 03898* 03966 03968 03971
03993 03994 04097 04389* 04399 04407 04420 04669*
04673* 04675* 04687* 04750* 04755 04760 04762 04851*
04878 04961 05004 05183* 05292 05388 05419 05522
05660 05738* 05739* 05740* 05752* 05761* 05765* 05767
05774 05895 05896 05915 06058 06324 06405 06406
06407 06420* 06440 06508 06528 06734* 06767 06770*
06771* 06775 06776 06866* 07274* 07276* 07280* 07281
07282 07288 07290 07291 07296 07304* 07306* 07307*
07321 07323 07325 07326 07416 07446* 07665* 07691*
07692* 07693* 07694 07696 07954 07958 07970 08375*
08377 08378 08391 08393 08780 08819 09210 09446
09468 09898
CREAS SECRETION STUDY TECHNIQUES
03993 06772* 06773*
CREAS SECRETION,DRUG EFFECTS ON
00863 02782*
CREAS SURGERY
00161 00550 00551 00558 00560 00562 00563 00564
00571 00573 01021* 01038* 01049 01059 01092 01134
01170* 01268 01489* 01490* 01496 01501 01502 01505
01509* 01526 01942 01952 02412* 02415 02425 02433
02442* 02457 02468 02781* 03004 03310* 03322 03324
03326 03328 03332* 03333* 03352 04037* 04039* 04308*
04386* 04387* 04388* 04391* 04393 04395 04396 04398
04400 04401 04405 04409* 04414 04420 04687* 04722*
04791* 05014 05037 05039 05382* 05384* 05389 05393
05394 05396 05398 05399 05402* 06398* 06403 06404
06406 06408 06410 06411 06413 06416 06426* 06439
06444 06642 07275* 07277* 07290 07291 07293 07294
07295 07297 07302* 07325 07326 07328 07550* 07583
07694 07868* 08045* 08376 08377 08380 08381 08392
08394 08403 08404 08412 08413 08421 08678 08748
08779 08856 09073 09431 09433 09434 09436 09438
09439 09440 09441 09442 09443 09444 09445 09448
09452 09456 09459 09461 09463 09466 09481 09803
09898
CREAS TRANSPLANTATION
00571 01505 01705 01990 02409* 02429 03310* 03898*
04388* 05051 05388 06399* 06413 07277* 07280* 07550*
CREAS TRAUMA
01038* 01489* 01490* 02442* 03325 03326 03341* 03910*
05394 06404 06427* 07294 07295 09433 09456 09488
NCREAS TREATMENT
05984* 08380
NCREAS TRYPSIN SECRETION
00058 00119* 00804 00805 00836* 00860 00930* 01762*
01765* 01771* 01935* 02407* 02442* 02448 02454 02787*
02804 03330* 03343* 04389* 04412* 04684* 04851* 06429*
06924 07692* 07693* 07954 08400* 09210
NCREAS ULTRASTRUCTURE
01500 02427 02430 02440 03314* 03696* 03699* 03704*
04399 04692* 05708 06414 07665* 08720* 08780 08924
08925
NCREAS,AGE EFFECTS ON
01506 02442* 03323 03677 04693* 04696 06414
NCREAS,ALCOHOL EFFECTS ON
06774 07695
NCREAS,CLINICAL STUDIES OF
00193* 00538* 00545 00553 00557 00568 00574* 01486*
01491* 01506 02418 02423
NCREAS,DISEASES ASSOCIATED WITH
00062* 00385* 00540* 00543 00569 00719 00771 01121
01692 01939* 02465 02917* 03060 03068 03351
NCREAS,DRUG EFFECTS ON
02827* 03711* 04380* 04383* 04399 04407 05765* 06416
06775 07690* 08395*
NCREAS,DRUG TREATMENT OF
02455 02460 02466 04418 05416 06446 07307* 07315
07319 07320 07321 07323 08376 08406 08407 09474
09484
NCREAS,EXPERIMENTAL STUDIES OF
02432
NCREAS,HORMONAL CONTROL OF
00044* 00099 00193* 00836* 00860 00869 01484* 01494
01505 01512* 01521 01523 01715 01751* 01752* 01759*
02409* 02699* 02782* 02786* 02787* 02791* 02803* 02810*
02827* 03672* 03752* 03753* 03765* 03766* 03777 04380*
04389* 04399 04407 04677 04750* 04762 04875 04961
05403 05522 05737* 05739* 05740* 05747* 05752* 05761*
05774 05895 05896 06407 06772* 06773* 06775 07289
07321 07323 07665* 07690* 07691* 07692* 07695 08375*
08400*
NCREAS,NERVOUS CONTROL OF
00193* 00559 00836* 00855 00860 00867 01484* 01505
01523 01751* 01759* 01763* 02407* 03333* 03757* 03783
04407 04427 04692* 04744* 04760 06425* 06774 06775
07145 07305* 09476

PANCREAS,TOXIC EFFECTS ON
00579* 00589 01500 01753* 02417 02430 03315* 04383*
04424 04669* 04684* 04687* 06401* 06771*
PANCREATECTOMY
04734 06029 06408 06412 08730*
PANCREATITIS
00584 00584 00648 01057 01104 01254* 01492* 01520
01521 01684 02409* 02434 02469 02708 02970 03338*
03354 03624* 03658 04098 04861* 05617 05649* 06630*
06853* 08195 08374* 08411 08666* 08696 09079 09481
09786
PANCREATITIS COMPLICATIONS
06445 08379 08398* 08402 09485
PANCREATITIS DIAGNOSIS
00107* 00539* 00582 00585 00587 00588 00591 00860
00930* 00949* 00988 01499 01510* 01514* 01515* 01519
01524 01647* 01873* 02443* 02446* 02447* 02449 02450
02463 02960 03001* 03334* 03336* 03341* 03349 03353
03354 03683 03909* 03910* 03947 03950 03952 03953
03962 03965 03969 03971 03972 03983 03984 03992
03995 04105 04415 04416 04417 04424 04851* 04878
04920 04921 04949 04960 04961 04962 04982* 05402*
05406 05407 05408 05413 05415 05417 05420 05854*
05856* 05857* 05880* 05971 05977 06422* 06430 06431
06433 06435 06440 06442 06444 06863* 06871* 06872*
06875* 07297 07304* 07311 07316 07317 07322 07329
07836* 07843* 07853* 07865* 07868* 07884 07907 07930
08379 08398* 08399* 08401 08410 08416 09052 09468
09471 09483 09486 09488 09756
PANCREATITIS DIAGNOSIS,CHRONIC
03608
PANCREATITIS EPIDEMIOLOGY
00574* 00591 02442* 04424 05411 05420 06433 08399*
09486
PANCREATITIS ETIOLOGY
00574* 00575* 00577* 00583 00585 00586 00593 00924*
01509* 01510* 01512* 01516* 01519 01523 01524 01525
01661* 02445* 02450 02459 02462 02467 02781* 03332*
03337* 03342* 03345 03347 03349 03350 03965 04117
04392* 04408* 04409* 04410* 04411* 04412* 04416 04424
04427 04428 04429 04430 04431 04778* 05028 05064
05388 05399 05401* 05403 05408 05409 05411 05413
05417 06407 06417* 06420* 06423* 06427* 06428* 06429*
06430 06431 06433 06434 06441 06442 06444 07294
07296 07307* 07308* 07319 07327 07329 07513 08379
08395* 08397* 08398* 08400* 08401 08405 08408 08415
08434* 09449 09456 09469 09470 09478 09480 09756
09782
PANCREATITIS IMMUNOLOGY
03337* 03608 03896* 06417* 06443
PANCREATITIS IN CHILDREN
00575* 03049 03337* 03341* 03342* 04429 04982* 05409
06422* 06427* 08399* 08418 09477 09480 09483 09485
09488
PANCREATITIS PATHOLOGY
00585 00593 01509* 01510* 03335* 03347 03349 03965
04412* 04416 04430 04431 04760 05401* 05415 05420
05856* 06421* 06422* 06442 07304* 07306* 07307* 07322
08402 08408 08410 08411 08415 08416 08419 08420
09470 09471 09473 09482 09483
PANCREATITIS PROPHYLAXIS
00590
PANCREATITIS SURGERY
00524 00562 00581 00588 00591 01510* 01519 01658*
02447* 03316* 03333* 03341* 03346 03349 03972 04308*
04390* 04409* 04413* 04419 04420 05398 05414 05421
05638* 06422* 06439 07291 07298* 07299* 07300* 07305*
07314 07327 07328 08397* 08403 08413 08418 08419
09470 09471 09482
PANCREATITIS TREATMENT
00562 00577* 00578 00581 00582 00588 00594 01022*
01509* 01510* 01514* 01519 01522 01524 01525 02442*
02443* 02444* 02446* 02447* 02448 02450 02452 02455
02460 02461 02466 02781* 03330* 03331* 03333* 03335*
03336* 03337* 03341* 03343* 03346 03348 03349 03352
03354 03788 03972 04409* 04413* 04414 04417 04418
04419 04420 04421 04422 04425 04430 04658 05402*
05406 05408 05410 05413 05416 05417 05420 05421
06103 06399* 06419* 06422* 06424* 06425* 06426* 06427*
06431 06436 06439 06440 06441 06444 06445 06446
07285 07298* 07300* 07301* 07302* 07303* 07307* 07314
07315 07316 07317 07319 07320 07321 07323 07325
07326 07327 07328 07538 08395* 08397* 08401 08402
08403 08404 08405 08406 08407 08408 08409 08410
08412 08413 08418 08419 08421 08820 09469 09471
09472 09474 09476 09478 09482 09483 09484 09756
PANCREATITIS,ACUTE
00418 00582 00588 00589 00592 00594 00949* 01483*
01491* 01499 01514* 01516* 01517 01519 01522 01523
01524 01525 02413* 02443* 02444* 02446* 02447* 02450
02451 02453 02454 02455 02457 02465 02466 03001*
03331* 03332* 03334* 03336* 03337* 03338* 03340* 03342*
03343* 03348 03351 03352 03353 03354 03475 03672*
03909* 03947 03953 03965 03972 03984 04043* 04209
04299 04392* 04406 04408* 04409* 04410* 04414 04417
04418 04421 04422 04423 04425 04426 04428 04429

PANCREATITIS,ACUTE
CONTINUED
04430 04558* 04920 04972* 05054 05398 05404 05406
05410 05412 05414 05415 05417 05419 05420 05421
05856* 05871* 06234 06418* 06419* 06420* 06422* 06424*
06426* 06428* 06431 06432 06435 06436 06437 06441
06443 06444 06446 07289 07298* 07301* 07303* 07306*
07307* 07308* 07310 07311 07312 07315 07316 07317
07319 07324 07327 07329 07496* 07836* 07843* 07930
08379 08395* 08397* 08404 08406 08407 08408 08409
08410 08411 08412 08416 08417 08418 08419 08420
08685 08725* 09452 09469 09471 09473 09474 09477
09479 09483 09484 09731 09756 09782
PANCREATITIS,AGE FACTORS IN
07299*
PANCREATITIS,ALCOHOLIC
00579* 02445* 03332* 04392* 04409* 04778* 05395 05411
05418 05740* 06423* 06426* 06442 06774 07327 08397*
PANCREATITIS,BILIARY
08405
PANCREATITIS,CHRONIC
00574* 00581 00587 01483* 01484* 01486* 01491* 01513*
01515* 01524 01526 02449 02449 02452 02461 02463
02468 03316* 03330* 03333* 03628* 03766* 03950 03952
03953 03981 04390* 04396 04409* 04413* 04414 04415
04416 04418 04420 04426 04878 05179* 05241 05398
05400* 05402* 05407 05408 05413 05416 05418 05419
05420 05421 05654 05854* 05880* 05971 05977 06414
06421* 06425* 06426* 06428* 06432 06433 06435 06438
06439 06440 06442 06871* 06872* 06880* 07036 07285
07286 07299* 07300* 07302* 07303* 07304* 07305* 07309*
07313 07314 07318 07321 07325 07326 07328 07838*
07958 08375* 08381 08397* 08398* 08403 08406 08411
08413 08421 09470 09472 09475 09476 09482 09485
09486 09756 09845
PANCREATITIS,CIRCULATION IN
02413* 02444* 04760 06421* 06992
PANCREATITIS,CLINICAL STUDIES OF
00583 00584 00586 00587 00592
PANCREATITIS,DISEASES ASSOCIATED WITH
00580 00586 01111 01499 01516* 01517 01518 01521
01661* 02434 02448 02458 03060 03332* 03342* 03344
03345 03672* 03908* 04391* 04392* 04396 04416 04423
04426 04429 04558* 05054 05179* 05288* 05383* 05400*
05404 05407 05408 05412 05414 05867* 06407 06411
06416 06418* 06419* 06421* 06424* 06429* 06432 06438
06992 07290 07299* 07302* 07309* 07310 07312 07313
07317 07318 07324 07558 07836* 08374* 08376 08379
08381 08388 08396* 08397* 08398* 08402 08408 08410
08411 08414 08418 08420 09442 09459
PANCREATITIS,DRUG-INDUCED
00575* 05403
PANCREATITIS,ELECTROLYTE METABOLISM IN
07309*
PANCREATITIS,EXPERIMENTAL
00579* 00590 00593 00949* 01044* 02413* 02424 02444*
02451 02453 02454 02456 02458 02462 02781* 03331*
03335* 03343* 03354 03672* 04098 04408* 04411* 04431
05383* 05401* 05856* 06417* 06419* 06420* 06434 06446
06871* 07298* 07306* 07307* 07308* 07310 07320 08395*
08400* 09487
PANCREATITIS,HEMORRHAGIC
00588 02433 03336* 03337* 03339* 04408* 04411* 04412*
06438 07298* 07312 08379 08408 08411
PANCREATITIS,METABOLIC CHANGES IN
07299* 07309* 08375*
PANCREOZYMIN
00026* 00044* 00119* 00540* 00836* 00859 00869 00877
01483* 01484* 01699* 01751* 01807* 02416 02699* 02804
02810* 02903* 03334* 04380* 04389* 04752* 04753 04762
04961 05736* 05739* 05895 05896 06718* 06770* 06773*
06775 07692* 07838* 08200 09725
PANETH CELLS
03239*
PARACENTESIS,ABDOMEN
SEE ALSO ABDOMEN DIAGNOSIS
00607* 03105* 03910* 05871* 09066 09925
PARASITES AND PARASITIC DISEASES
SEE ALSO AMEBIASIS
SEE ALSO SCHISTOSOMIASIS
00122* 00124* 00125* 00136 00180* 00183* 00190* 00194*
00203 00204 00205 00206 00236 00238 00239 00241
00256 00458 00463 00580 00602* 00675 00680 00936*
00938* 00946* 00997 01003 01018* 01061 01068 01445
01548* 01554 01573 01575 01741 01853* 01923* 01924*
01995 02608 02627* 02641 02985* 03003 03055 03396
03657 05495 05496 05612* 05989* 05992* 06044 06054
06065 06154* 06256* 06450* 06463* 06467* 06487 06553*
06655 07199 07570 08142 08246 08251* 08339 08464
08578 08584 08725* 08869 08909 09002 09080 09084
09087 09559 09619 09634 09637 09652 09657 09659
09830 09843 09849 09863 09881 09952 09972 09981
09994
PARASITIC DISEASE DIAGNOSIS
00125* 00156 00180* 00458 00602* 01575 03943 03946
03957 04001 04066 04072 04075 04084 04104 04109
04365 04503 04930 04998 05046 05459 05499 05877*
05887 05893 05905 06862* 06874* 06877* 06894 07182*
07351* 07414 07417 07515 07590 07877 07920 07971
08490 08723* 08724* 08753 08766 08798 08863 08865
08882 08890 08902 09025 09217 09510 09639 09640
09794 09855 09923 09956 09971 09998 02478* 02479*
02529
PARASITIC DISEASE EPIDEMIOLOGY
00194* 01018* 01853* 02991* 04066 04075 04104 04109
04503 05022 05046 05459 06005* 06054 06065 06072
06079 07182* 07576 07590 08490 08718* 08724* 08753
08834 08868 08880 08882 08883 08889 08890 09819
09879 09884 09915 09955 09973 09976 09984
PARASITIC DISEASE ETIOLOGY
05436*
PARASITIC DISEASE IMMUNOLOGY
04066 04072 04084 05877* 05989* 06874* 07182* 07920
08268* 09639 09819 09829 09980 09998
PARASITIC DISEASE PATHOLOGY
00180* 00247 03063 04236 04363 04365 04493 04989*
05004 05989* 06062 06463* 07116 07182* 07833 08139
08490 08865 08890 09163 09227 09510 09634 09635
09636 09639 09640 09855 09864
PARASITIC DISEASE PREVENTION
00190* 00194* 01061 05022 09819 09919
PARASITIC DISEASE TREATMENT
00124* 00128* 00136 00463 01019* 01061 01068 01548*
01575 01853* 03657 03897* 03905* 03928 03932 03948
03956 03988 04075 04104 04489 04862* 04923 04925
04989* 04998 04998 05459 05499 05982* 06073 06074
06075 06076 06083 06085 06089 06094 06095 06102
06331* 06464* 06511 06655 06877* 07351* 07383 07414
07515 07551 07564 07588 07590 07591 08333 08456
08570 08571 08572 08573 08585 08723* 08724* 08750
08754 08766 08781 08795 08799 08827 08837 08844
08857 08860 08861 08862 08865 08882 08883 08889
08890 08892 08897 09510 09640 09818 09877 09896
09914 09918 09924 09956 09974
PARASITIC DISEASE-INDUCED SECONDARY MALABSORPTION
01923* 04024* 04236 04989* 05004 06072 08718* 08889
09323
PARASITIC DISEASES
SEE PARASITES AND PARASITIC DISEASES
PARASITIC DISEASES IN CHILDREN
03063 05982* 06072 06332* 06422* 08766 08865 08882
08890 08897 09503
PARASITIC DISEASES,BIOCHEMICAL DIAGNOSIS OF
00180* 04072 04084 08546* 08718*
PARASITIC DISEASES,CLINICAL STUDIES OF
00128* 00136 00180* 00256 00463
PARASITIC DISEASES,DISEASES ASSOCIATED WITH
01068 01564 01924* 04493 05004 06332* 06422* 07339*
07570 07992* 08250* 08253* 08339 08521 08671 08833
08882 08889 09163 09354 09503 09541 09622 09635
09878
PARASITIC DISEASES,PARASITES AND
SEE ALSO AMEBIASIS
SEE ALSO SCHISTOSOMIASIS
00122* 00124* 00125* 00136 00180* 00183* 00190* 00194*
00203 00204 00205 00206 00236 00238 00239 00241
00256 00458 00463 00580 00602* 00675 00680 00936*
00938* 00946* 00997 01003 01018* 01061 01068 01445
01548* 01554 01573 01575 01741 01853* 01923* 01924*
01995 02608 02627* 02641 02985* 03003 03055 03396
03657 05495 05496 05612* 05989* 05992* 06044 06054
06065 06154* 06256* 06450* 06463* 06467* 06487 06553*
06655 07199 07570 08142 08246 08251* 08339 08464
08578 08584 08725* 08869 08909 09002 09080 09084
09087 09559 09619 09634 09637 09652 09657 09659
09830 09843 09849 09863 09881 09952 09972 09981
09994 02478* 02479* 02529
PARATHYROID GLAND
05028 07135* 07309* 08856
PARATYPHOID FEVER
SEE ALSO SALMONELLOSIS
08765 08803 08875
PARIETAL-CELL ANTIBODY,GASTRIC
00308* 01180* 02943 03109* 03111* 03113* 06176

PATHOPHYSIOLOGY,MALABSORPTION
00115* 00126* 00223 00354* 00426* 00429* 00431* 00433*
00434* 00773 00923* 00925* 00971 01048* 01113 01115
01248* 01254* 01260* 01372* 01373* 01374* 01376 01589
09338 09954
PAVLOV POUCH STUDIES
00042* 00048* 00049* 00842* 00847* 03770* 05748* 05750*
06749* 07135* 08168*
PENTAGASTRIN
01716 01767* 01768* 01770* 01774* 01788 01790 01791
01809* 01810* 02800* 02809* 03748* 03766* 03768* 03770*
03773 04256* 04740* 04746* 04754 04758 04765 05736*

PENTAGASTRIN
CONTINUED
05746* 05763* 06767 07675* 07680* 07682* 07684 07690*
07835 08949
PEPSIN
00266* 03786* 04680 06833
PEPSIN SECRETION,STOMACH
00041* 00045* 00064* 00275 00297* 00299* 00834* 00837*
00838* 00845* 00846* 00850* 00858 00859 00862 00872
00880 00955 01090 01166* 01296* 01301* 01749* 01754*
01755* 01757* 01758* 01761* 01762* 01767* 01777* 01781
01784 01794* 02657* 02785* 02788* 02789* 02795* 02796*
02801* 02806 02807 02992* 03136 03705* 03760* 03761*
03775 03776 04216 04740* 04756 05742* 05743* 05749*
05754* 05755* 05762* 05764* 05766 05773 06002* 06155*
06166 06258* 06738* 06739* 06740* 06742* 06746* 06748*
06749* 06753* 06754* 06763 06764 06766 06767 06769
06841* 06858* 06902 06934 07063 07446* 07674* 07682*
07684 07688 07854* 08059 08180 08200 09078 09294
PEPTIC ULCER COMPLICATIONS
00182* 00186* 00218 00284 00297* 00387* 00395* 00400
00414 00416 01016 01032* 01036* 01051 01089 01103
01168* 01270 01297* 01302* 01306* 01307* 01312* 01321*
01325 01328 01335 01342 01345 01352 01360 01363
01652* 01655* 01855* 01956 01958 02045* 02194* 02195*
02198* 02201* 02203* 02205* 02224 02227 02230 02231
02232 02237 02239 02253 02257 02263 03004 03044
03182* 03202 03203 03206 03207 03408 03910* 03949
04060 04063 04073 04212 04217 04223 04255* 04260*
04261* 04262* 04269* 04270* 04273 04277 04278 04284
04285 04289 04292 04295 04298 04889 04985* 04997
05076 05124 05214* 05215* 05220* 05221* 05231* 05234
05237 05240 05241 05242 05243 05244 05245 05246
05249 05251 05252 05253 05256 05259 05265 05267
05995* 06002* 06064 06069 06239* 06241* 06243* 06246*
06254* 06257* 06265 06269 06272 06274 06280 06283
06290 06296 06301 06304 06309 07018* 07056 07072
07128* 07133* 07136* 07141 07159 07162 07165 07166
07178 07396 07397 07561 07562 07968 08042* 08166*
08173* 08177* 08186 08189 08193 08194 08199 08202
08209 08214 08669 08700 08730* 08779 08816 08832
08836 08871 09048 09175 09186 09248 09249 09252
09258 09275 09277 09285 09286 09288 09289 09295
09296 09301 09304 09728 09812 09912 09935
PEPTIC ULCER DIAGNOSIS
00053 00374 00382* 00404 00407 00417 00834* 00837*
00922* 00956 00965 00966 01014 01081 01090 01288
01307* 01308* 01332 01357 01362 01797* 01855* 01858*
01859* 01867* 02211* 02221 02222 02235 02237 02899*
03099* 03182* 03215 03748* 03749* 03887* 03907* 03918*
03921* 03944 03945 03996 04004 04105 04258* 04281
04291 04889 05230* 05232* 05246 05248 05261 05263
05265 05879* 05912 05945 06016 06268 06280 06281
06305 06305 06311 06313 06841* 06843* 06850* 06860*
06891 06904 06918 06919 06927 06956 06979 07019*
07020* 07128* 07140 07158 07163 07839* 07846* 07855*
07882 07888 07935 08205 08217 08871 09045 09211
09256 09257 09260 09276 09277 09283 09285 09308
09312 09317
PEPTIC ULCER DIAGNOSIS,ENDOSCOPY IN
00132* 09254
PEPTIC ULCER DIAGNOSIS,RADIOLOGY IN
00132* 00920* 00975 01013 02205* 03191* 04855* 05253
06151* 06251* 06253* 06294 06313 07020* 07125* 07160
07882 07910 08217 09254
PEPTIC ULCER EPIDEMIOLOGY
00414 00415 00419 01055 01081 01301* 01323 01360
01925* 02010* 02011* 02115* 02200* 02230 02235 02991*
03219 04272* 04277 04280 04281 04287 04294 04634*
05221* 05224* 05252 05254 05260 05264 05272 05626
06238* 06242* 06256* 06261* 06268 06753* 07133* 07134*
07136* 07140 07158 07159 08166* 08171* 08194 08204
09257 09265 09299 09317
PEPTIC ULCER ETIOLOGY
00309* 00382* 00389* 00393* 00398* 00407 00410 00415
00419 00861 00936* 00953* 01021* 01036* 01168* 01171*
01247* 01277 01298* 01301* 01303* 01314* 01316* 01317*
01341 01362 01366 01751* 01760* 01781 01899 01925*
02074 02196* 02198* 02200* 02215* 02235 02240 02261
03004 03119 03180* 03197 03198 03768* 03918* 04037*
04100 04212 04255* 04267* 04268* 04270* 04277 04281
04290 04922 04972* 05018 05028 05066 05178* 05187*
05213* 05215* 05223* 05224* 05225* 05230* 05246 05250
05252 05260 05261 05263 05264 05266 05271 05272
05274 05626 05757* 06000* 06014 06208 06238* 06240*
06242* 06243* 06244* 06245* 06247* 06248* 06250* 06256*
06260* 06261* 06262* 06268 06281 06287 06288 06291
06298 06309 06311 06582* 06755 07039 07127* 07130*
07133* 07134* 07135* 07141 07148 07150 07151 07152
07154 07159 07167 07169 07179 07275* 07396 07581
07582 07583 07584 07689 08158 08165* 08167* 08168*
08172* 08175* 08179 08180 08189 08190 08200 08204
08205 08720* 08769 08804 08824 08826 09246 09257
09258 09264 09267 09279 09280 09283 09294 09297
09298 09299 09300 09301 09315 09809 09824 09828
PEPTIC ULCER HISTOLOGY
07126*
PEPTIC ULCER IMMUNOLOGY
06748*
PEPTIC ULCER PATHOLOGY
01175* 01179* 01317* 02074 02271 03061 05142* 06179
06313 07126* 07167 07168 07545* 07968 08207 08948
09257 09262 09279 09306 09312 09316 09317 09318
PEPTIC ULCER PERFORATION
00376* 00400 00414 01005 01036* 01051 01103 01121
01302* 01306* 01312* 01321* 01325 02195* 02209* 02224
02227 02230 02231 02232 02237 02257 03182* 03207
03910* 04255* 04260* 04262* 04270* 04277 04284 04289
04889 05214* 05215* 05220* 05221* 05231* 05234 05237
05244 05245 05246 05251 05253 05259 05995* 06239*
06246* 06265 06272 06296 06301 06304 07056 07141
07165 07178 08173* 08189 08194 08202 08209 08779
08832 08871 09248 09249 09286 09288 09289 09301
09812
PEPTIC ULCER SECRETION
00299* 00383* 03768* 05764* 07274*
PEPTIC ULCER SURGERY
00109* 00297* 00307* 00311* 00312* 00377* 00379* 00381*
00387* 00388* 00395* 00400 00405 00406 00411 00855
00934* 00970 01004 01028* 01036* 01097 01126 01166*
01173* 01178* 01183* 01190 01191 01192 01193 01195
01200 01206 01211 01214 01260* 01278 01298* 01299*
01302* 01303* 01304* 01305* 01306* 01307* 01308* 01321*
01322* 01324 01325 01326 01327 01328 01329 01330
01331 01333 01334 01336 01337 01338 01339 01340
01343 01345 01347 01348 01349 01351 01352 01353
01355 01358 01359 01362 01363 01673 01749* 01859*
01956 02011* 02195* 02199* 02203* 02209* 02215* 02226
02228 02231 02235 02236 02237 02259 02264 02270
02663* 02780* 02793* 02909* 03009 03117* 03182* 03183*
03184* 03185* 03186* 03187* 03188* 03189* 03192* 03196
03197 03201 03205 03206 03207 03212 03221 03222
03223 03224 03776 04040* 04051* 04085 04086 04158
04166* 04178* 04181* 04194 04198 04201 04208 04210
04256* 04260* 04261* 04262* 04263* 04264* 04265* 04266*
04269* 04274 04275 04281 04282 04283 04292 04293
04296 04297 04299 04742* 04743* 04748* 04758 04773*
05031 05034 05037 05053 05130* 05152 05153 05216*
05217* 05220* 05221* 05222* 05225* 05227* 05229* 05231*
05233* 05234 05236 05237 05240 05244 05245 05246
05247 05248 05249 05251 05253 05255 05257 05258
05259 05261 05262 05265 05267 05273 05695* 05758*
05840* 06018 06153* 06157* 06168 06186 06239* 06241*
06246* 06248* 06251* 06254* 06263* 06264* 06265 06266
06269 06270 06271 06272 06275 06277 06278 06279
06280 06283 06285 06286 06288 06289 06293 06295
06296 06297 06299 06300 06303 06304 06305 06306
06308 06309 06310 06311 06312 06726* 06883* 06950
07010* 07026* 07062 07115 07128* 07129* 07132* 07138*
07139 07140 07145 07146 07147 07149 07151 07153
07155 07163 07164 07166 07168 07177 07178 07329
07849* 07885 08050 08082 08141 08165* 08169* 08170*
08172* 08173* 08174* 08177* 08180 08181 08182 08183
08184 08185 08186 08187 08188 08192 08193 08194
08197 08201 08202 08203 08205 08209 08210 08211
08214 08215 08694 08717* 08729* 08730* 08741* 08769
08779 09076 09093 09093 09200 09247 09248 09249
09256 09258 09259 09261 09266 09267 09268 09269
09271 09278 09285 09289 09291 09293 09300 09301
09304 09306 09308 09309 09313 09314 09318 09865
PEPTIC ULCER TREATMENT
00109* 00309* 00313* 00377* 00379* 00384* 00386* 00388*
00390* 00391* 00392* 00393* 00394* 00396* 00397* 00405
00406 00953* 00986 01024* 01025* 01036* 01296* 01302*
01313* 01318* 01319* 01320* 01328 01344 01350 01354
01356 01798* 02195* 02197* 02202* 02204* 02205* 02206*
02223 02227 02228 02235 02237 02252 02257 02264
02894* 02897* 03184* 03202 03204 03207 03208 03221
03223 03982 04268* 04275 04276 04281 04286 04288
04290 04297 04883 04922 04982* 05005 05053 05099
05216* 05218* 05228* 05229* 05231* 05238 05239 05243
05248 05251 05254 05257 05261 05262 05263 05267
05268 05269 05270 05273 05763* 06000* 06105 06149*
06151* 06182 06211 06236* 06237* 06247* 06248* 06249*
06250* 06251* 06255* 06258* 06259* 06265 06267 06268
06269 06270 06271 06273 06274 06276 06280 06281
06284 06289 06292 06294 06297 06302 06305 06310
06754* 06763 07038 07064 07115 07125* 07142 07143
07144 07148 07149 07153 07155 07156 07157 07160
07161 07163 07170 07171 07172 07173 07174 07175
07176 07177 07180 07675* 08169* 08174* 08178* 08188
08191 08194 08197 08198 08202 08209 08210 08211
08729* 08741* 08837 08871 08879 09248 09251 09254
09256 09259 09260 09261 09262 09266 09270 09273
09274 09278 09284 09285 09291 09298 09301 09303
09304 09308 09309 09311 09313 09318 09816 09842
09894 09911 09927
PEPTIC ULCERS
00195* 00773 00843* 00846* 01100 01129 01172* 01310*
01312* 01346 01932* 01982 02016* 02074 02207* 02208*
02220 02238 02241 02242 02247 02249 02251 02265

PEPTIC ULCERS
CONTINUED
02271 02957 03061 03192* 03199 03209 03219 04025*
04979* 05134* 05276 05795* 05961 06021 06238* 06251*
06252* 06282 07000 07015* 07073 07131* 07453* 08054
08102 08158 08206 08207 08207 08208 08208 08213
08217 08725* 08856 08909 09290 09305 09310 09845
09922
PEPTIC ULCERS IN CHILDREN
00382* 00387* 01328 01361 01363 02225 03049 04281
06250* 06268 06272 07159 07165 07882 08194 08203
08214 08217 08729* 09301
PEPTIC ULCERS,AGE FACTORS IN
01028* 01312* 01317* 01347 01354 01360 01925* 02200*
04255* 05264 06241* 06248* 06257* 07136* 07150 09295
09311
PEPTIC ULCERS,ANASTOMOTIC
01005 05213* 06248* 07128* 07160 07164 09274
PEPTIC ULCERS,BIOCHEMICAL DIAGNOSIS OF
00144 01129 03918* 08206 09077 09078
PEPTIC ULCERS,BLEEDING
00182* 00395* 00416 01016 01032* 01297* 01312* 01321*
01328 01360 01956 01958 01961 02045* 02194* 02195*
02198* 02205* 02213* 02217* 02237 02239 02263 02998*
03004 03044 03182* 03202 03203 03206 03408 03949
04063 04073 04212 04255* 04260* 04261* 04262* 04269*
04270* 04273 04277 04285 04292 04295 04298 05221*
05249 05256 05265 05267 06002* 06177 06241* 06243*
06246* 06254* 06257* 06274 06283 07133* 07396 07968
08193 08424* 08448 08779 08813 08816 08832 08836
08842 08845 09048 09252 09275 09285 09295 09296
09479 09905 09907 09908 09909 09912 09927 09935
PEPTIC ULCERS,CIRCULATION IN
00396*
PEPTIC ULCERS,CLINICAL STUDIES OF
00144 00377* 00380* 00398* 00410 00412 00415 00416
02232
PEPTIC ULCERS,DISEASES ASSOCIATED WITH
00284 00385* 01034* 01168* 01242 02201* 02235 02253
02998* 03047 04280 05131* 05144* 05221* 05241 06195
06242* 06256* 06286 06290 06508 06757 07134* 07136*
07137* 07152 07154 07155 07162 07179 07180 07274*
07304* 07327 07545* 07580 07689 08166* 08167* 08171*
08179 08196 08199 09728
PEPTIC ULCERS,DRUG TREATMENT OF
03190* 03195 03198 08198
PEPTIC ULCERS,FREEZING TREATMENT OF
01356 04216 05270 06160* 09911
PEPTIC ULCERS,GASTRECTOMY-INDUCED
00377*
PEPTIC ULCERS,HORMONAL CONTROL OF
00410 01319* 01344 01758* 01925* 02202* 02235 02240
04289 05264 05271 09315
PEPTIC ULCERS,MEDICATIONS IN TREATMENT OF
00380* 00391* 00392* 00394* 00404 00409 00412 01296*
01300* 01318* 01319* 01320*
PEPTIC ULCERS,METABOLIC CHANGES IN
00103* 00144 00298* 01315* 01932* 02260 06743* 07016*
07019* 07135* 07274* 08206 09263
PERISTALSIS
00263* 04777* 05786* 05967 06719* 07069 07078* 07850*
07901 08111 08335 08767 09215 09256 09936
PERITONEOSCOPY
00611 00981 04011 07874
PERITONEUM
00247 00512 00960 01041* 01391* 01902 06024 06035
07940 08650 08905 09655 09774 09969 09981 09987
PERITONEUM DIAGNOSIS
01974 03645 05002 05026 09235 09929
PERITONEUM RADIOLOGY
SEE PERITONEUM DIAGNOSIS
PERITONEUM ULTRASTRUCTURE
04984*
PERITONITIS
00244 00371 00376* 00424 00610 00645 00752* 00939*
00960 01028* 01045* 01049 01103 01112 01114 01254*
01278 01368 01397* 01669 01860* 01927* 01974 01979
01997 02136 02187 02667* 02669* 02708 03005 03040
03179 03182* 03261* 03923* 03972 04027* 04034* 04071
04107 04195 04198 04209 04239 04247 04299 04645
04662 04861* 05041 05135* 05197 05320* 05980* 06026
06068 06329* 06988 07077* 07118 07217* 07319 07497*
07500* 07558 07938 08128 08150 08212 08266* 08307
08311 08312 08316 08331 08568 08728* 08734* 08751
08757 08763 08770 08785 08806 08848 08895 08897
08906 08916 09066 09212 09235 09351 09370 09398
09565 09623 09630 09761 09786 09790 09812 09833
09846 09853 09876 09886
PERITONITIS ETIOLOGY
09885 09929
PERITONITIS TREATMENT
00960 03645 04034* 05199 05394 05995* 07207* 07253
08789 08810 08905 08906 09852 09885 09929 09949
PERITONITIS,BILE
01044* 03645 03883* 04033* 04662 04984* 04992 06621*
06658 06665 06964 08671 09785
PERNICIOUS ANEMIA
00308* 00320 00321 01180* 01963 03241* 04193 04850*
05056 05127* 05129* 05148* 05149* 05155 05884* 06162
06176 06673* 06844* 07030* 07031* 07068 07856* 07857*
08076 08090 08242 09098 09176 09180
PERSONALITY FACTORS
00382* 04302* 05230*
PEUTZ-JEGHERS SYNDROME
00364 01256* 02133 02305* 02343 04082 04334* 04340
05194 08123 08140 08147 08337
PHLEBOGRAPHY
01591
PHOSPHATASE,ACID
00891* 00892* 01541* 03872 04810 04811 07751* 09556
PHOSPHATASE,ALKALINE
00166 00757* 01255* 01852* 02479* 02824 02858 02916*
03357* 03365* 03531 03681 03685 03962 04033* 04813
04843* 04845* 04879 05142* 05305 05306 05616 05666*
05687* 05919 06466* 06621* 06799* 06852* 06853* 06870*
06923 06928 07369 07448* 07751* 07754 07829 07837*
07840 07941 07962 08103 08135 08441* 08530 08586
09017 09079 09102 09458 09556 09576 09797
PHOSPHATASES
00011* 00622 00789* 00910 01563 02655 03239* 04451*
05160
PHOSPHATE
00608* 02616 04941 05071
PHOSPHOLIPASE SECRETION,PANCREAS
01517 07308*
PHOSPHOLIPASES
04680
PHOSPHOLIPID METABOLISM
00063* 01513* 02501 03641 04458* 05813* 06774
PHOSPHOLIPID METABOLISM,LIVER
00912 01648* 02880 03852 03867 04508 04521* 04541
04682 04683* 04816 05804* 05805* 06779* 06821 06824
07705* 07784 08969
PHOSPHORUS
05233*
PHOSPHORUS ABSORPTION
00032 00978 01315* 02802* 03401 09458
PHOSPHORUS EXCRETION
01245
PHYSICAL TREATMENT OF GASTROINTESTINAL TRACT
01005 09860
PHYSIOLOGY OF MOTILITY
00073* 00074* 00080 00229 00797 01808* 01813 03794*
PIGMENTATION
02328* 05179* 07398
PINEAL GLAND
07756
PITUITARY GLAND
06468* 06757 07127* 07152 07169 07623* 07665* 07698*
07731* 08856 09514
PLASMA PROTEINS
00064* 00171 00193* 00439 00718 00739 00796 00908
00927* 00928* 01002 01025* 01082 01261* 01376 01530*
01537* 01543* 01578 01581 01616* 01627 01631 01919
01993 02049* 02063 02283* 02298* 02381* 02389* 02394*
02404 02410* 02411* 02471* 02472* 02479* 02534 02581*
02581* 02582* 02597* 02602* 02955 02965 03529* 03579
03584* 03586* 03842 03859 03901* 03914* 03934 03958
03959 03960 03961 03976 03981 03991 04020 04021
04058 04062 04112 04385* 04443* 04444* 04478 04479
04504 04581 04596* 04614 04650 04872 04927 04932
04947 04948 04952 04977* 04980* 05074 05223* 05242
05297 05427* 05434* 05464 05478 05543* 05546* 05548*
05576* 05583 05601* 05606* 05622 05637* 05672* 05673*
05806* 05809* 05818* 05822* 05831 05832 05849* 05926
05970 05999* 06025 06129 06155* 06176 06220* 06286
06380* 06394 06400* 06443 06498 06513 06524 06526
06533* 06557* 06559* 06561 06572 06591 06612 06615
06621* 06651 06707* 06777* 06793* 06820 06824 06845*
06873* 06901 06939 07051 07053 07060 07085* 07119
07204 07264* 07276* 07336* 07351* 07355 07362 07379
07411 07424 07450* 07451* 07467* 07699* 07723* 07745*
07770 07837* 07862* 07870 07913 07939 08208 08246
08425* 08452 08465 08477 08486 08539* 08563 08565
08598* 08617 08888 08913 08977 09046 09450 09518
09633 09693 09923
PLASMA PROTEINS,LIVER
00900 01297* 02706* 03377* 03533 03534 03608 03610
03615 07752* 08561* 08603*
PLETHYSMOGRAPHY
01184*
PLUMMER-VINSON SYNDROME
09160
PNEUMATOSIS CYSTOIDES INTESTINALIS
00350* 00375 00507 03236* 04123 08330
POLYPOSIS
00258 00341 00342 00343 00501 00502 00504 00505
00508 00510 01445 01940* 02995* 03053 07235 07917
07935 08078 08140 08762 09408 09419

POLYPOSIS,RECTOCOLONIC
00519 00519 04082 04334* 04340 04361 05335 07208*
07213* 07249 08259* 08271 08288 08315 08337 09199
09346 09352 09897
POLYPS
SEE ALSO PEUTZ-JEGHERS SYNDROME
04334* 05319*
POLYPS,ESOPHAGUS
08008
POLYPS,GALLBLADDER
00784 02681 03646 08681 08684 08686 08688 08697
08703
POLYPS,GASTROINTESTINAL
01060 01083 01957 07917 08140 09361 09823 09897
09982
POLYPS,LARGE INTESTINE
00224 00349* 00448* 00450* 00500 00501 00502 00503
00504 00505 00507 00509 00510 00530 00948* 00956
01379* 01416 01437 01445 01453 02305* 02312* 02343
02344 02361 02961* 02995* 03254* 04334* 04340 04352
04361 05319* 05335 05346 05357 05359 06049 06328*
06330* 06357 06361 06882* 06921 06949 07208* 07213*
07229 07230 07231 07235 07237 07244 07249 07268
07557 07917 08259* 08271 08315 08317 08336 08337
08368 08729* 09346 09352 09357 09408 09419 09823
09897
POLYPS,RECTUM
00224 00470 00506 00935* 01445 02305* 02343 04340
04361 04931 05319* 05359 06330* 06358 06361 08259*
08336 08352
POLYPS,SMALL INTESTINE
00364 01256* 01274 01280 02133 04233* 05194 07903
08123 08144 08743* 08762 09361 09823 09897
POLYPS,STOMACH
00332 00337 00340 00341 00342 00343 00920* 00948*
01167* 01174* 01175* 01182* 01256* 02076 03099* 03126
03135 04193 04223 06150* 06169 06191 06195 06843*
07032 07045 07068 07073 07329 07916 08046 08084
08089 08101 08246 08389 08762 09098 09197 09206
09216 09823 09897
PORPHYRIA
00913 00914 01835 01970 02491 03845 03973 04502
04506 05062 05444* 05453* 05505 05506 05507 05508
05509 05512 05931 05987* 05988* 06917 07374 07392
07573 07798 08450 08476 08479 08518 08757 09008
09571 09598 09601 09625 09651
PORTACAVAL SHUNT
01528* 01609 01751* 01845 01926* 02032 02033 02054*
02483* 02484* 02517 02638* 02645 02646 03119 03355*
03408 03413 03423 03592* 03593* 03594* 03599* 04010
04064 04454* 04468 04490 04495 04598* 04599* 04603*
04606* 04612 04613 04617 04623 04846* 05100 05424*
05451* 05455* 05513 05514 05518 05592* 05593* 05602*
05603* 05605* 05617 05627 06450* 06503 06504 06579*
06583* 06586 06587 06597 06599 06601 06605 06607
06973* 07331* 07332* 07333* 07396 07403 07405 07407
07423* 07468* 07470* 07472 07481 07483 07487 07762
07858* 07922 07984* 08437* 08457 08458 08472 08473
08482 08488 08496 08632* 08635* 08647 08952 08988
09060 09236 09497 09507 09514 09523 09538 09539
09541 09547 09553 09561 09592 09594 09702 09703
09705
PORTACAVAL SHUNT IN PORTAL HYPERTENSION TREATMENT
00264* 00653 00672 00674 01032* 01089 01642*
PORTAL CIRCULATION
01528* 02721 02906* 03360* 03403 03413 03481* 04456*
04467 04526* 04799* 04846* 04848* 04953 05451* 05511
05520 05848* 05869* 05881* 05899 05966 06421* 06450*
06454* 06457* 06459* 06474 06482 06503 06504 06518
06578* 06592 06594 06705* 06864* 06948 06992 07331*
07340* 07347* 07353 07375 07384 07396 07403 07405
07406 07407 07412 07481 07486 07487 07499* 07722*
07762 07822 07860* 07922 07955 08458 08535 08536
08633* 08635* 09038 09059 09507 09519 09523 09552
09613 09658 09705 09708 09828
PORTAL HYPERTENSION
00051* 00226 00601* 00620 00654 00700 00773 01089
01112 01117 01590 01845 02032 02033 02036 02444*
02452 02483* 02496 02511 02529 02569 02628* 02638*
02650 02736* 03379* 03459 03622 04248 04603* 04938
04997 05452* 05482 05492 05518 05556 05593* 05596*
05597* 05598* 05601* 05611* 05625 05627 05671* 05881*
06096 06421* 06503 06504 06510 06610 06611 06968*
06992 07254 07344* 07368 07429 07481 07922 08458
08463 08503 08526 08530 08534 08642 09528 09565
09705 09708 09725 09727 09728 09902 09908
PORTAL HYPERTENSION DIAGNOSIS
00942* 01862* 01930* 03355* 03936 04449* 04621 04916
05848* 05869* 06467* 06474 06601 06840* 06948 06960
07889 07897 08471 09060 09115 09733
PORTAL HYPERTENSION DIAGNOSIS,RADIOLOGY IN
01117 01889 02906* 03379* 04621 05966 06421* 07860*
08448 09059
PORTAL HYPERTENSION ETIOLOGY
01538* 01926* 01930* 02644 03474 03502 03504 03599*
04449* 04515 04621 05607* 06474 06588 07318 07331*
07393 07405 07406 08448 08459 08640 09059 09496
09512 09521 09541 09548 09575 09707 09730
PORTAL HYPERTENSION IN CHILDREN
06970* 08473 08729* 09496 09538 09539
PORTAL HYPERTENSION PATHOLOGY
03379* 04621 08647 09528
PORTAL HYPERTENSION TREATMENT
00607* 00672 00673 00674 00747 01642* 01926* 01930*
02021 02473* 02533 03119 03355* 03413 03423 03593*
03616 03949 04010 04014 04137 04140 04449* 04454*
04468 04490 04598* 04599* 04606* 04613 04623 04916
04926 05100 05451* 05455* 05603* 05617 05620 05869*
06450* 06454* 06457* 06467* 06578* 06579* 06582* 06583*
06586 06587 06594 06596 06597 06599 06601 06602
06613 06616 06679* 06970* 06973* 07331* 07340* 07341*
07347* 07353 07359 07396 07403 07405 07406 07407
07465* 07468* 07472 07473 07483 07486 07487 07984*
07996* 07998 08380 08387 08448 08457 08471 08472
08482 08632* 08633* 08635* 08640 08647 08729* 08845
09060 09130 09236 09497 09504 09528 09538 09539
09541 09547 09567 09613 09702 09703 09724
PORTAL HYPERTENSION TREATMENT,PORTACAVAL SHUNT IN
00264* 00653 00672 00674 01032* 01089 01642*
PORTAL HYPERTENSION,DISEASES ASSOCIATED WITH
01117 01952 02473* 02640 02644 02645 03046 04449*
05612* 06474 06549 06588 06980 06995 07318 07858*
07998 08380 08448 08635* 09059 09060 09523 09528
09552 09613 09707
POTASSIUM
02521 02738 03029 03600* 03763* 04110 04456* 05219*
07478 08424* 08516
POTASSIUM ABSORPTION
01412 02753* 04674* 05731 07633* 07642* 09737
POUCH STUDIES,HEIDENHAIN
00042* 00043* 00045* 00048* 00051* 00305* 00309* 00807
00841* 00842* 00847* 00857 00870 00871 00873 00874
01314* 01779* 01782 02046* 02780* 02788* 02792* 03181*
03736 03756* 03758* 03764* 03767* 03770* 04740* 04741*
04747* 04748* 04749* 04752* 04761 05455* 05740* 05745*
05751* 05758* 05760* 06760 07682* 07685 08168* 08458
08952
POUCH STUDIES,PAVLOV
00042* 00048* 00049* 00842* 00847* 03770* 05748* 05750*
06749* 07135* 08168*
PREGNANCY
00620 00670 00695* 00782 00913 01555 01571 01606
01834* 02202* 02450 02496 02599* 02604* 03120 03385
03523* 04012 04496 04594 04647 04651 04831 05040
05041 05256 05500 05515 05526* 05602* 05635* 06392
06443 06566 07084* 07121 07364 07373 07376 07377
07447* 07588 07691* 07700* 08128 08334 08601* 08617
08677 09358 09525 09549 09605 09689 09713 09956
PREGNANCY,HEPATITIS IN
00708 03383* 03561 03562 08617
PRESSURE STUDIES,MOTILITY
SEE ALSO ANUS MOTILITY
SEE ALSO BILIARY TRACT MOTILITY
SEE ALSO ESOPHAGUS MOTILITY
SEE ALSO GALLBLADDER MOTILITY
SEE ALSO GASTROINTESTINAL MOTILITY
SEE ALSO LARGE INTESTINE MOTILITY
SEE ALSO PANCREAS MOTILITY
SEE ALSO SMALL INTESTINE MOTILITY
SEE ALSO STOMACH MOTILITY
00009* 00069* 00071* 00072* 00073* 00074* 00229 00263*
00785* 00883* 01172* 01381* 01410* 01786 01796* 01798*
01800* 01801* 01802* 01812* 01813 01816 01817 01821
02829* 02830* 02972* 03078* 03258* 03794* 03800 04138
04164 04771* 04774* 05079 05080* 05085* 05086* 05781*
05782* 05788* 05789* 05791* 05795* 05797 05936 06110*
06190 06872* 06967* 07001 07498* 07657* 07659* 07661*
07662* 07863* 07986* 08340 09062 09122
PREVENTION,PARASITIC DISEASE
00190* 00194* 01061 05022 09819 09919
PREVENTION,SALMONELLOSIS
05021
PREVENTION,TYPHOID FEVER
04907 08736* 09951
PRIMARY MALABSORPTION
00186* 00354* 00433* 00438 01177* 01251* 01255* 01373*
01374* 01451 02274 02280* 02281* 02290 02292 02293
02324* 02911* 02913* 02991* 03049 03051 03229* 03230*
03231* 03235* 03242* 03243* 03248 03989 04002 04230*
04311 04312 04313 04314 04315 04316 04317 04318
04320 04324 04941 05070 05284* 05287* 05290* 05291*
05293 05295 05297 05299 05305 05306 05307 06323
06836* 06844* 06851* 06863* 07189* 07192* 07194* 07196
07198 07625* 07838* 08158 08229* 08247 08248 08375*
09074 09082 09083 09320 09324 09325 09326 09327
09331 09332 09333 09336 09337 09338 09339

PROCTOSIGMOIDOSCOPY
05319* 05344 05346 07874
PROCTOSTOMY
00500
PROLAPSE
02376 03126 05172 06195 06328* 06338 06353 07044
07068 07214* 07243 08303 08762 09372
PROLAPSE,RECTUM
01390* 01425 08279 09342
PROSTHESIS,ESOPHAGUS
00277 02029 03094 04157 04163 05089 07988* 08003
08030 09135
PROTEASE
04680 05832 06790*
PROTEASE SECRETION,PANCREAS
00193* 00860 02729* 03785* 04960 05382*
PROTEIN ABSORPTION
00033 00345* 00426* 01191 01742 02483* 02955 03152*
03229* 04062 04240 04242 04672* 05676 05712* 06324
06379* 06712 06820 07195 07197 07626* 07648 07653
07696 08623 08905 08979 09326 09328 09450 09967
09970
PROTEIN DIGESTION
00033 00062* 00064* 00193* 01765* 02473* 02603* 02755*
02807 02813 02825* 03019 03233* 03769* 03785* 03786*
03789 03930 04062 04230* 04714* 05676 05738* 06769
06833 07577 07914 08208 09967 09970
PROTEIN EXCRETION
06034
PROTEIN METABOLISM
04064 04081 04826 05166 05841* 06315* 07626* 07696
09663 09811
PROTEIN METABOLISM,LIVER
00082* 00604* 00671 00685* 00745 00897 00900 00903
00905 00908 01002 01261* 01547* 01605 01606 01829*
01846 01848 02595 02597* 02623 02839* 02841* 02847
02869 02870 02872 03358* 03409 03476 03488* 03524*
03822* 03841 03842 03843 03844 03845 03847 03849
03859 03864 03865 03866 03870 03876 04014 04519*
04607* 04676* 04794* 04817 04832 04836 04932 04947
04952 04977* 05324 05427* 05505 05512 05548* 05598*
05601* 05622 05689* 05721* 05806* 05809* 05810* 05813*
05818* 05831 05832 05833 06033 06481 06530 06533*
06535* 06557* 06591 06611 06612 06780* 06793* 06795*
06801 06823 06824 07355 07379 07421* 07424 07428
07437 07455* 07699* 07703* 07708* 07716* 07721* 07735*
07740* 07753 07756 07763 07767 07770 07789 07799
07805 07806 07862* 08451 08465 08485 08509 08539*
08560* 08563 08617 08972 08976 08979 08981 08983
08992 09030 09518 09673 09710 09719
PROTEIN SECRETION,PANCREAS
01752* 02782* 02803* 03752* 04684* 04744*
PROTEIN SYNTHESIS
00801 00908 01261* 01297* 01604 01706* 01733* 01846
01849 02730* 03409 03686 03711* 03842 03844 03845
03847 03849 03859 03865 03866 04253 04440* 04478
04553 04672* 04947 04977* 05362* 05427* 05627 05806*
05809* 05817* 05818* 05833 06481 06530 06557* 06685
06737 06780* 06793* 06795* 06820 07424 07467* 07604
07665* 07699* 07753 07757 07763 07770 07789 07799
07800 07805 07813* 08226* 08451 08495 08539* 08612
08983 09518
PROTEIN-LOSING ENTEROPATHY
00149 00345* 00433* 00436 01025* 01043* 01261* 01274
01289 01376 01431 01451 01868* 01937* 02274 02298*
02324* 03231* 03915* 03981 04003 04029* 04030* 04081
04112 04315 04318 04903 04939 04947 04948 04951
04952 05002 05008 05063 05304 05368 05963 05973
06025 06315* 06873* 06903 07085* 07089 07119 07199
07204 07558 07699* 07919 08085 08129 08246 08760
08888 09450 09726 09957
PROTEINS
00345* 03815* 05742* 05749*
PROTEINS,LIVER PLASMA
00900 01297* 02706* 03377* 03533 03534 03608 03610
03615 07752* 08561* 08603*
PROTEINS,PLASMA
00064* 00171 00193* 00439 00718 00739 00796 00908
00927* 00928* 01002 01025* 01082 01261* 01376 01530*
01537* 01543* 01578 01581 01616* 01627 01631 01919
01993 02049* 02063 02283* 02298* 02381* 02389* 02394*
02404 02410* 02411* 02471* 02472* 02479* 02534 02581*
02581* 02582* 02597* 02602* 02955 02965 03529* 03579
03584* 03586* 03842 03859 03901* 03914* 03934 03958
03959 03960 03961 03976 03981 03991 04020 04021
04058 04062 04112 04385* 04443* 04444* 04478 04479
04504 04581 04596* 04614 04650 04872 04927 04932
04947 04948 04952 04977* 04980* 05074 05223* 05242
05297 05427* 05434* 05464 05478 05543* 05546* 05548*
05576* 05583 05601* 05606* 05622 05637* 05672* 05673*
05806* 05809* 05818* 05822* 05831 05832 05849* 05926
05970 05999* 06025 06129 06155* 06176 06220* 06286
06380* 06394 06400* 06443 06498 06513 06524 06526
06533* 06557* 06559* 06561 06572 06591 06612 06615
06621* 06651 06707* 06777* 06793* 06820 06824 06845*
06873* 06901 06939 07051 07053 07060 07085* 07119
PROTEINS,PLASMA
CONTINUED
07204 07264* 07276* 07336* 07351* 07355 07362 07379
07411 07424 07450* 07451* 07467* 07699* 07723* 07745*
07770 07837* 07862* 07870 07913 07939 08208 08246
08425* 08452 08465 08477 08486 08539* 08563 08565
08598* 08617 08888 08913 08977 09046 09450 09518
09633 09693 09923
PROTEINURIA
03981 08746*
PRURITUS ANI
00517 07225 08344
PSEUDOCIRRHOSIS
06940
PSEUDOCYSTS,PANCREAS
00561 00569 01516*
PSEUDOMEMBRANOUS ENTEROCOLITIS
03271 03287
PSEUDOPOLYPS,LARGE INTESTINE
03253*
PSYCHOLOGICAL FACTORS
00006* 00212 00382* 00841* 01046* 02254 02809* 03120
03252* 03294* 03412 04052* 04135 04190 04210 04287
04302* 04379 04924 05073 05230* 05331 05369 05847*
06003* 06054 06084 06101 06333* 06397 07179 07181*
07392 08219* 08290 08349 08367 08496 08767 08794
09093 09183 09260 09640 09838 09887 09917
PSYCHOLOGICAL STUDIES
SEE ALSO PEPTIC ULCERS
SEE ALSO ULCERATIVE COLITIS
00520 00577* 00642 01046* 01456 01476 01477 02386*
04381* 06333* 09305
PYLORIC ANTRUM
00243 00309* 00572 00786* 00856 00859 00886* 01034*
01035* 01170* 01195 01215 01298* 01303* 01308* 01341
01349 01351 01789 01867* 01915 02109 02110 02208*
02250 02283* 02780* 02785* 02789* 02815 02819 02833
03110* 03218 03695* 03708* 03757* 03779 04169* 04171*
04181* 04186 04207 04699 04748* 04749* 04751* 04773*
04958 05138* 05215* 05695* 05740* 05766 05797 06143*
06202 06244* 06245* 06749* 06756 06849* 06850* 07010*
07658* 07849* 07885 08165* 08168* 09178
PYLORIC OBSTRUCTION
00286 00572 00786* 00832* 00835* 00868 01049 01172*
01300* 01303* 01747* 01950 02799* 04123 04132 04169*
04180* 04200 04205 04207 04211 04212 04217 04277
05163 05172 05174 05175 05221* 05939 06122 06127
06143* 06172 06187 06288 06849* 07012* 07043 07058
07059 07137* 07166 07846* 08048 08079 08088 08174*
08187 08203 08837 09169 09178 09256
PYLORIC SPHINCTER
07009* 09111 09966
PYLOROPLASTY
00309* 00313* 00379* 01298* 01299* 01302* 01304* 01305*
01306* 01322* 01358 01359 02011* 02194* 02199* 02209*
02235 02236 02663* 03102* 03185* 03189* 03192* 03194*
03776 04040* 04085 04123 04177* 04260* 04261* 04262*
04263* 04264* 04266* 04274 04282 04292 04293 04297
04730* 04869 05112 05169 05217* 05221* 05236 05237
05240 05253 05256 05262 05756* 05763* 06239* 06246*
06254* 06263* 06264* 06270 06275 06279 06283 06285
06288 06300 06308 06310 06883* 07043 07139 07166
07177 07178 07680* 07685 08079 08165* 08169* 08174*
08192 08197 08201 08203 08210 08443* 08835 09139
09167 09248 09256 09268 09269 09309 09781
PYLORUS
08095 08214 08712

RADIATION THERAPY IN CANCER TREATMENT
00262* 01053 01128 01137* 01269 01380* 01427 01461
01488* 03079* 03259*
RADIATION,TOXIC EFFECTS OF
SEE ALSO TOXIC EFFECTS ON ABDOMEN
SEE ALSO TOXIC EFFECTS ON ABSORPTION
SEE ALSO TOXIC EFFECTS ON BILIARY TRACT
SEE ALSO TOXIC EFFECTS ON ESOPHAGUS
SEE ALSO TOXIC EFFECTS ON GASTROINTESTINAL TRACT
SEE ALSO TOXIC EFFECTS ON LARGE INTESTINE
SEE ALSO TOXIC EFFECTS ON LIVER
SEE ALSO TOXIC EFFECTS ON PANCREAS
SEE ALSO TOXIC EFFECTS ON RECTUM
SEE ALSO TOXIC EFFECTS ON SALIVARY GLANDS
SEE ALSO TOXIC EFFECTS ON SMALL INTESTINE
SEE ALSO TOXIC EFFECTS ON STOMACH
00016 00810 00906 01054 01181* 01287 01404* 01718
01737* 01833* 01935* 02134 02158 02159 02160 02161
02162 02163 02166 02167 02168 02171 02172 02173
02174 02175 02176 02177 02178 02179 02240 02297
02417 02430 02549 02718 02719 02720 02723 02724
02725 02726 02727 02744 02835 02836 02983* 02985*
03031 03032 03033 03034 03368* 03443 03674* 03734*
03844 04253 04519* 04544 04735 04736 04987* 05083*
05188* 05465 05552* 05561 05563 05568 05958 06010
06047 06144* 06219* 06336 07002 07080* 07419* 07436
07565 07736* 07814* 07819 07882 08256* 08302 08454
08559* 08577 08722* 08870 08968 09225 09932

RADIOIMMUNOASSAY
01698* 02993* 03000*
RADIOISOTOPE STUDY TECHNIQUES
SEE TRACER STUDIES
RADIOLOGY IN BLEEDING DIAGNOSIS
00952* 01089 02961 03149* 04177* 05052 05868* 05873*
05897 07968 08106 08131 08161 08873 09836 09904
RADIOLOGY IN CANCER DIAGNOSIS
00106* 00132* 00296* 00920* 00963 00969 00999 01053
01178* 01183* 02050* 02052* 02369 02373 02557 02927
02966 03075* 03099* 03105* 03269 03485* 03486* 03486*
03889* 04362 04890 04900 04933 04937 05839* 05874*
05906 06222 06842* 06910 06936 07066 07120 07258
07286 07847* 07880 07901 08015 08070 08305 08326
08382 09051 09056 09085 09101 09113 09364 09371
09377
RADIOLOGY IN CIRRHOSIS DIAGNOSIS
01887 01889 02889* 03601* 06600
RADIOLOGY IN GASTRITIS DIAGNOSIS
00339 03890* 03940 05912
RADIOLOGY IN JAUNDICE DIAGNOSIS
00996 02964 04842* 05880* 06962 09558
RADIOLOGY IN LARGE INTESTINE DIAGNOSIS
08164
RADIOLOGY IN PEPTIC ULCER DIAGNOSIS
00132* 00920* 00975 01013 02205* 03191* 04855* 05253
06151* 06251* 06253* 06294 06313 07020* 07125* 07160
07882 07910 08217 09254
RADIOLOGY IN PORTAL HYPERTENSION DIAGNOSIS
01117 01889 02906* 03379* 04621 05966 06421* 07860*
08448 09059
RADIOLOGY IN ULCERATIVE COLITIS DIAGNOSIS
00532* 01013 01464* 01472* 02382* 02383* 03888* 04370*
04375* 09423
RADIOLOGY,ABDOMEN
SEE ALSO ABDOMEN DIAGNOSIS
00141 00142 00155 00973 00976 01006 01054 01119
01929* 02889* 02915* 02917* 02933 02944 03105* 03465
04150 04222 04855* 04936 04937 04942 04945 04946
05026 05873* 05876* 05914 05920 05964 06895 06899
06910 06926 06964 07255 07918 07930 07940 07945
08460 08751 08752 08840 08864 08895 08897 09061
09091 09192 09236 09382 09661 09790
RADIOLOGY,ANUS
SEE ALSO ANUS DIAGNOSIS
03799 04788
RADIOLOGY,BILIARY TRACT
SEE ALSO BILIARY DISEASE DIAGNOSIS
00131* 00598* 00751* 00924* 00977 00980 01101 01249*
01503 01652* 01655* 01660* 01664* 01670 01671 01680
01681 01866* 01921 02182 02693 02919* 02938 02962
03634 03635 03656 03661 03926 03950 03979 04017
04019 04643 04657 04839* 04842* 04856* 04861* 04864*
04871 04884 04886 04890 04965 04966 05633* 05636*
05647* 05658 05659 05661 05662 05872* 05880* 05901
05909 05914 05917 05941 05955 06625* 06647 06652
06658 06674* 06853* 06857* 06878* 06879* 06892 06935
06937 06962 07402 07495* 07525 07660* 07841* 07878
07879 07884 07886 07898 07932 07938 07957 07974
07980 08171* 08434* 08662* 08667* 08682 09055 09062
09067 09068 09071 09072 09090 09233 09478 09730
09741 09767 09776
RADIOLOGY,DUODENUM
03950 04244 05930 06234 06308 06313 06843* 07158
08019 08121 08138 08146 08151 08171* 08420 09050
09054 09217 09223 09256 09273
RADIOLOGY,ESOPHAGUS
00147 00279 00280 00281 00473 00942* 00980 01034*
01035* 01145 01154 01155 01157 01160 01864* 01908
02007* 02010* 02011* 02013* 02040 02990* 03076* 03083
03086 03093 03911* 04130* 04131* 04133 04138 04146
04147 04148 04149 04154 04161 04164 04165 04263*
04846* 04916 04969 05082* 05093 05094 05101 05102
05111 05114 05120 05990* 06112 06119 06121 06130
06131 06133 06136 06140 06141 06253* 06920 06941
06942 06945 06960 06971* 06972* 06975* 06976* 06987
06988 06994 07001 07004 07046 07600 07858* 07875
07889 07982* 07985* 08004 08005 08007 08010 08013
08019 08482 09051 09132 09140 09147 09154 09155
09159 09160
RADIOLOGY,GALLBLADDER
00139 00754* 00778 00779 00781 00977 02671 04019
04625* 04856* 04888 05872* 05901 05909 05917 05922
05961 06718* 06879* 06933 06963 07494* 07824 07894
07932 07974 08171* 08681 08684 08686 08694 08697
08703 09034 09037 09055 09067 09776 09791
RADIOLOGY,GASTROINTESTINAL
SEE ALSO GASTROINTESTINAL DIAGNOSIS
00074* 00126* 00131* 00148 00154 00164 00167 00170
00191* 00242 00243 00244 00253 00512 00932* 00943*
00969 00980 00989 00998 01006 01027* 01050 01057
01100 01101 01252* 01797* 01800* 01867* 01909 01918
01920 01921 01985 02182 02918* 02961 02990* 03033
03126 03182* 03922* 03947 03973 04019 04068 04096
04101 04107 04115 04123 04222 04263* 04786 04844*
04855* 04871 04903 04933 04940 04942 04962 04968
RADIOLOGY,GASTROINTESTINAL
CONTINUED
04996 05020 05044 05045 05047 05079 05248 05844*
05847* 05873* 05921 05933 05935 05958 05967 06016
06021 06056 06070 06106 06186 06264* 06730 06886
06891 06909 06944 06961 06962 07068 07074 07113
07589 07590 07872 07901 07910 07923 07924 07956
07960 07981 08021 08073 08402 08434* 08782 08807
08825 08828 08855 08874 09063 09069 09070 09121
09774 09868 09982
RADIOLOGY,LARGE INTESTINE
00120* 00121* 00131* 00254 00420* 00442* 00450* 00454*
00455* 00456 00471 00472 00474 00501 00507 00507
00508 01053 01091 01124 01388* 01389* 01411 01431
01432 01442 01443 01453 01454 01801* 01805* 02299*
02300* 02315* 02341 02350 02382* 02383* 02387* 02742
02949 03156 03252* 03253* 03269 03278 03296* 03304
03888* 03931 03941 03997 03998 03999 04344 04350
04366 04368 04370* 04378 04770* 04775* 04867 04971*
05060 05278 05315* 05333 05342 05357 05367 05845*
05943 05975 05978 06171 06326* 06334 06347 06348
06349 06351 06378* 06887 06921 06944 07226 07895
07917 08065 08249* 08276 08284 08317 08330 08337
08348 08354 08363 08369 08370 09075 09088 09104
09356 09366 09385 09395 09402 09420 09426 09427
09996
RADIOLOGY,LIVER
SEE ALSO LIVER DIAGNOSIS
00131* 00137 00148 00150 00173 00175 00623 00627
00646 00667 00677 00746 00941* 00987 01117 01533*
01537* 01538* 01545* 01554 01561 01856* 01861* 01869*
01877* 01879* 01885 01887 01889 02500 02519 02530
02542 02605* 02651 02906* 02936 02937 02938 02939
02941 02953 02966 02967 03376* 03417 03457 03465
03486* 03526* 03595* 03596* 03601* 03885* 03936 03938
03939 03942 04010 04013 04076 04457* 04487 04551
04694* 04701 04846* 04848* 04900 04906 04934 04938
04942 04943 04953 04955 04956 05440* 05465 05472
05516 05556 05611* 05675 05843* 05844* 05874* 05891
05892 05893 05898 05907 05913 05918 05924 05927
05937 05944 05948 05959 05966 05969 05974 06482
06493 06600 06840* 06842* 06853* 06864* 06876* 06884
06885 06893 06894 06930 06938 06940 06959 06960
06965 07351* 07402 07844* 07860* 07877 07889 07892
07908 07922 07925 07926 07931 07941 07942 07943
07948 07949 07965 08454 08456 08468 08647 09026
09028 09032 09033 09038 09039 09041 09042 09053
09080 09087 09115 09489 09493 09503 09519 09523
09593 09596 09599 09660 09705 09720 09730 09733
RADIOLOGY,MESENTERY
00135 00219 01927* 02944 02997* 04967 07606 07903
07926 08130 08154 08738* 08850 09457 09517 09815
09822
RADIOLOGY,PANCREAS
00538* 00539* 00567 00568 00591 00836* 00967 00988
00994 01021* 01488* 01510* 01519 01889 01890 02415
02463 02898* 02917* 02926 02927 02934 02946 03323
03947 03952 03971 03975 03992 03995 04386* 04387*
04391* 04400 04415 04419 04858* 04893 04894 04917
04944 04962 04967 05396 05854* 05855* 05857* 05878*
05904 05911 05915 05949 05953 05976 05977 06409
06840* 06857* 06866* 06875* 06935 06963 07297 07322
07326 07583 07847* 07848* 07865* 07868* 07880 07884
07886 07907 08373* 08379 08381 08392 08414 09052
09057 09058 09073 09449 09470
RADIOLOGY,PERITONEUM
SEE PERITONEUM DIAGNOSIS
RADIOLOGY,RECTUM
01384* 01438 03255* 03257* 03259* 03265* 03799 03920*
03999 04788 05333 05344 05978 06888 07214* 08363
08370 09075 09420
RADIOLOGY,SALIVARY GLAND
01131 02910* 07967 09119
RADIOLOGY,SMALL INTESTINE
00115* 00131* 00165 00346* 00353* 00374 00420* 00925*
00966 00968 00988 00994 01013 01048* 01091 01124
01267 01279 01285 01288 01318* 01388* 01619* 01876*
01878* 01907 01985 02115* 02119* 02120* 02125* 02131
02132 02742 02902* 02911* 02960 02989* 02990* 03126
03154* 03791* 03891* 03935 04236 04243 04248 04303
04312 04315 04324 04666 04855* 04877 04878 04890
05060 05150* 05186* 05187* 05191 05192 05226* 05791*
05930 05990* 06013 06217* 06223 06230 06305 06625*
06857* 06890 06898 06929 06944 07089 07152 07181*
07182* 07903 07906 07933 07966 08101 08126 08149
08161 08222 08229* 08346 08943 09054 09071 09084
09104 09117 09177 09217 09223 09228 09245 09321
09429
RADIOLOGY,STOMACH
00292* 00317 00334 00337 00340 00417 00572 00920*
00959 00975 00999 01014 01034* 01035* 01048* 01167*
01168* 01174* 01176* 01178* 01182* 01185 01198 01199
01200 01209 01216 01217 01221 01228 01229 01232
01234 01298* 01319* 01320* 01361 01872* 01909 01915
01952 01985 02052* 02059 02078 02079 02080 02091
02108 02109 02110 02236 02885* 02886* 02905* 02969

RADIOLOGY,STOMACH
CONTINUED
03099* 03103* 03126 03135 03889* 03890* 03921* 03927
03935 03940 03945 03996 04004 04019 04176* 04181*
04186 04191 04197 04202 04203 04205 04211 04869
04882 04957 05134* 05138* 05163 05168 05173 05235
05839* 05900 05902 05912 05939 05940 05942 05945
05990* 06013 06016 06143* 06163 06169 06170 06171
06178 06185 06192 06195 06203 06207 06212 06843*
06883* 06912 06920 06936 06941 06963 07020* 07057
07071 07076 07128* 07180 07600 07845* 07846* 07850*
07857* 07875 07888 07896 07900 07912 07916 07929
07982* 08019 08048 08065 08066 08075 08081 08082
08084 08091 08093 08101 08171* 08389 08790 08931
09051 09056 09065 09085 09098 09109 09118 09155
09169 09184 09185 09203 09207 09215 09274 09284
09306 09307 09308
RADIOTELEMETRY
05879* 06828 07839*
RADIOTHERAPY
05188*
RADIOTHERAPY IN CANCER TREATMENT
01993 02008* 02035 02056* 02297 02309* 04065 04143
04146 04246 04333* 04866 05081* 05106 05107 05328
05329 06010 06047 06108* 06119 06128 06129 06135
06199 06336 06370 06398* 06936 06972* 06996 07565
08015 08070 08083 08302 08322 08325 08498 08722*
08876 09144 09159 09376 09831
RECTOCELE
01385* 01408*
RECTOCOLITIS,HEMORRHAGIC
08370 08375*
RECTOCOLONIC POLYPOSIS
00519 00519 04082 04334* 04340 04361 05335 07208*
07213* 07249 08259* 08271 08288 08315 08337 09199
09346 09352 09897
RECTUM
SEE ALSO HEMORRHOIDS
SEE ALSO POLYPS
SEE ALSO PROCTOSIGMOIDOSCOPY
SEE ALSO ULCERATIVE COLITIS
00502 01434 02949 03173 03252* 03284 03906* 04975*
06379* 06680* 07826 09400
RECTUM ABSORPTION
00030 02763*
RECTUM ANGIOGRAPHY
03257* 06888
RECTUM ANOMALIES
00511 00523 01900 06362 08331 09985
RECTUM BIOPSY
00422* 01398* 01865* 01880 01900 02390* 02732* 03255*
03257* 03265* 05688* 07944 08249* 08263* 08298 08338
08341 08364 08365 08751 09365
RECTUM CANCER
00447* 00466 00935* 01017* 01020* 01033* 01380* 01383*
01407* 01426 01427 01448 01936* 02304* 02306* 02309*
02310* 02342 02360 02367 02371 02373 02975* 02977*
03256* 03259* 03276 03293 04328* 04332* 04333* 04351
04352 04355 04361 05313* 05327 05329 05338 05339
05340 05356 05865* 06330* 06350 06358 06359 06361
06367 06370 06882* 06888 06949 07206* 07207* 07213*
07232 07257 07258 08220* 08260* 08265* 08274 08275
08288 08296 08302 08310 08320 08322 08325 08326
08345 08368 08722* 08862 09101 09364 09371 09376
09377 09392 09415 09577 09892 09982
RECTUM CIRCULATION
01704* 03257* 04353 06341 06888 08345 08920
RECTUM CYSTS
00514 00518
RECTUM DEVELOPMENT
06680*
RECTUM DIAGNOSIS
00422* 00470 00496 00935* 00957 01398* 01399* 01854*
01865* 01880 01900 02335 02390* 03255* 03256* 03257*
03265* 03906* 03920* 04364 04929 04931 05344 05356
05359 05978 06350 06353 06680* 06862* 06888 06889
06913 06949 07214* 07944 08259* 08295 08341 08352
08363 08364 08370 09035 09075 09088 09420
RECTUM DISEASE TREATMENT
00514 00518 01423
RECTUM DISEASES
00447* 00519 01399* 01409* 01417 02335 03255* 03257*
03265* 03906* 05059 05344 06333* 06350 06396 07228
08289 08321 08338 08349 08352 09365 09372
RECTUM DIVERTICULA
00459 02314*
RECTUM ENDOSCOPY
00935* 01399* 03265* 04364 04929 04931 06913 06949
07259 08363 08364 08365 08781 09035 09088
RECTUM FISTULAS
01382* 01408* 02380 02387* 05312* 05322 07212* 09341
09386
RECTUM HISTOLOGY
06371 07272 08298 08336 08365
RECTUM IMMUNOLOGY
01405* 01406* 01871*
RECTUM IN CHILDREN
00511 01900 04364 05350 05688* 06362 08932 09398
RECTUM MICROORGANISMS
01694* 08338
RECTUM MORPHOLOGY
02376 02732* 03285 03999 05355 05688* 08932 09386
RECTUM MOTILITY
00186* 02333 03799 04768* 04777* 04787 04788 05058
05059 05062 06373 07214* 09075
RECTUM MUCOSA
00443* 00468 01082 01404* 01405* 01406* 01473* 01478
01900 02361 02732* 03255* 03257* 04330* 05344 05688*
06336 06341 06350 06353 06371 06381* 06396 06862*
07272 07944 08226* 08259* 08365 08370 08932 09365
09386 09657
RECTUM NEOPLASMS
00440* 01380* 01409* 01439 04331* 05314* 06368
RECTUM NEOPLASMS,BENIGN
00443* 00470 04352 06330* 08273 08288 08336 08352
09199
RECTUM NEOPLASMS,MALIGNANT
01417 01438 02368 04352 05359 06330* 06350 07206*
08297 09392 09415 09982
RECTUM NEOPLASMS,VILLOUS
00440* 00443* 00444* 00446* 00457 00525 02337 02363
05319* 05351 06341 09199
RECTUM OBSTRUCTION
01438 03280 09985
RECTUM PATHOLOGY
00506 00514 00525 01384* 01409* 01423 03265* 05333
06366 06882* 08279 08298 08327 08343 08345 08349
08364 08370 09075 09365
RECTUM PERFORATION
01069 01383* 03278 09088 09398
RECTUM POLYPS
00224 00470 00506 00935* 01445 02305* 02343 04340
04361 04931 05319* 05359 06330* 06358 06361 08259*
08336 08352
RECTUM PROLAPSE
01390* 01425 08279 09342
RECTUM RADIOLOGY
01384* 01438 03255* 03257* 03259* 03265* 03799 03920*
03999 04788 05333 05344 05978 06888 07214* 08363
08370 09075 09420
RECTUM SECRETION
00468
RECTUM SURGERY
00440* 00443* 00444* 00446* 00447* 00466 01058 01385*
01398* 01409* 01419 01425 01438 01448 01456 02304*
02306* 02309* 02310* 02316* 02367 02380 03259* 03282
04332* 04355 04371* 04975* 05312* 05314* 05322 05327
05338 05339 05340 05348 05351 06327* 06328* 06331*
06338 06341 06353 06364 06367 06369 06370 06680*
06882* 07206* 07242 07243 07257 08273 08274 08275
08279 08296 08297 08310 08315 08327 08345 08351
08352 08362 08370 09199 09341 09350 09361 09372
09376 09386 09392 09401
RECTUM TRAUMA
01069 03278 05350
RECTUM TREATMENT
06353
RECTUM ULCERS
02335 08756
RECTUM ULTRASTRUCTURE
08298 08338
RECTUM,AGE EFFECTS ON
05058
RECTUM,DISEASES ASSOCIATED WITH
00506 01053 09341
RECTUM,NERVOUS CONTROL OF
08364
RECTUM,TOXIC EFFECTS ON
01404* 05058

RECURRENT ULCERS
01344 05220* 07142 08165* 09254
RECURRENT ULCERS,GASTRECTOMY-INDUCED
00132* 00312* 00400 00413 01315* 02221 03183* 03187*
03188* 03189* 03205 04204 04260* 04265* 04275 04283
04299 05233* 05255 05263 05840* 06248* 06293 06297
06310 06311 06841* 07036 07128* 07132* 07164 07177
07855* 08049 08058 08185 08730* 08769 08871 09253
09258 09284 09300 09309 02899*
REFLUX,ESOPHAGUS
00273 00843* 00887* 00940* 00950* 01139* 01158 01159
02006* 02013* 02015* 02887* 02990* 02994* 03069* 03070*
03074* 03078* 03088 03205 04134 04135 04136 04141
04142 04147 04165 04203 04969 05080* 05090 05092
05097 05101 05121 05123 06113 06114 06132 06189
06945 06971* 06978 06982 06984 06986 06988 07002
07006 07145 07156 07972 07983* 07986* 07987* 07990*
08009 08010 08013 08655* 09136 09138 09140 09148
09153 09155 09164 09291

REGENERATION,LIVER
00656 00662 00906 01104 01531* 01557 01834* 01838
02477* 02480* 02490 02506 02515 02517 02576* 02642
02704* 02706* 02714 02842* 02880 03378* 03382* 03382*
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03436 03437 03438 03439 03440 03441 03442 03443
03445 03446 03448 03449 03450 03451 03453 03454
03455 03456 03457 03504 03513 03620 03808* 03815*
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05568 05692* 05704 05816* 05831 05832 06535* 06549
06777* 06790* 06798* 06801 06805 06806 06817 07137*
07333* 07419* 07440 07454* 07455* 07703* 07705* 07706*
07708* 07722* 07723* 07724* 07730* 07731* 07733* 07736*
07738* 07752* 07755 07762 07766 07795 08423* 08433*
08445 08492 08502 08510 08536 08559* 08581 08593*
08616 08641 08654 08968 08973 08983 08985 09574
09615 09616 09618 09619 09645 09697 09698 09719
REGIONAL ENTERITIS
00186* 00201 00202 00354* 00420* 00421* 00422* 00423
00424 00442* 00521 00526 01115 01261* 01367* 01368
01369 01370 01403* 01436 01451 01470* 01474 01922*
01925* 01969 02275* 02277 02278 02281* 02290 02299*
02301* 02339 02341 02385* 02387* 03049 03225 03279
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04330* 04374* 04867 05277 05278 05279 05280 05281
05282 05283 05284* 05333 05342 05343 05367 06011
06042 06105 06314* 06315* 06316* 06317 06318 06319
06320 06321 06326* 06360 06366 06385 06844* 07085*
07181* 07182* 07183* 07184 07185 07186 07212* 07251
07252 07263* 07267 07269 07271 07895 07903 07933
08218* 08221 08222 08223 08224 08246 08327 08354
08370 08758 09083 09320 09322 09355 09356 09359
09360 09379 09835 09866 09982
REGIONAL ENTERITIS DIAGNOSIS
04307*
REGIONAL ILEITIS
01967 03891* 04302* 05277 05280 05283 06011 06316*
06319 06320 06321 07182* 07212* 08219* 08220* 08230*
08232* 09321
RESECTION-INDUCED SECONDARY MALABSORPTION,INTESTINAL
00434* 01403* 03246* 04062 04249 04730* 05206 05867*
06029 06844* 07183* 07193* 08077 08112* 08133 08230*
RESPIRATORY COMPLICATIONS
00169 00733 01161 03481* 08848 08914 09294 09447
09706
RETICULOENDOTHELIAL SYSTEM,LIVER
00017 00595* 03391 03924* 04076 04457* 05540* 05669*
06059 06557* 06692 06697 06838* 07335* 07433 07442*
07836* 09608 09635
RIBONUCLEASE SECRETION,PANCREAS
00804 00805 00836* 04684*
ROUX GASTROENTEROSTOMY
02053* 02105 02188
ROUX-EN-Y GASTROENTEROSTOMY
00294* 03181*
RUPTURE,DIAPHRAGM
04120 04125* 05096 06050 06999 08002 08759 09874
09895

SALIVA
00055 00290* 00398* 00793 00833* 00854 00878* 02220
02790* 03684 03742 04385* 07668* 07683 09464 09465
SALIVARY GLAND BIOCHEMISTRY
00055 00793 00794 00801 00802 01110 03683 03684
03783 04119 04385* 05381* 07666* 07672 07683 07936
SALIVARY GLAND CHEMICAL COMPOSITION
05381*
SALIVARY GLAND CIRCULATION
03759*
SALIVARY GLAND DEVELOPMENT
09931
SALIVARY GLAND DIAGNOSIS
00185* 00214 01131 02910* 07927 07967 09119
SALIVARY GLAND DISEASES
00185* 00214 01131 03344 04428 07927 07967 09119
09931
SALIVARY GLAND ELECTROLYTES
01772* 03759*
SALIVARY GLAND ENZYMES
01773* 03683 07652 07666* 07672 07936
SALIVARY GLAND HISTOLOGY
07665*
SALIVARY GLAND IMMUNOLOGY
03684 04385* 05016
SALIVARY GLAND METABOLISM
03783 07672
SALIVARY GLAND NEOPLASMS
00185* 04119 07927 07967 09932
SALIVARY GLAND NEOPLASMS,BENIGN
02910* 09831 09931
SALIVARY GLAND NEOPLASMS,MALIGNANT
09831 09931
SALIVARY GLAND PERFUSION
00050* 03759* 08372*
SALIVARY GLAND RADIOLOGY
01131 02910* 07967 09119
SALIVARY GLAND SECRETION
00050* 00055 00793 00797 00801 00854 01766* 01772*
01786 03750* 03759* 03774 03783 07665* 07666* 07668*
07671 08032 08372*
SALIVARY GLAND SURGERY
00185* 02910* 09831 09931
SALIVARY GLAND ULTRASTRUCTURE
01110 05381* 07665*
SALIVARY GLANDS
01705 04402 04403 04404 07967
SALIVARY GLANDS IN CHILDREN
04385*
SALIVARY GLANDS,DISEASES ASSOCIATED WITH
00185* 03344 04385*
SALIVARY GLANDS,DRUG EFFECTS ON
07672
SALIVARY GLANDS,HORMONAL CONTROL OF
00050* 01773*
SALIVARY GLANDS,NERVOUS CONTROL OF
01766* 01772* 03783 07671
SALIVARY GLANDS,TOXIC EFFECTS ON
00801 00802
SALMONELLOSIS
SEE ALSO PARATYPHOID FEVER
SEE ALSO TYPHOID FEVER
00209 00255 01945 01980 04035* 05019 05996* 06057
06952 07755 08849 09848 09863 09881 09882 09883
09946 09951
SALMONELLOSIS CARRIER STATE
04045* 05019 06856* 08775 08786 09953
SALMONELLOSIS DIAGNOSIS
04026* 05991* 06856* 06907 06955 07905 07951 08735*
09808 09971
SALMONELLOSIS EPIDEMIOLOGY
02991* 04026* 04045* 05019 05021 08735* 08749 08797
09814 09953 09975 09979
SALMONELLOSIS ETIOLOGY
00209 00255 05019 05021
SALMONELLOSIS IMMUNOLOGY
01052 04907 05991*
SALMONELLOSIS IN CHILDREN
00938* 01052 03990 04026* 04035* 04045* 05019 05991*
07544* 08726* 08736* 08749 08775 08786 09230 09814
09953 09994
SALMONELLOSIS PATHOLOGY
09864 09953
SALMONELLOSIS PREVENTION
05021
SALMONELLOSIS TREATMENT
00255 00938* 03990 06829 06832 07560 08735* 08745*
08765 08775 08786 08803 09953
SALMONELLOSIS,BIOCHEMICAL DIAGNOSIS OF
06870*
SALMONELLOSIS,CLINICAL STUDIES OF
00255 01466*
SALMONELLOSIS,EXPERIMENTAL STUDIES OF
02384* 08744*
SALT ABSORPTION,BILE
02122* 02129* 03150* 03227* 03633* 05717*
SARCOMA
01253* 01504 02986* 05173 05923 06027 06030 06194
06627* 07067 07119 07441 07916 08080 08109 08155
08428* 08440* 08460 08476 08719* 08941 09222
SCANNING,SCINTILLATION
00137 00148 00150 00677 00746 00941* 00967 00969
00987 00988 01010 01561 01885 01890 01916 02605*
02891* 02898* 02903* 02906* 02910* 02915* 02922 02936
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04944 04945 04946 04957 05837* 05839* 05843* 05855*
05857* 05878* 05882* 05891 05892 05893 05907 05913
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05965 05974 05976 05977 06452* 06709 06842* 06864*
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07583 07848* 07857* 07877 07890 07892 07907 07908
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07943 07947 07948 07949 07950 07965 08077 08384
08433* 08468 08571 08581 09032 09033 09036 09039
09052 09058 09080 09109 09112 09120 09125 09489
09544
SCHISTOSOMIASIS
00190* 00204 00236 00241 00580 00675 00680 01573
02529 02646 03003 03396 05495 07405 09002 09236
09512 09652 09878 09884 09972
SCHISTOSOMIASIS DIAGNOSIS
00125* 00205 00957 01880 02489 03957 04904 06487
06877* 07876 08496 08578 08754 08755 08833 09657
SCHISTOSOMIASIS EPIDEMIOLOGY
00183* 00192* 00194* 01018* 02489 04089 05022 06256*
06487 07586 08754 08755 08868 08880 09952 09955

SCHISTOSOMIASIS IMMUNOLOGY
00125* 00190* 00238 00239 03957 07586 08578 09829
09923 09952 09998
SCHISTOSOMIASIS PATHOLOGY
00236 04076 04457* 05496 08578 08755 09541 09564
09657
SCHISTOSOMIASIS PROPHYLAXIS
00190* 00194* 04089 04489 08866 08868 08880 09830
SCHISTOSOMIASIS TREATMENT
00122* 00124* 00183* 00241 02489 03928 03956 03988
04489 04925 06073 06083 06331* 06487 06877* 07564
07570 08754 08833 08866 09564
SCHISTOSOMIASIS,BIOCHEMICAL DIAGNOSIS OF
05612*
SCHISTOSOMIASIS,CLINICAL STUDIES OF
00192* 00205
SCHISTOSOMIASIS,DISEASES ASSOCIATED WITH
09708
SCHISTOSOMIASIS,EXPERIMENTAL STUDIES OF
00680
SCINTILLATION SCANNING
00137 00148 00150 00677 00746 00941* 00967 00969
00987 00988 01010 01561 01885 01890 01916 02605*
02891* 02898* 02903* 02906* 02910* 02915* 02922 02936
02937 02938 02939 02941 02953 02966 02967 03372*
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04944 04945 04946 04957 05837* 05839* 05843* 05855*
05857* 05878* 05882* 05891 05892 05893 05907 05913
05915 05918 05921 05924 05927 05937 05944 05949
05965 05974 05976 05977 06452* 06709 06842* 06864*
06875* 06876* 06930 06938 06940 06959 06961 06964
07583 07848* 07857* 07877 07890 07892 07907 07908
07919 07923 07924 07925 07927 07931 07941 07942
07943 07947 07948 07949 07950 07965 08077 08384
08433* 08468 08571 08581 09032 09033 09036 09039
09052 09058 09080 09109 09112 09120 09125 09489
09544
SCLERODERMA
05990* 06110* 06204 06971* 06998
SEASONAL FACTORS
09915
SECRETIN
SEE ALSO PANCREAS
00026* 00044* 00052 00119* 00540* 00836* 00849* 00869
01483* 01484* 01492* 01506 01715 01750* 01751* 01752*
01764* 01768* 01780 01783 01933* 02416 02437 02699*
02784* 02786* 02787* 02791* 02803* 02804 02810* 02827*
02903* 03194* 03334* 03752* 03753* 03765* 03768* 03777
03994 04049* 04380* 04389* 04677 04741* 04750* 04753
04762 04875 04961 05203 05213* 05740* 05747* 05752*
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06866* 06880* 07575 07690* 07691* 07692* 07695 07838*
07853* 07954 08200 08375*
SECRETION DISORDERS,STOMACH
00040* 00041* 00118* 00188* 00189* 00290* 00291* 00294*
00312* 00320 00383* 00845* 00869 00935* 00970 00974
01034* 01166* 01361 01485* 01872* 02125* 03000* 03109*
03113* 03723* 03749* 03768* 03887* 03945 03974 03978
04031* 04049* 04193 04213 04258* 04989* 05031 05053
05098 05131* 05266 05418 05435* 05626 05764* 05852*
05867* 05879* 05884* 05928 06000* 06014 06018 06145*
06150* 06166 06208 06210 06240* 06252* 06598 06742*
06748* 06766 06858* 06934 06956 06958 07014* 07019*
07027* 07039 07040 07041 07068 07134* 07152 07154
07160 07176 07361 07362 07581 07582 07583 07584
07839* 07846* 07856* 08044* 08168* 08176* 08458 08768
08769 08779 08780 08798 08824 08826 09097 09210
09298 09313 09396 09809 09824 09865
SECRETION IN DISEASE,STOMACH
00042* 01090 01933* 02215* 02820 02905* 02947 02948
02980* 02988* 03000* 03036 03193* 03237* 03761* 05141*
. 05143* 07446* 09194
SECRETION STUDY TECHNIQUES
05747* 05752* 05767 06618* 06734* 06880* 06914 06924
07694 07854*
SECRETION STUDY TECHNIQUES,BILE
04006 04007 07660*
SECRETION STUDY TECHNIQUES,PANCREAS
03993 06772* 06773*
SECRETION STUDY TECHNIQUES,STOMACH
00040* 00049* 00049* 00060 00133 00291* 00392* 00397*
00413 00807 00838* 00847* 00848* 00858 00865 00934*
00974 00993 00995 01001 01090 01194 01198 01199
01200 01308* 01313* 01754* 01755* 01756* 01757* 01770*
01774* 01775* 01781 01794* 01859* 01872* 01912 02094
02235 02236 02697* 02790* 02800* 02801* 02820 02899*
02905* 02909* 03065 03180* 03705* 03748* 03749* 03751*
03755* 03756* 03757* 03758* 03760* 03767* 03773 03887*
03945 03978 04224 04291 04740* 04746* 04759 04881
04914 05217* 05746* 05756* 05759* 05840* 05879* 05884*
05928 06252* 06667* 06750* 06755 06858* 06902 06922
06934 06950 06956 06958 07469* 07674* 07675* 07677*
07678* 07679* 07687 07839* 07845* 07857* 07896 08165*
08168* 09107 09192
SECRETION,BILE
00023* 00076* 00101 00756* 00797 00839* 00851* 00867
00875 00913 01353 01637* 01688 01760* 01841 01851
02704* 02782* 02815 02858 02874 03360* 03369* 03491
03522* 03639 03641 03642 03757* 03765* 03774 03784
03811* 03881 04006 04007 04539 04670* 04795* 04805*
05072 05648* 05652 05658 05765* 05767 05793* 05806*
05814* 05938 05956 06449* 06483 06508 06529 06620*
06623* 06626* 06634 06734* 06770* 06782* 06789* 06803
06914 06932 07193* 07276* 07356 07367 07398 07514
07523 07745* 07747* 07747* 07762 07969 08387 08426*
08430* 08431* 08441* 08688 08819 08988 09511 09540
09766
SECRETION,DRUG EFFECTS ON BILE
07823
SECRETION,DRUG EFFECTS ON PANCREAS
00863 02782*
SECRETION,DRUG EFFECTS ON STOMACH
00040* 00041* 00043* 00046* 00328 00383* 00384* 00397*
00819* 00831* 00832* 00842* 00846* 00847* 00853 00867
00871 00872 00888 00953* 00986 01172* 01310* 01313*
01314* 01702* 01703* 01748* 01749* 01767* 01768* 01770*
01775* 01778* 01782 01791 02090 02216* 02788* 02789*
02792* 02796* 02799* 02808 02821 03000* 03122 03180*
03190* 03193* 03695* 03747* 03762* 03769* 03778 03796*
04141 04256* 04259* 04756 04764 05140* 05146* 05215*
05218* 05239 05268 05748* 05755* 05763* 06210 06235*
06252* 06258* 06259* 06287 06744* 06752* 06754* 06769*
07675* 07676* 07678* 07682* 07685 07687 08059 08178*
08200 09246 09307
SECRETION,DUODENUM
00048* 00849* 01259* 01266 01366 01492* 01762* 01807*
01912 02215* 02408* 03707* 04001 04411* 05777 06214*
07448* 07614 07976
SECRETION,GALLBLADDER
00086* 00776 00839* 03641 05652 05656 05660 05956
06671* 07489* 07807 08677
SECRETION,GASTROINTESTINAL
00402 00403 00797 02071 02816 02823 03017 03062
03195 03746* 04115 04677 05020 05778 06828 07656*
SECRETION,HORMONAL CONTROL OF STOMACH
00046* 00049* 00054 00057 00831* 00832* 00833* 00840*
00841* 00846* 00847* 00848* 00850* 00852* 00859 00862
00870 00871 00873 00993 01310* 01314* 01358 01359
01748* 01749* 01750* 01754* 01755* 01758* 01774* 01775*
01776* 01777* 01782 01789 01790 01791 01810* 01899
02235 02703* 02779* 02780* 02785* 02788* 02792* 02794*
02796* 02800* 02801* 02806 02814 03000* 03693 03755*
03756* 03758* 03760* 03764* 03767* 03768* 03770* 03772
03773 03776 03784 04037* 04267* 04740* 04741* 04745*
04746* 04748* 04749* 04752* 04755 04756 04758 04763
05017 05018 05213* 05229* 05686* 05695* 05696* 05736*
05741* 05742* 05745* 05746* 05753* 05754* 05757* 05758*
05759* 05760* 05763* 05766 05769 05770 05771 05772
05773 05775 05776 06000* 06014 06213 06247* 06255*
06739* 06741* 06742* 06746* 06747* 06749* 06750* 06752*
06756 06757 06758 06760 06761 06767 06768 06769
06922 06956 07127* 07152 07154 07169 07665* 07675*
07676* 07679* 07682* 07683 07684 08176* 08190 08720*
08769 08948 08949 09315
SECRETION,KIDNEY ELECTROLYTE
00090* 03774 04445* 08055 08227*
SECRETION,LARGE INTESTINE
02003 02325* 04343 07448* 07636* 07637* 07638*
SECRETION,LIVER
00101 00839* 01996 02071 03369* 03641 04794* 05537*
06792* 07615 07726* 07729* 07807 07969 08527 08563
08636* 09508 09540
SECRETION,NERVOUS CONTROL OF STOMACH
00043* 00054 00059 00299* 00307* 00324 00831* 00841*
00842* 00843* 00848* 00852* 00856 00857 00859 00862
00867 00868 00871 00872 00873 00874 01358 01359
01362 01749* 01759* 01778* 01794* 02212* 02235 02779*
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05229* 05250 05258 05679 05748* 05753* 05772 06252*
06258* 06259* 06289 06292 06295 06667* 06738* 06741*
07039 07154 07275* 07604 07675* 07684 08165* 08168*
08172* 08188 08203
SECRETION,PANCREAS
00023* 00026* 00052 00188* 00193* 00193* 00540* 00546
00590 00797 00836* 00849* 00855 00860 00863 00864
00866 00867 00869 00875 00877 01092 01494 01514*
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01771* 01779* 01933* 01935* 01939* 01996 02071 02125*
02279* 02400 02408* 02421 02422 02423 02431 02432
02678 02707 02791* 02803* 02810* 02811 02827* 02970
03004 03323 03331* 03350 03672* 03752* 03753* 03757*
03765* 03766* 03783 03784 03898* 03966 03968 03971
03993 03994 04097 04389* 04399 04407 04420 04669*
04673* 04675* 04687* 04750* 04755 04760 04762 04851*
04878 04961 05004 05183* 05292 05388 05419 05522
05660 05738* 05739* 05740* 05752* 05761* 05765* 05767
05774 05895 05896 05915 06058 06324 06405 06406

SECRETION,PANCREAS
CONTINUED
06407 06420* 06440 06508 06528 06734* 06767 06770*
06771* 06775 06776 06866* 07274* 07276* 07280* 07281
07282 07288 07290 07291 07296 07304* 07306* 07307*
07321 07323 07325 07326 07416 07446* 07665* 07691*
07692* 07693* 07694 07696 07954 07958 07970 08375*
08377 08378 08391 08393 08780 08819 09210 09446
09468 09898
SECRETION,PANCREAS AMYLASE
00579* 00836* 00860 00930* 01489* 01498 01751* 01753*
01762* 01771* 02407* 02409* 02443* 02729* 02782* 02784*
02786* 02805 02970 03330* 03334* 03343* 03711* 03953
03983 04380* 04389* 04424 04427 04921 05391 05765*
05971 06435 06642 06871* 07278* 07293 07311 07690*
07692* 07693* 07695 07853* 07936 07954 07958 07970
08415 09096 09210
SECRETION,PANCREAS BICARBONATE
00930* 01483* 01492* 01751* 01752* 01753* 01759* 01764*
01780 02409* 02797* 02803* 02804 03752* 03753* 04380*
04389* 04750* 04875* 05203 05747* 06423* 06772* 06880*
07690* 07692* 07853* 08375*
SECRETION,PANCREAS CALCIUM
00547 00589 01521 06407 06429*
SECRETION,PANCREAS CARBOXYPEPTIDASE
00836* 00860 03689 03690 03691 03692 04389*
SECRETION,PANCREAS CHLORIDE
02797* 05203
SECRETION,PANCREAS CHYMOTRYPSIN
00058 00119* 00804 00805 00836* 00860 01765* 01771*
02804 04389* 04412* 07838* 07937
SECRETION,PANCREAS ELASTASE
00836* 03338* 03339* 03340* 04412*
SECRETION,PANCREAS LIPASE
00027* 00836* 00860 00930* 01371* 01489* 01759* 02407*
02729* 02784* 02804 03699* 03983 04080 04268* 04389*
04921 05382* 05779* 05850* 05932 06423* 06435 06773*
07278* 07651 07954 08239 08415 09210
SECRETION,PANCREAS PHOSPHOLIPASE
01517 07308*
SECRETION,PANCREAS PROTEASE
00193* 00860 02729* 03785* 04960 05382*
SECRETION,PANCREAS PROTEIN
01752* 02782* 02803* 03752* 04684* 04744*
SECRETION,PANCREAS RIBONUCLEASE
00804 00805 00836* 04684*
SECRETION,PANCREAS TRYPSIN
00058 00119* 00804 00805 00836* 00860 00930* 01762*
01765* 01771* 01935* 02407* 02442* 02448 02454 02787*
02804 03330* 03343* 04389* 04412* 04684* 04851* 06429*
06924 07692* 07693* 07954 08400* 09210
SECRETION,PEPTIC ULCER
00299* 00383* 03768* 05764* 07274*
SECRETION,RECTUM
00468
SECRETION,SALIVARY GLAND
00050* 00055 00793 00797 00801 00854 01766* 01772*
01786 03750* 03759* 03774 03783 07665* 07666* 07668*
07671 08032 08372*
SECRETION,SMALL INTESTINE
00841* 01697* 02003 02157 02817 02826* 03756* 03782
03784 04671* 04712* 04716* 04785 05185* 05203 05616
05707 05724* 05777 05796* 05938 06214* 06417* 06687
06873* 07085* 07631* 07976 09021 09243 09292 09898
SECRETION,STOMACH
00039 00053 00056 00057 00109* 00196* 00223 00307*
00328 00339 00393* 00398* 00797 00833* 00850* 00854
00861 01092 01104 01181* 01194 01209 01299* 01702*
01719* 01727* 01747* 01768* 01769* 01770* 01780 01790
01791 01989 02054* 02071 02087 02093 02392* 02703*
02754* 02761* 02806 02822 02823 02892* 03063 03145
03746* 03762* 03775 03801 03981 04097 04213 04436*
04602* 04674* 04754 04757 04764 05167 05169 05274
05455* 05684* 05740* 05743* 05750* 05751* 05761* 05762*
05768 05839* 06154* 06177 06200 06275 06307 06309
06690 06755 06759 07030* 07031* 07062 07135* 07594*
07681* 07682* 07686 07688 07689 07835 07983* 08006
08033* 08950 08951 08952 09077 09176 09180 09184
09292 09302 09303 09304 09319 09573 09737 09921
SECRETION,STOMACH ACID
00042* 00045* 00051* 00056 00134 00188* 00297* 00299*
00301* 00309* 00313* 00390* 00392* 00403 00831* 00832*
00835* 00840* 00843* 00844* 00845* 00848* 00850* 00853
00856 00857 00858 00859 00862 00867 00868 00870
00871 00872 00873 00874 00876 00888 00934* 00935*
00936* 00953* 00974 00993 01001 01014 01034* 01051
01090 01166* 01172* 01174* 01177* 01198 01199 01259*
01296* 01301* 01305* 01308* 01310* 01313* 01314* 01315*
01316* 01317* 01700* 01702* 01716 01748* 01749* 01750*
01754* 01755* 01756* 01757* 01758* 01761* 01762* 01764*
01767* 01775* 01776* 01777* 01778* 01782 01785 01787
01788 01792 01810* 01859* 01872* 01924* 02011* 02046*
02197* 02204* 02212* 02235 02236 02246 02408* 02631*
02705* 02723 02779* 02780* 02785* 02788* 02789* 02790*
02792* 02793* 02794* 02795* 02796* 02798* 02799* 02800*
02801* 02809* 02813 02820 02899* 02905* 02909* 02992*
SECRETION,STOMACH ACID
CONTINUED
03004 03063 03064 03065 03113* 03119 03122 03136
03140 03180* 03181* 03190* 03193* 03197 03198 03209
03220 03693 03705* 03732* 03747* 03748* 03749* 03754*
03755* 03756* 03757* 03758* 03760* 03761* 03763* 03764*
03765* 03767* 03768* 03770* 03773 03776 03796* 03887*
03978 04039* 04141 04173* 04216 04224 04256* 04258*
04259* 04267* 04286 04288 04290 04291 04718* 04742*
04743* 04745* 04746* 04747* 04748* 04749* 04751* 04752*
04755 04756 04758 04761 04763 04765 04881 04914
05098 05133* 05140* 05141* 05155 05156 05213* 05215*
05218* 05221* 05228* 05229* 05236 05239 05243 05252
05268 05273 05418 05435* 05626 05736* 05741* 05742*
05744* 05745* 05746* 05749* 05753* 05754* 05755* 05756*
05758* 05759* 05760* 05762* 05763* 05764* 05766 05769
05771 05772 05773 05775 05776 05840* 05852* 05879*
05884* 05928 06014 06145* 06151* 06160* 06166 06210
06213 06235* 06236* 06240* 06245* 06252* 06255* 06258*
06259* 06260* 06287 06292 06295 06298 06598 06667*
06728 06738* 06741* 06742* 06743* 06746* 06747* 06748*
06749* 06750* 06751* 06752* 06754* 06756 06757 06758
06760 06761 06762 06763 06764 06765 06767 06769
06841* 06858* 06904 06922 06934 06950 06971* 07016*
07027* 07028* 07038 07063 07132* 07151 07157 07164
07171 07172 07173 07174 07175 07176 07177 07191*
07275* 07396 07446* 07469* 07613 07674* 07675* 07676*
07678* 07679* 07680* 07681* 07682* 07683 07684 07685
07687 07688 07689 07695 07839* 07845* 07857* 08059
08072 08166* 08168* 08175* 08176* 08180 08190 08200
08204 08208 08211 08458 08730* 08779 08780 08790
08835 08871 08948 08949 08953 09045 09076 09078
09097 09107 09114 09192 09205 09294 09300 09307
09308 09313 09396
SECRETION,STOMACH ELECTROLYTE
00301* 00305* 00819* 00859 01492* 01754* 01755* 01756*
01757* 01777* 01787 02235 02817 03732* 03747* 03763*
03770* 03772 03796* 04745* 04747* 04763 06744* 06747*
07665* 07678* 07688 08044* 08168* 09316
SECRETION,STOMACH MUCUS
00047* 00398* 00858 00859 00862 01794* 02783* 03694*
03695* 03705* 03757* 03769* 03771 03775 03779 04216
06741* 06750* 06934 07037 07125* 07688
SECRETION,STOMACH PEPSIN
00041* 00045* 00064* 00275 00297* 00299* 00834* 00837*
00838* 00845* 00846* 00850* 00858 00859 00862 00872
00880 00955 01090 01166* 01296* 01301* 01749* 01754*
01755* 01757* 01758* 01761* 01762* 01767* 01777* 01781
01784 01794* 02697* 02785* 02788* 02789* 02795* 02796*
02801* 02806 02807 02992* 03136 03705* 03760* 03761*
03775 03776 04216 04740* 04756 05742* 05743* 05749*
05754* 05755* 05762* 05764* 05766 05773 06002* 06155*
06166 06258* 06738* 06739* 06740* 06742* 06746* 06748*
06749* 06753* 06754* 06763 06764 06766 06767 06769
06841* 06858* 06902 06934 07063 07446* 07674* 07682*
07684 07688 07854* 08059 08180 08200 09078 09294
SECRETOR STATUS,BLOOD GROUP
00389* 04270* 06748* 08177* 09017
SEROLOGICAL DIAGNOSIS
01012 03925* 03961 04072 04084 04090 04479 05046
05905 05991* 06554* 07066 07068 07119 07754 07905
07920 07921 07939 08206 08208 08366 08402 08456
08500 08580 08610 08875 08884 08897 09086 09120
09192 09423 09517 09637 09667 09687 09688 09705
09938 09941 09943 09944 09998
SEROTONIN
SEE ALSO CARCINOID SYNDROME
00002* 00089* 00210 00310* 00882* 01062 01074 01300*
01713 01932* 02173 02198* 02207* 02522 02779* 03622
03782 03880 04069 04407 04525* 04833 05017 05023
05243 05706 05748* 05802 05803 06213 06670* 06678*
07060 07659* 08498 08925 08927 08929 09021 09446
SERUM BILE PIGMENTS IN JAUNDICE
00596* 00756* 01618* 01677 03380* 03381* 03827 04501
04873 05494 07402 07809 08511
SERUM CHOLESTEROL
07083* 07810*
SERUM ELECTROLYTES
00002* 00046* 00184* 00299* 00303* 00385* 00441* 00444*
00578 00756* 01395* 02337 02571 02753* 02978* 03029
03041 03042 03075* 03367* 03934 04456* 05289* 05604*
05720* 06001* 06017 06020 07139 07574 07807 08055
08072 08425* 08537 09737
SERUM ENZYMES,LIVER
SEE ALSO ALKALINE PHOSPHATASE
SEE ALSO LIVER DISEASE DIAGNOSIS
00166 00638 00715 00931* 00937* 00947* 01002 01003
01539* 01543* 01552* 01603 01615* 01616* 01631 01639*
01668 01852* 01863* 01869* 02470* 02576* 02597* 02598*
02602* 02604* 02606 02609 02622 02706* 02916* 02958
02959 03001* 03356* 03365* 03377* 03482* 03484* 03491
03499 03507 03523* 03531 03532 03536 03542 03549
03609 03615 03685 03806* 03835 03962 03967 03984
04020 04441* 04446* 04496 04523* 04541 04585 04813
04843* 04845* 04872 04879 04885 04915 04920 04932
05201 05442* 05466 05494 05536* 05546* 05553* 05560

SERUM ENZYMES,LIVER
CONTINUED
05572* 05576* 05592* 05599* 05617 05637* 05859* 05866*
05889 05919 05926 05934 05959 06286 06460* 06513
06515 06532* 06545 06553* 06554* 06561 06568 06572
06577 06651 06777* 06789* 06852* 06853* 06854* 06855*
06865* 06867* 06869* 06923 06928 07351* 07369 07418*
07422* 07440 07444* 07449* 07459 07626* 07752* 07763
07806 07840 07842* 07864* 07867* 07870 07914 07962
08366 08424* 08425* 08441* 08474 08537 08541* 08549*
08561* 08562* 08594* 08595* 08598* 08601* 08612 08626
08986 09033 09086 09430 09625 09628 09641 09663
09673 09693
SERUM HEPATITIS
02486* 03383* 03522* 03529* 04562* 04569* 04575 04585
04590 04905 05572* 05588 06545 06556* 06563 06574
07447* 08592* 08608 08628
SERUM IRON
02534 03104* 04384* 04980* 05368 05444* 06606 08525
09725
SEX FACTORS
00399 03106* 03131 03313* 03523* 04175* 04624* 05131*
05139* 05422* 06142* 06241* 06244* 06245* 06248* 06330*
06426* 06556* 06558* 06627* 07026* 07086* 07136* 07343*
07956 08428* 08550* 08556* 08602* 08649 08719* 08899
09280 09973 09976
SHIGELLOSIS
SEE ALSO DYSENTERY
00452* 00938* 01971 01988 03882* 04035* 06053 06830
06953 06954 07210* 07571 09879 09944 09945 09946
09994
SHIGELLOSIS DIAGNOSIS
06915 06916 08894
SHIGELLOSIS EPIDEMIOLOGY
02991* 05019 07546* 08800 08838 08839 08894 08897
09978 09999
SHIGELLOSIS IMMUNOLOGY
07546* 08838 08839
SHIGELLOSIS IN CHILDREN
07544* 08726*
SHIGELLOSIS TREATMENT
08788 08801 08802 08838 08839 08894
SHISTOSOMIASIS TREATMENT
06450*
SHOCK
00184* 00578 00654 00892* 01079 02476* 02708 03367*
03384 04028* 04108 04117 04252 04421 04422 04446*
04498 04659 05077 05180* 05183* 05526* 05545* 05671*
05829 05995* 06482 06611 07122 07123 07873 08136
08451 08547* 08743* 08776 08823 08845 08867 09812
09911 09912 09925
SHUNT IN PORTAL HYPERTENSION TREATMENT,PORTACAVAL
00264* 00653 00672 00674 01032* 01089 01642*
SHUNT,PORTACAVAL
01528* 01609 01751* 01845 01926* 02032 02033 02054*
02483* 02484* 02517 02638* 02645 02646 03119 03355*
03408 03413 03423 03592* 03593* 03594* 03599* 04010
04064 04454* 04468 04490 04495 04598* 04599* 04603*
04606* 04612 04613 04617 04623 04846* 05100 05424*
05451* 05455* 05513 05514 05518 05592* 05593* 05602*
05603* 05605* 05617 05627 06450* 06503 06504 06579*
06583* 06586 06587 06597 06599 06601 06605 06607
06973* 07331* 07332* 07333* 07396 07403 07405 07407
07423* 07468* 07470* 07472 07481 07483 07487 07762
07858* 07922 07984* 08437* 08457 08458 08472 08473
08482 08488 08496 08632* 08635* 08647 08952 08988
09060 09236 09497 09507 09514 09523 09538 09539
09541 09547 09553 09561 09592 09594 09702 09703
09705
SHUNT,SPLENOCAVAL
05455*
SHUNT,SPLENOPORTAL
07730*
SHUNT,SPLENORENAL
06594 06973* 07331* 07340* 07341* 07468* 07486 08471
08472 08482 08496 09703
SIALOGRAPHY
01766* 09119
SIALOLITHIASIS
07967
SICKLE CELL ANEMIA
06631*
SIGMOID
SEE ALSO LARGE INTESTINE
00164 00186* 00245 00444* 00457 00459 00478 00479
00480 00481 00483 00485 00486 00487 00488 00491
00492 00493 00494 00495 00496 00528 00530 00620
01027* 01091 01396* 01402* 01413 01420 01431 01434
01437 01442 01798* 01801* 01802* 01865* 02299* 02300*
02309* 02351 02388* 03252* 03257* 03258* 03264* 03268
03274 03276 04332* 04335* 04339 04348 04353 04364
04996 05311* 05313* 05340 05978 06318 06367 06379*
06680* 07211* 07227 07233 07238 07250 08319 08357
09350 09367 09370 09383 09385 09389 09403 09405
09409 09416 09417
SIGMOIDITIS
00477 00478 00479 00480 00481 00482 00483 00484
00485 00486 00487 00488 00489 00490 00491 00492
00493 00494 00495 01053 04360 06010 06336 06346
SIGMOIDOSCOPY
01399* 01400* 03253* 03257* 03920* 04860* 04897 06352
07258 07259 07917 08144 08259* 08303 08333 08337
08582 08724* 08756 08757 08807 08855 08860 08863
09139 09371 09405 09950
SIMPLE NONSPECIFIC ULCERS
SEE NONSPECIFIC ULCERS
SJOGRENS SYNDROME
00540* 09446
SMALL INTESTINE
SEE ALSO ABSORPTION
SEE ALSO DUODENUM
SEE ALSO ENTERITIS
SEE ALSO ILEOCECAL VALVE
SEE ALSO ILEUM
SEE ALSO ILEUS
SEE ALSO INTUSSUSCEPTION
SEE ALSO JEJUNUM
SEE ALSO MALABSORPTION
SEE ALSO PEPTIC ULCERS
00355 00886* 03061 03062 03066 03158 03160 03178
04230* 05182* 05719* 07084* 07826 08153 08918 09893
SMALL INTESTINE ABSORPTION
00011* 00022* 00023* 00028* 00035 00063* 00347* 00785*
00812* 00813* 00814* 00815* 00816* 00817* 00818* 00820
00823 00825 00826 00829 00830 00895* 00925* 00926*
01260* 01372* 01403* 01721* 01722* 01723* 01724* 01729*
01731* 01732* 01733* 01736* 01739 01744 01870* 02169
02172 02247 02279* 02284* 02285* 02289 02290 02291
02293 02296 02324* 02352 02483* 02603* 02702* 02734*
02748* 02750* 02751* 02753* 02756* 02757* 02758* 02759*
02760* 02762* 02765 02767 02769 02777 02829* 02988*
03002* 03150* 03166 03229* 03230* 03232* 03234* 03242*
03246* 03500 03633* 03639 03674* 03688 03720* 03725*
03726* 03727* 03728* 03731* 03733* 03735 03736 03737
03739 03744 03745 03980 04024* 04044* 04060 04189
04236 04240 04249 04305* 04306* 04308* 04312 04313
04320 04323 04442* 04672* 04676* 04710* 04711* 04712*
04713* 04715* 04716* 04717* 04718* 04720* 04721* 04723*
04729* 04730* 04732 04734 04736 04739 04782 04783
04850* 04959 04977* 04981* 05127* 05184* 05185* 05196
05198 05202 05287* 05288* 05300 05307 05361* 05666*
05676 05687* 05709* 05710* 05712* 05714* 05715* 05718*
05719* 05721* 05722* 05723* 05724* 05725* 05726 05727
05729 05730 05733 05864* 05910 06214* 06302 06686
06691 06699* 06700* 06701* 06703* 06705* 06706* 06707*
06710 06715 06717 06723* 06836* 06844* 06881* 07031*
07079* 07123 07162 07188* 07189* 07190* 07191* 07193*
07194* 07446* 07492* 07575 07620 07621 07623* 07624*
07625* 07626* 07627* 07628* 07629* 07630* 07634* 07635*
07639* 07641* 07642* 07647 07648 07655 07915 08077
08112* 08116* 08134 08159 08160 08230* 08232* 08234*
08236 08934 08935 08936 08937 08938 08943 08944
09018 09043 09074 09084 09218 09242 09322 09324
09325 09326 09327 09331 09332 09334 09336 09429
09916 09926
SMALL INTESTINE ANGIOGRAPHY
00420* 01013 03154* 06889 06890 06929 07903 07906
08382
SMALL INTESTINE ANOMALIES
00360 01215 01267 01271 01295 01946 01991 02114*
02156 02186 03301* 03319 04228* 04232* 05009 05187*
05210 06221 06232 07084* 07109 07110 07609 08026
08092 08124 08139 08901 08916 09219 09224 09233
09238 09245 09862
SMALL INTESTINE ATRESIA
07084* 08835
SMALL INTESTINE BIOCHEMISTRY
00011* 00024* 00063* 00179 00432* 00892* 00922* 00923*
01255* 01371* 01373* 01374* 01718 01731* 01793* 02157
02246 02391* 02728 02730* 02731* 02734* 02736* 02765
02812 02824 02826* 02827* 02902* 02913* 03002* 03110*
03228* 03233* 03239* 03243* 03245* 03500 03673* 03677
03681 03682 03685 03686 03687 03703* 03703* 03718
03727* 03735 03737 03930 03966 03968 04227* 04229*
04253 04305* 04306* 04313 04676* 04685* 04686* 04688*
04695* 04704 04709 04713* 04714* 04737 04868 05160
05181* 05295 05386* 05666* 05681* 05687* 05714* 05715*
05727 05853* 06260* 06691 06700* 06700* 06708* 06735*
06827* 07083* 07085* 07203 07593* 07594* 07595* 07597*
07620 07621 07626* 07629* 07634* 07667* 07810* 07811*
07816* 07829 07915 07976 08225* 08226* 08233* 08237
08263* 08918 08926 09020 09022 09292 09322 09332
09333 09429 09898
SMALL INTESTINE BIOPSY
00371 00436 00811 00922* 00923* 00925* 00982 01250*
01266 01289 02248 02988* 02989* 03152* 03228* 03232*
03233* 03891* 03986 04002 04231* 04234 04246 04271*
04311 04313 04314 04315 04686* 04868 05182* 05303
05851* 05886 06220* 07187* 07189* 07192* 07202 07611
07861* 07881 08063 08144 08229* 08233* 08234* 08243
08246 08263* 08918 09084 09110 09225 09234 09726

ALL INTESTINE BIOPSY
ONTINUED
09889 09920
ALL INTESTINE CANCER
00259 01126 01253* 01281 01282 01501 01943 02118*
02131 02155 02191 02290 02977* 03225 04053 04185
04244 04246 04699 04890 05017 05212 05894 06222
06223 06227 06234 07081* 07086* 07557 08147 08149
08155 08220* 08220* 08392 08743* 09832 09843 09875
09889 09892
ALL INTESTINE CHEMICAL COMPOSITION
02734* 04733
ALL INTESTINE CIRCULATION
00007* 00008* 00359 00830 01248* 01270 01375 01518
01878* 02121* 02137 02179 02192 02698* 02702* 02776
02888* 03148* 03167 03670* 03671* 03680 03710* 03908*
04240 04245 04252 04695* 04782 04783 04849* 05029
05190 05196 05201 06890 06897 06898 06977 07024*
07084* 07088 07089 07090 07204 07557 07592* 07648
08118* 08717* 09036 09232 09236
ALL INTESTINE CONTENTS
02246 02825* 03679
ALL INTESTINE CYSTS
08776
ALL INTESTINE DEVELOPMENT
01271 01708* 02717 02914* 05178* 05723* 07109 07813*
08119*
ALL INTESTINE DIAGNOSIS
00131* 00254 00357 00371 00420* 00811 00921* 00922*
00923* 00925* 00966 00972 00982 01013 01091 01250*
01266 01288 01289 01503 01619* 01876* 01883 02114*
02115* 02119* 02120* 02131 02142 02181 02911* 02914*
02989* 03126 03149* 03154* 03227* 03228* 03232* 03239*
03891* 03935 03986 04001 04002 04231* 04232* 04234
04244 04246 04271* 04311 04313 04315 04855* 04868
04877 04890 04897 04959 05060 05182* 05187* 05192
05197 05204 05226* 05279 05280 05303 05851* 05853*
05886 05887 05894 05930 05963 06223 06225 06226
06230 06320 06323 06625* 06857* 06889 06890 06897
06898 06929 07089 07090 07097 07104 07119 07181*
07187* 07192* 07861* 07871 07881 07903 07906 07915
07933 07952 07959 07966 08063 08101 08113* 08126
08148 08149 08218* 08222 08229* 08232* 08235 08346
08784 08943 09050 09071 09084 09104 09110 09111
09117 09123 09217 09223 09224 09228 09238 09245
09321 09889 09936 09986
ALL INTESTINE DISACCHARIDASES
00011* 00065 01255* 02281* 02391* 02392* 02393* 02731*
03238* 03245* 04092 04227* 04229* 04236 04253 04312
04325 04688* 04709 04739 04880 05181* 05293 05295
05386* 06669* 06699* 06707* 06851* 07202 07203 07621
08231* 08237
ALL INTESTINE DISEASE EPIDEMIOLOGY
06226 09937 09940
ALL INTESTINE DISEASE ETIOLOGY
08218*
ALL INTESTINE DISEASES
00350* 00357 00366 00375 01065 01087 01290 01518
01954 02119* 02122* 02352 03153* 03162 03243* 04230*
04877 05197 05199 05205 05207 05303 06028 06221
07089 07094 07096 07116 07744* 07915 08113* 08129
08150 08160 08162 08236 09071 09084 09117 09232
09239 09335 09355 09726 09819 09875 09880 09909
09919 09933
ALL INTESTINE DISEASES,BIOCHEMICAL DIAGNOSIS OF
04307*
ALL INTESTINE DIVERTICULA
00367 00368 00966 01034* 01274 01285 01286 01288
02115* 02143 02190 02289 03149* 05039 05159 05178*
05191 05192 06230 07100 07104 07110 07695 08121
08124 08131 08161 08176* 09223 09242
ALL INTESTINE DIVERTICULITIS
02349 03167 09231
ALL INTESTINE ENDOSCOPY
00921* 02119* 04897 05887 07861* 07871
ALL INTESTINE ENZYMES
00011* 00179 00432* 01371* 01373* 01374* 01697* 01731*
01765* 01793* 02003 02051* 02157 02391* 02456 02731*
02812 02824 02826* 02827* 02902* 02913* 03002* 03110*
03228* 03233* 03239* 03243* 03245* 03673* 03677 03681
03682 03685 03687 04227* 04229* 04253 04305* 04306*
04313 04686* 04704 04714* 05160 05181* 05295 05666*
05687* 06669* 06708* 06827* 07448* 07595* 07620 07621
07652 07816* 07829 07833 07915 07976 08129 08225*
08237 08263* 08918 08926 09100 09243 09322 09898

ALL INTESTINE EXTRACTS
07594*
ALL INTESTINE FISTULAS
00199* 01038* 01273 01275 01276 01278 01652* 01655*
02149 03008 03030 03066 04032* 04741* 05027 05211
07124 07183* 07565 07626* 08132 08143 08221 08222
09237 09898
SMALL INTESTINE HISTOLOGY
02167 02168 02178 02193 02713 02988* 02989* 03152*
03239* 03891* 04003 04226* 04239 04686* 04695* 04781*
05182* 05184* 05188* 05202 05292 05386* 05673* 05980*
06216* 06325 06670* 06691 06694 06836* 07079* 07086*
07090 07122 07123 07189* 07196 07593* 07595* 07620
07621 07653 07810* 07812* 07813* 07814* 07819 08116*
08119* 08142 08150 08155 08160 08235 08263* 08268*
08917 08918 09018 09020 09225 09227 09234 09330
09855
SMALL INTESTINE IMMUNOLOGY
00198* 00925* 01376 02911* 02979* 03019 03737 03930
06215* 06417* 06707* 07186 07263* 07620 07621 07832
08268*
SMALL INTESTINE IN CHILDREN
01267 01295 01373* 01923* 02114* 02122* 02141 03157
03176 03179 03318 04228* 04234 04243 05009 05178*
05187* 05190 05193 05851* 06081 06232 06669* 07091
07097 07105 07106 07109 07112 07113 07744* 08112*
08115* 08144 08149 08234* 08776 08835 08848 08901
09111 09238 09240 09819 09986
SMALL INTESTINE INJURIES
02159 02160 02174 02176 02177 02178 03154* 03171
03674* 04849* 04987* 05029 05198 05202 07080* 07095
07106 07107 07565 08126 08136 08822 09226
SMALL INTESTINE LYMPHOGRAPHY
05963 06898
SMALL INTESTINE METABOLISM
00024* 00063* 00826 02169 03673* 03688 03745 04061
04229* 04712* 05676 05853* 06228 06691 07626* 07629*
07813* 07816* 07825 08939 09022
SMALL INTESTINE MICROORGANISMS
00196* 00348* 00354* 00369 00972 01695* 01696* 01735*
02138 02317* 02664* 03236* 03991 04307* 04913 04977*
04987* 04990* 05006 05184* 05887 06053 06434 06477
07825 08233* 08898 08904 09024 09025 09819 09880
09926 09937 09938 09939 09940 09941 09942 09943
09944 09945 09946 09983
SMALL INTESTINE MORPHOLOGY
00012* 00016 00710 00821 01714 01718 02126* 02129*
02137 02731* 03670* 03714* 03717 03718 03737 04024*
04229* 04739 05178* 05182* 05226* 05719* 06216* 06221
06302 06687 06897 06898 07112 07299* 07592* 07609
07614 07812* 07832 08129 08142 08917 08926 08938
09324 09330 09862
SMALL INTESTINE MOTILITY
00007* 00008* 00075* 00077* 00079* 00186* 00353* 00785*
00787* 00881* 00885* 00889 00921* 01796* 01797* 01800*
01803* 01808* 01809* 01813 01814 01820 01821 01867*
02125* 02138 02290 02829* 02911* 03194* 03782 03791*
03792* 03795* 03798 03804 03891* 03898* 04086 04203
04249 04772* 04780* 04781* 04782 04783 04785 04786
04790 04849* 04878 04986* 05196 05198 05205 05207
05210 05781* 05786* 05787* 05789* 05791* 05793* 05794*
05795* 05799 05800 05801 06217* 06302 06687 06722*
06723* 06727 06731 06733 06872* 07060 07078* 07106
07122 07123 07162 07191* 07299* 07657* 07658* 07660*
07691* 07959 08118* 08129 08906 08943 08946 09054
09229 09245
SMALL INTESTINE MUCOSA
00115* 00198* 00310* 00347* 00817* 00826 00922* 00923*
01027* 01048* 01080 01082 01177* 01255* 01260* 01264
01273 01287 01289 01373* 01374* 01376 01619* 01696*
01705 01708* 01721* 01722* 01723* 01724* 01741 01745
01760* 01910 01923* 01935* 01937* 02116* 02121* 02126*
02134 02139 02165 02166 02175 02274 02279* 02281*
02282* 02287 02290 02324* 02352 02391* 02392* 02393*
02702* 02716 02728 02731* 02733* 02765 02777 02807
02824 02973* 02983* 03002* 03148* 03151* 03152* 03153*
03228* 03231* 03235* 03238* 03239* 03248 03670* 03671*
03673* 03674* 03680 03682 03686 03687 03703* 03710*
03714* 03718 03728* 03729* 03735 03737 03745 03777
03782 03871 03891* 03930 03986 03989 04003 04024*
04051* 04092 04226* 04234 04236 04238 04239 04252
04253 04310* 04311 04312 04313 04315 04319 04323
04326 04671* 04686* 04703 04704 04709 04716* 04733
04736 04737 04782 04783 04790 04868 04987* 05160
05179* 05182* 05183* 05184* 05185* 05188* 05196 05198
05199 05202 05203 05207 05219* 05226* 05228* 05285*
05294 05295 05297 05300 05303 05361* 05386* 05670*
05691* 05696* 05697* 05709* 05712* 05714* 05726 05778
05853* 05886 05980* 06013 06147* 06215* 06216* 06220*
06313 06323 06325 06669* 06686 06691 06692 06694
06700* 06707* 06717 06834 06836* 06881* 07024* 07079*
07080* 07082* 07085* 07112 07116 07122 07123 07162
07182* 07183* 07189* 07192* 07196 07200 07592* 07593*
07594* 07597* 07609 07611 07620 07621 07625* 07626*
07627* 07630* 07634* 07653 07673 07812* 07813* 07819
07856* 07871 07966 08063 08116* 08119* 08134 08135
08136 08142 08160 08223 08225* 08226* 08231* 08232*
08234* 08286 08901 08917 08918 08934 08935 08936
08938 09018 09020 09021 09025 09037 09084 09110
09225 09231 09234 09240 09244 09322 09323 09330
09332 09333 09920

SMALL INTESTINE NEOPLASMS
00355 00373 00374 01265 01503 02119* 02120* 03163
03175 05186* 06026 06223 06224 06889 08137 08148
08243 08762 09084 09893
SMALL INTESTINE NEOPLASMS,BENIGN
00165 00346* 00361 00370 00418 01051 01083 01262
01263 01270 01272 01282 01370 02118* 02123* 02139
02154 03149* 03165 04250 04251 05060 05194 05209
05212 06233 06929 07086* 08101 08122 08130 08131
08151 08161 08743* 09823 09905
SMALL INTESTINE NEOPLASMS,MALIGNANT
00346* 01029* 01083 01253* 01268 01269 01279 02118*
02132 02145 02184 02986* 03149* 03168 03225 04065
04246 04251 05012 05189 05212 05963 06027 06225
06227 06229 06234 07067 07081* 07086* 07092 07101
07119 07186 08125 08149 08155 08678 08743* 09110
09222 09875 09889
SMALL INTESTINE NEOPLASMS,VILLOUS
04042*
SMALL INTESTINE OBSTRUCTION
00245 00352* 00372 01091 01124 01125 01171* 01246*
01247* 01249* 01254* 01263 01277 01278 01279 01290
01291 01294 01876* 01878* 01951 02114* 02139 02140
02143 02187 02196 02346 02981* 03168 03171 03172
03176 03236* 03318 03336* 04043* 04054 04107 04124
04232* 04235 04243 04247 04254 04322 04661 04666
04667 04772* 04877 04986* 04993 04996 05009 05055
05137* 05150* 05186* 05187* 05189 05193 05195 05197
05200 05205 05209 05219* 06036 06063 06090 06223
06226 06228 06232 06415 06625* 07087* 07091 07093
07097 07098 07099 07102 07103 07105 07106 07111
07113 07119 07121 07181* 07183* 07505 07537 07565
07579 08114* 08115* 08118* 08123 08124 08126 08161
08162 08195 08221 08223 08346 08382 08784 08916
09104 09220 09221 09229 09231 09241 09244 09245
09460 09746 09759 09761 09786 09841 09847 09866
09890 09895 09986
SMALL INTESTINE PATHOLOGY
00198* 00223 00350* 00358 00359 00361 00375 00421*
00892* 01264 01280 01286 01339 01373* 01745 01935*
02003 02324* 02734* 02736* 02973* 02980* 02988* 03152*
03168 03969 04024* 04061 04226* 04250 04307* 05204
05219* 05280 05285* 05685* 06053 06062 06063 06153*
06234 06317 06319 06321 07090 07101 07116 07181*
07187* 07189* 07200 07212* 07545* 07557 07933 08113*
08114* 08122 08124 08125 08129 08135 08148 08149
08150 08155 08158 08159 08160 08161 08218* 08223
08224 08232* 08263* 08346 08672 08728* 08901 08904
09071 09117 09182 09227 09323 09324 09819 09875
09889 09890 09905 09907 09909
SMALL INTESTINE PERFORATION
00371 00376* 00421* 00424 01069 01251* 01269 01273
01290 01368 01951 02115* 02136 02141 02996* 03151*
03164 03171 03179 03271 03986 04027* 04239 04303
05039 06304 07095 07096 07107 07117 07118 07397
08128 08157 08210 08212 08221 08222 08901 08906
09092 09232 09565 09876
SMALL INTESTINE PERFUSION
00007* 00008* 00818* 02127* 02698* 02748* 02777 02999*
03714* 04712* 04720* 05180* 05748* 06699* 06701* 06706*
07624* 07625* 07627* 07634* 07641* 07811* 08234* 08938
SMALL INTESTINE POLYPS
00364 01256* 01274 01280 02133 04233* 05194 07903
08123 08144 08743* 08762 09361 09823 09897
SMALL INTESTINE RADIOLOGY
00115* 00131* 00165 00346* 00353* 00374 00420* 00925*
00966 00968 00988 00994 01013 01048* 01091 01124
01267 01279 01285 01288 01318* 01388* 01619* 01876*
01878* 01907 01985 02115* 02119* 02120* 02125* 02131
02132 02742 02902* 02911* 02960 02989* 02990* 03126
03154* 03791* 03891* 03935 04236 04243 04248 04303
04312 04315 04324 04666 04855* 04877 04878 04890
05060 05150* 05186* 05187* 05191 05192 05226* 05791*
05930 05990* 06013 06217* 06223 06230 06305 06625*
06857* 06890 06898 06929 06944 07089 07152 07181*
07182* 07903 07906 07933 07966 08101 08126 08149
08161 08222 08229* 08346 08943 09054 09071 09084
09104 09117 09177 09217 09223 09228 09245 09321
09429
SMALL INTESTINE SECRETION
00841* 01697* 02003 02157 02817 02826* 03756* 03782
03784 04671* 04712* 04716* 04785 05185* 05203 05616
05707 05724* 05777 05796* 05938 06214* 06417* 06687
06873* 07085* 07631* 07976 09021 09243 09292 09898
SMALL INTESTINE STRICTURE
09232
SMALL INTESTINE SURGERY
00201 00202 00207 00351* 00352* 00358 00360 00365
00366 00368 00424 00434* 00524 00563 00573 00780
00787* 01038* 01058 01091 01134 01248* 01249* 01254*
01258* 01268 01271 01276 01277 01278 01281 01284
01293 01294 01307* 01369 01526 01652* 01975 02115*
02119* 02123* 02127* 02133 02141 02144 02146 02157
02277 02290 02399 02665* 02780* 02802* 02860 02893*
02914* 02974* 02996* 03036 03066 03091 03149* 03150*
03162 03166 03171 03172 03174 03176 03178 03185*
SMALL INTESTINE SURGERY
CONTINUED
03186* 03194* 03205 03227* 03230* 03271 03328 03333*
03339* 03633* 03756* 03768* 03800 03955 04039* 04051*
04054 04095 04096 04124 04232* 04240 04242 04243
04247 04249 04296 04513 04666 04849* 04928 04975*
04977* 05014 05039 05137* 05186* 05187* 05189 05190
05193 05195 05197 05199 05200 05204 05206 05208
05209 05210 05211 05212 05214* 05279 05280 05283
05389 06045 06152* 06219* 06227 06231 06319 06415
06477 06635 06836* 06977 07042 07077* 07078* 07081*
07083* 07087* 07088 07091 07098 07099 07102 07103
07104 07107 07111 07113 07114 07119 07124 07129*
07181* 07183* 07184 07193* 07508 07520 07565 07657*
08003 08030 08056 08074 08077 08101 08112* 08114*
08115* 08117* 08118* 08122 08125 08126 08127 08132
08133 08141 08143 08144 08150 08157 08159 08163
08182 08201 08212 08213 08221 08230* 08346 08362
08403 08469 08692 08717* 08743* 08776 08784 08835
08901 08916 09084 09151 09177 09182 09232 09237
09241 09247 09261 09434 09746 09755 09761 09847
09868 09875 09893 09912 09930
SMALL INTESTINE TRANSPLANTATION
00787* 01151 01273 01276 01705 01990 02116* 02127*
04203 05051 05084* 06123 06131 06977 06997 07024*
07078* 07277* 08003 08815 08867 09151 09164 09350
SMALL INTESTINE TRAUMA
00365 01038* 01069 01251* 02996* 03171 04238 04245
04254 04284 05211 07095 07107 07108 07113 07117
08026 08114* 09226 09235
SMALL INTESTINE TREATMENT
02277 03164 05204 05207 06090 06224 06225 06320
07097 08143 08158 08158 08222 08235 08743* 09239
09880
SMALL INTESTINE ULCERS
06251* 08223 08730* 08779
SMALL INTESTINE ULCERS,NONSPECIFIC
01080 01283 01311* 02257 02914* 03151* 03162 03172
03173 04245 05219* 07114 07141 08131 09255
SMALL INTESTINE ULTRASTRUCTURE
02193 03707* 03718 03989 04226* 04229* 04234 04710*
04868 05184* 05196 05285* 05687* 05691* 05707 06215*
06686 06687 06691 06692 06881* 07079* 07080* 07082*
07187* 07196 07597* 07602 07609 07611 07612 07629*
07819 07832 08119* 08134 08135 08139 08223 08233*
08243 08246 08926 08934 09330 09920
SMALL INTESTINE VOLVULUS
00326 01125 03058 04322 06226 07098 08346 08776
08835 09219
SMALL INTESTINE,AGE EFFECTS ON
05196 05709* 05722* 08926
SMALL INTESTINE,CLINICAL STUDIES OF
00357 00359 00360 02169
SMALL INTESTINE,DISEASES ASSOCIATED WITH
00347* 00437 03061 03062 04029* 08159 09890
SMALL INTESTINE,DRUG EFFECTS ON
04978* 05710* 05777 07639* 08118* 08157 08158 09021
09022
SMALL INTESTINE,FOREIGN BODIES IN
08128
SMALL INTESTINE,HORMONAL CONTROL OF
00812* 01722* 02734* 04785 04941 05070 05071 05072
05203 05307 05707 05777 06722* 06723* 06727 07060
07655
SMALL INTESTINE,NERVOUS CONTROL OF
00885* 00889 01813 01814 02894* 02895* 03148* 03792*
03804 04781* 04790 05294 05787* 05794* 06243* 06670*
06722* 06723* 07545* 07602 07612 09229 09913
SMALL INTESTINE,TOXIC EFFECTS ON
00016 00022* 00372 01079 01257* 01287 01718 01734*
01935* 01937* 02134 02165 02166 02167 02168 02173
02174 02175 02179 02317* 02747 02983* 03674* 03717
04245 04253 04323 04714* 04735 04781* 05180* 05188*
05202 05715* 06010 06215* 06703* 06727 07080* 07082*
07085* 07187* 07189* 07639* 07812* 07814* 07819 08134
08135 08559* 09225
SMOOTH MUSCLE
SEE ALSO MOTILITY
01821 05128* 05179* 07415 07602 07612
SMOOTH MUSCLE MORPHOLOGY
01818
SMOOTH MUSCLE PHARMACOLOGY
00007* 00008* 00075* 00881* 00882* 01804* 01809* 01814
01815 01819 02138 02207* 02832 03797* 04116 04707
05790* 05792* 06721* 06727 07264* 07663 07818 08946
09019 09873 09926
SMOOTH MUSCLE PHYSIOLOGY
00001* 00007* 00008* 00070* 00071* 00074* 00079* 00881*
00884* 00885* 01381* 01429 01814 01815 01818 01819
02102 02334 03793* 03797* 04116 04117 04760 04780*
04781* 04790 04849* 05095 05706 05778 05781* 05784*
05785* 05790* 05799 05800 06037 06039 06058 06687
06689 06696 06725* 06731 07660* 07663 07818 07986*
08767 08858 08917 08928 09019 09418

SODIUM ABSORPTION
00038 00095 01412 01824* 02170 02738 02753* 02758*
02764 02770 03002* 03674* 03730* 04713* 05682* 07622*
07624* 07633* 07642* 08255* 08939 08944 09737 09989
SODIUM EXCRETION
00090* 06034 06569
SPASMOLYTIC AGENTS
08837
SPASMS,DRUG TREATMENT OF ESOPHAGUS
00269 04141
SPASMS,ESOPHAGUS
00269 02026 04145 05073 05085* 05086* 05095 06106
06115* 08023
SPASMS,GASTROINTESTINAL
04668
SPHINCTER OF ODDI
SEE ALSO BILIARY TRACT
00076* 00351* 00766 00839* 01022* 01651* 01658* 01684
01796* 02668* 02919* 03177 04085 04086 04419 04484
04658 04778* 05409* 05566 05636* 05644* 05647* 05658
05782* 05792* 05793* 05798 06423* 06441 06620* 06624*
06630* 06661 06662 06689 06720* 06914 07327 07498*
07501* 07533 07660* 07878 07879 07977 08051 08659*
08685 08686 08696 08698 08710 09431 09773 09775
09779 09782
SPHINCTER,ANUS
01377* 01381* 01386* 01799* 01804* 01854* 02333 03799
04767* 04768* 04788 04789 05312* 05316* 05325 05326
05330 06328* 06373 06689 06882* 07243 07863* 08310
08340 08341 09108 09122 09363 09373 09387
SPHINCTER,ESOPHAGUS
00070* 00884* 00887* 00990 01138* 01152 01155 01158
02006* 02994* 03070* 03078* 04136 04147 04165 04771*
04774* 04784 05080* 05097 05101 05115 06052 06110*
06136 06185 06724* 06835 06920 06971* 06974* 06982
07001 07600 07982* 07983* 07986* 08010 08021 09164
SPHINCTER,PYLORIC
07009* 09111 09966
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00151 00734 01515* 02452 02643 03404 03820* 03936
04451* 04502 04887 05458 05567 05671* 05701* 05848*
06421* 06455* 06503 06578* 06596 06602 06615 06616
06777* 07285 07318 07341* 07353 07412 07653 07877
07897 07949 08450 08472 08480 08530 08577 08870
09014 09039 09053 09168 09460 09539 09541 09547
09564 09567 09663 09704 09708 09711 09981
SPLENOCAVAL SHUNT
05455*
SPLENOMEGALY
00170 03605 05597* 07464* 08457 08642 08645 08728*
08755 08995 09236 09454 09602 09657 09661 09681
09726
SPLENOPORTAL SHUNT
07730*
SPLENOPORTOGRAPHY
00151 00268 00565 00567 00768 01515* 06959 06960
07858* 08471 08473
SPLENORENAL SHUNT
06594 06973* 07331* 07340* 07341* 07468* 07486 08471
08472 08482 08496 09703
SPRUE
00186* 01251* 01451 02274 02281* 02290 02293 03242*
04315 04324 05070 05284* 05306 06323 06836* 06851*
07189* 07197 07625* 07838* 08158 08246 08247 08248
09320 09334
SPRUE,TROPICAL
00438 01177* 01374* 02991* 03229* 03242* 05290* 06323
07192* 07194* 07198 07576 08229* 09325
STAINING TECHNIQUES
01709* 05708 07010* 07073 07607 07613 07614 07616
07617 07653 07869 07885 08139 08146 09143 09462
09501 09662
STAPHYLOCOCCAL ENTEROCOLITIS
07548* 07549* 08785
STARVATION
02730* 02731* 05811* 06400* 06795* 07288 07758 08119*
STEATORRHEA
00223 00354* 00429* 00431* 00434* 00773 00971 01113
01260* 01372* 01376 01923* 02279* 02280* 02282* 02283*
02285* 02286* 02287 02289 02290 02291 02292 02603*
02988* 03150* 03226* 03227* 03232* 03234* 03236* 03246*
03248 03725* 03912* 04242 04307* 04309* 04310* 04311
04315 04316 04326 04382* 04730* 04941 05004 05030
05031 05179* 05184* 05206 05284* 05285* 05293 05294
05303 05306 05309 05361* 05392 05841* 05910 05985*
06322 06863* 07079* 07183* 07188* 07191* 07192* 07193*
07194* 07195 07200 07290 08113* 08116* 08226* 08228*
08232* 08243 08399* 08443* 08938 09082 09126 09210
09239 09323 09327
STEATOSIS,ALCOHOLIC
00606* 03362* 03502 03503 03836 04517* 05448* 05925
06490 06520 06521 06522 06523 07401 07749* 07808
08540* 08589 08989 09621 09626 09631
STEROID ABSORPTION
02757*
STEROID METABOLISM,LIVER
02875 03398 04619 04797* 04823 05475 05512 05594*
05689* 05830 08475
STEROIDS
SEE ALSO CORTICOSTEROIDS
SEE ALSO LIVER CHOLESTEROL METABOLISM
00404 00589 00621 00644 00913 00916 01464* 02014*
02287 02511 02521 02737 02870 02992* 03097* 03113*
03337* 03350 03556 04036* 04257* 04372* 04517* 05512
06035 07144 07321 07471* 07794 08011 08172* 08594*
08641 08967 09246
STOMACH
SEE ALSO BEZOARS
SEE ALSO GASTRITIS
SEE ALSO PYLORIC OBSTRUCTION
SEE ALSO ULCERS
00081 01122 01195 01303* 01341 01349 01351 01867*
02102 02208* 02283* 02785* 02815 02819 03062 03124
03144 03147 03218 03220 03222 04169* 04172* 04173*
05135* 05684* 06667* 07826 07832
STOMACH ABSORPTION
00813* 01727* 01729* 01732* 01787 02770 03733* 03736
03739 04602* 04718* 05735 06159* 08054 08077 08945
STOMACH ACID SECRETION
00042* 00045* 00051* 00056 00134 00188* 00297* 00299*
00301* 00309* 00313* 00390* 00392* 00403 00831* 00832*
00835* 00840* 00843* 00844* 00845* 00848* 00850* 00853
00856 00857 00858 00859 00862 00867 00868 00870
00871 00872 00873 00874 00876 00888 00934* 00935*
00936* 00953* 00974 00993 01001 01014 01034* 01051
01090 01166* 01172* 01174* 01177* 01198 01199 01259*
01296* 01301* 01305* 01308* 01310* 01313* 01314* 01315*
01316* 01317* 01700* 01702* 01716 01748* 01749* 01750*
01754* 01755* 01756* 01757* 01758* 01761* 01762* 01764*
01767* 01775* 01776* 01777* 01778* 01782 01785 01787
01788 01792 01810* 01859* 01872* 01924* 02011* 02046*
02197* 02204* 02212* 02235 02236 02246 02408* 02631*
02705* 02723 02779* 02780* 02785* 02788* 02789* 02790*
02792* 02793* 02794* 02795* 02796* 02798* 02799* 02800*
02801* 02809* 02813 02820 02899* 02905* 02909* 02992*
03004 03063 03064 03065 03113* 03119 03122 03136
03140 03180* 03181* 03190* 03193* 03197 03198 03209
03220 03693 03705* 03732* 03747* 03748* 03749* 03754*
03755* 03756* 03757* 03758* 03760* 03761* 03763* 03764*
03765* 03767* 03768* 03770* 03773 03776 03796* 03887*
03978 04039* 04141 04173* 04216 04224 04256* 04258*
04259* 04267* 04286 04288 04290 04291 04718* 04742*
04743* 04745* 04746* 04747* 04748* 04749* 04751* 04752*
04755 04756 04758 04761 04763 04765 04881 04914
05098 05133* 05140* 05141* 05155 05156 05213* 05215*
05218* 05221* 05228* 05229* 05236 05239 05243 05252
05268 05273 05418 05435* 05626 05736* 05741* 05742*
05744* 05745* 05746* 05749* 05753* 05754* 05755* 05756*
05758* 05759* 05760* 05762* 05763* 05764* 05766 05769
05771 05772 05773 05775 05776 05840* 05852* 05879*
05884* 05928 06014 06145* 06151* 06160* 06166 06210
06213 06235* 06236* 06240* 06245* 06252* 06255* 06258*
06259* 06260* 06287 06292 06295 06298 06598 06667*
06728 06738* 06741* 06742* 06743* 06746* 06747* 06748*
06749* 06750* 06751* 06752* 06754* 06756 06757 06758
06760 06761 06762 06763 06764 06765 06767 06769
06841* 06858* 06904 06922 06934 06950 06971* 07016*
07027* 07028* 07038 07063 07132* 07151 07157 07164
07171 07172 07173 07174 07175 07176 07177 07191*
07275* 07396 07446* 07469* 07613 07674* 07675* 07676*
07678* 07679* 07680* 07681* 07682* 07683 07684 07685
07687 07688 07689 07695 07839* 07845* 07857* 08059
08072 08166* 08168* 08175* 08176* 08180 08190 08200
08204 08208 08211 08458 08730* 08779 08780 08790
08835 08871 08948 08949 08953 09045 09076 09078
09097 09107 09114 09192 09205 09294 09300 09307
09308 09313 09396
STOMACH ANGIOGRAPHY
02113
STOMACH ANOMALIES
04200 04205 04207 04211 04212 04217 05099 05138*
05939 06187 07012* 07057 07058 07059 08031 08087
08092 09169
STOMACH BIOCHEMISTRY
00001* 00013* 00040* 00041* 00047* 00053 00056 00061
00064* 00133 00134 00289* 00298* 00378* 00398* 00789*
00838* 00853 00880 01179* 01181* 01235 01309* 01700*
01701* 01761* 01769* 01781 01794* 02064 02741 02783*
02947 02980* 03110* 03694* 03708* 03715 03732* 03769*
03778 03779 04179* 04215 04685* 04702 04745* 04764
05141* 05143* 05147* 05275 05673* 05683* 05686* 05695*
05696* 05702 05741* 05742* 05743* 05750* 05751* 05754*
05762* 05768 05769 05770 05771 06148* 06155* 06208
06307 06677* 06690 06737 06750* 06755 06769* 06833
07048 07061 07440 07603 07604 07667* 07687 07689
07835 08044* 08947 09205 09292 09302

STOMACH BIOPSY
00132* 00146 00304* 00339 00789* 00834* 01034* 01166*
01167* 01177* 01209 01216 01232 01234 01242 01888
02050* 02052* 02912* 02928 02971 03106* 03889* 03890*
03940 03945 04184 04882 04958 05133* 05232* 05894
05900 06151* 06178 06188 06212 06673* 06859* 06860*
06936 06946 06951 07025* 07028* 07030* 07839* 07875
07896 08052 08068 08073 08211 08888 09179 09180
09184
STOMACH CANCER
00290* 00292* 00296* 00298* 00300* 00306* 00316 00318
00322 00331 00333 00344 00609 00837* 00929* 00935*
00948* 00963 00975 00999 01020* 01028* 01033* 01035*
01047* 01090 01129 01167* 01169* 01170* 01175* 01178*
01179* 01182* 01183* 01186 01188 01189 01190 01212
01215 01216 01219 01222 01224 01226 01227 01232
01235 01239 01240 01241 01243 01244 01895 01936*
01941* 02044* 02047* 02049* 02050* 02053* 02055* 02058*
02060 02061 02073 02077 02088 02092 02095 02096
02097 02234 02407* 03105* 03887* 03889* 03907* 03921*
03996 04004 04053 04063 04155 04175* 04183 04184
04185 04186 04188 04191 04193 04194 04196 04198
04203 04206 04208 04215 04223 04275 04299 04691*
04699 04882 04891 04997 04999 05081* 05130* 05131*
05132* 05134* 05135* 05139* 05142* 05143* 05158 05161
05168 05222* 05835* 05863* 05865* 05884* 05894 05912
05923 05929 06049 06061 06143* 06148* 06150* 06156*
06161* 06164 06168 06170 06171 06178 06179 06183
06184 06191 06198 06199 06202 06204 06212 06748*
06755 06788* 06838* 06843* 06859* 06904 06936 07007*
07008* 07014* 07015* 07018* 07019* 07020* 07021* 07023*
07026* 07029* 07032 07034 07035 07041 07048 07050
07051 07052 07053 07054 07055 07056 07061 07065
07066 07071 07072 07073 07076 07146 07147 07155
07180 07286 07329 07545* 07587 07850* 07869 07900
07916 08037* 08038* 08039* 08042* 08044* 08047 08049
08055 08058 08064 08066 08070 08071 08076 08078
08082 08083 08086 08089 08090 08091 08093 08096
08101 08102 08109 08111 08246 08392 08620 08669
08722* 08725* 08742* 08764 08836 08913 09031 09048
09051 09056 09064 09065 09085 09094 09172 09173
09188 09190 09193 09194 09197 09206 09212 09213
09293 09577 09921 09927
STOMACH CARCINOGENESIS
01173* 01233 01243 01702* 01943 02082 02088 02108
02258 04122 06142* 06209 07008* 07014* 07018* 07022*
07023* 07126* 08080 09194
STOMACH CHEMICAL COMPOSITION
00040* 00041* 00064* 00134 03694* 03769* 05702 05762*
06307
STOMACH CIRCULATION
00004* 00808 00861 01175* 01704* 01711* 01747* 01762*
01952 02045* 02111 02112 02709 02779* 02798* 02800*
02892* 03693 04099 04192 04218 05136* 05161 05223*
05667* 05668* 05679 07126* 07127* 07130* 07167 07900
07912 07997 08033*
STOMACH CONTENTS
03679 06179 06180 07687 08032
STOMACH CYSTS
08069
STOMACH DEVELOPMENT
00861 01187 01217 01220 01221 01712* 0205 4698
04706 04708 05154 06677* 06695 06751* 0705
STOMACH DIAGNOSIS
00105* 00130* 00132* 00146 00162 00163 92* 00304*
00317 00318 00335 00336 00337 00339 834* 00920*
00959 00965 00970 00974 01007 01014 01035*
01111 01166* 01167* 01168* 01169* 01174* [illegible] 01177*
01178* 01182* 01185 01201 01203 01209 01216 01220
01228 01229 01232 01234 01242 01332 01858* 01872*
01888 01899 01973 02050* 02052* 02059 02070 02076
02079 02080 02088 02094 02098 02099 02106 02110
02242 02885* 02886* 02892* 02904* 02905* 02912* 02921
02928 02942 02954 02969 02971 02980* 03099* 03108*
03114* 03126 03130 03134 03135 03137 03141 03146
03887* 03889* 03890* 03907* 03921* 03927 03935 03940
03945 03977 03985 03996 04004 04176* 04181* 04183
04184 04186 04196 04197 04205 04211 04215 04223
04874 04876 04882 04891 04914 04957 04958 05057
05133* 05138* 05168 05173 05232* 05835* 05839* 05852*
05863* 05883* 05884* 05894 05900 05902 05903 05912
05923 05928 05939 05940 05942 05945 05946 05962
06148* 06151* 06158* 06163 06165 06169 06170 06171
06172 06178 06185 06188 06193 06195 06203 06207
06212 06740* 06838* 06841* 06843* 06846* 06849* 06850*
06859* 06860* 06883* 06902 06912 06913 06918 06919
06920 06927 06936 06941 06946 06951 06956 06958
06979 06985 07019* 07020* 07025* 07032 07040 07057
07069 07070 07071 07076 07839* 07845* 07846* 07850*
07854* 07855* 07856* 07857* 07872 07885 07888 07896
07900 07912 07916 07929 07935 07952 07982* 08046
08046 08048 08052 08064 08065 08066 08068 08075
08081 08082 08084 08091 08093 08096 08101 08104
08107 08171* 08389 09031 09040 09044 09051 09056
09064 09065 09085 09097 09098 09107 09109 09118
09155 09168 09169 09184 09185 09193 09197 09199
STOMACH DIAGNOSIS
CONTINUED
09203 09208 09215 09272 09274 09282 09284 09306
09307 09308 09936
STOMACH DIAGNOSIS,GASTRIC ANALYSIS IN
00053 00118* 00317 00340 00343 00383* 00413 00848*
00979 01309* 02064 02087 02094 03945 04881 05133*
05134* 05879* 05879* 06904 09192
STOMACH DISEASE EPIDEMIOLOGY
00337 03106*
STOMACH DISEASE ETIOLOGY
00228
STOMACH DISEASES
00130* 00243 00310* 00329 00417 00819* 01035* 01055
01087 01100 01185 01231 01926* 01952 01999 02046*
02053* 02068 02099 02107 02206* 02216* 02942 03111*
03120 03127 03139 03147 03228* 04168* 04173* 04179*
05012 05135* 05157 05902 06162 06163 06193 06203
06508 07049 07069 08099 08104 08107 08110 08762
09182 09184 09460 09784 09875
STOMACH DISEASES,BIOCHEMICAL DIAGNOSIS OF
01129 02905* 02980*
STOMACH DIVERTICULA
02079 04176* 05157 05159 06185 06207 07249 08762
08773 09186
STOMACH ELECTROLYTE SECRETION
00301* 00305* 00819* 00859 01492* 01754* 01755* 01756*
01757* 01777* 01787 02235 02817 03732* 03747* 03763*
03770* 03772 03796* 04745* 04747* 04763 06744* 06747*
07665* 07678* 07688 08044* 08168* 09316
STOMACH ENDOSCOPY
00105* 00130* 00132* 00146 00162 00163 00317 00335
00965 01007 01111 01167* 01182* 01186 01203 01209
01216 01232 01242 01332 01858* 01973 02050* 02052*
02080 02098 02099 02886* 02904* 02912* 02921 02942
02954 02971 03108* 03114* 03137 03146 03889* 03907*
03921* 03940 03945 03977 03996 04004 04184 04874
04876 04957 05133* 05835* 05900 05902 05903 05942
05945 05962 06021 06158* 06172 06212 06841* 06843*
06846* 06849* 06850* 06859* 06860* 06918 06919 06927
06936 06941 06946 06951 06985 07013* 07032 07068
07069 07128* 07845* 07849* 07855* 07872 07873 07885
07888 07896 07916 07935 08046 08052 08389 08807
08855 09031 09040 09044 09065 09094 09097 09098
09183 09184 09198 09282 09308
STOMACH ENZYMES
00001* 00298* 00789* 00838* 00880 01309* 01701* 01761*
01781 01794* 02980* 03110* 03715 04179* 04215 04702
05143* 05147* 05686* 05741* 05768 06148* 06155* 06677*
06690 06737 06740* 06769* 06833 07048 07440 08076
08947 09205
STOMACH FISTULAS
00041* 00046* 00397* 01655* 01754* 01755* 01763* 01767*
01779* 01785 01949 02803* 03746* 03764* 04032* 04741*
04749* 05027 05935 06761 07682* 08065 08309 08910
09200 09774
STOMACH HISTOLOGY
02057* 03112* 03118 03695* 03697* 03705* 03712* 03713*
03890* 04213 04214 04215 04698 04706 04708 04958
05129* 05136* 05140* 05141* 05148* 05169 05215* 05626
05673* 05683* 05684* 05686* 05744* 05835* 05945 06144*
06148* 06158* 06159* 06161* 06169 06183 06188 06672*
06673* 06677* 06740* 07008* 07012* 07016* 07018* 07025*
07032 07048 07061 07062 07130* 07665* 07839* 07849*
08069 08073 08076 08084 08093 08096 08099 08389
08931 09114 09179 09197 09205 09206 09214 09274
09279
STOMACH IMMUNOLOGY
SEE ALSO GASTRIC PARIETAL-CELL ANTIBODY
00308* 00320 00321 01180* 01209 01241 01899 02943
02943 03109* 03111* 03113* 03237* 04167* 04173* 05016
05129* 05148* 05149* 05702 06162 06176 06748* 07030*
07031* 07055 07594* 07856* 09176 09183 09209 09921
STOMACH IN CHILDREN
00286 00786* 01097 01210 01213 01222 03120 03127
03129 03139 04180* 04200 04211 04212 04771* 05170
05174 05175 05939 06143* 06175 06187 06695 06739*
06751* 07012* 07035 07058 07059 07070 07074 07600
08031 08048 08104 08203 08214 09272 09301
STOMACH INJURIES
06127 06144* 09856
STOMACH INTRINSIC FACTOR
00040* 00189* 00858 00859 01180* 01230 01310* 02749*
03000* 03109* 03237* 03241* 04031* 04213 05127* 05129*
05148* 05149* 05167 06707* 06748* 06751* 07031* 07190*
09180
STOMACH METABOLISM
00001* 00298* 02798* 05757* 06695 08110
STOMACH MICROORGANISMS
00196* 00369 05134* 06150*
STOMACH MORPHOLOGY
00060 00061 00295* 00304* 00315 00384* 00408 00813*
01181* 01205 01309* 01703* 01711* 01712* 02057* 02058*
02069 02103 02109 02270 03118 03701* 03712* 03713*
04186 04699 04708 04757 04869 05684* 05695* 05903
06667* 06729 07059 07845* 07846* 07849* 07896 07912

TOMACH MORPHOLOGY
CONTINUED
08207 08790 09155 09215 09279 09330
TOMACH MOTILITY
00046* 00068* 00072* 00073* 00079* 00103* 00104* 00186*
00309* 00766* 00886* 00887* 00888 00890 01048* 01171*
01172* 01176* 01316* 01359 01704* 01796* 01797* 01800*
01803* 01807* 01809* 01810* 01822 01855* 01859* 01867*
02072 02091 02117* 02198* 02789* 02830* 02833 02972*
03064 03100* 03144 03765* 03793* 03794* 03795* 03796*
03800 03801 03802 03803 04086 04132 04170* 04766*
04769* 04772* 04773* 04869 05156 05679 05771 05787*
05797 05883* 05946 06052 06122 06154* 06201 06236*
06244* 06245* 06282 06726* 06728 06729 06733 06767
06883* 06912 07038 07062 07063 07069 07132* 07589
07658* 07659* 07662* 07679* 07850* 08048 08110 08187
08805 09175 09215 09936
TOMACH MUCOSA
00001* 00004* 00021* 00061 00105* 00289* 00295* 00298*
00301* 00304* 00305* 00310* 00315 00339 00378* 00384*
00789* 00808 00810 00834* 00835* 00840* 00844* 00845*
00862 00876 00975 01027* 01048* 01051 01080 01082
01166* 01171* 01173* 01176* 01177* 01179* 01180* 01181*
01197 01202 01220 01232 01301* 01309* 01314* 01335
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01781 01784 01787 01924* 02045* 02046* 02057* 02059
02078 02090 02196* 02705* 02709 02741 02770 02779*
02783* 02798* 02799* 02800* 02801* 02806 02807 02813
02971 02992* 03099* 03103* 03112* 03119 03122 03136
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03713* 03715 03732* 03747* 03778 03779 03884* 03890*
03927 03965 04100 04168* 04179* 04192 04213 04214
04216 04220 04290 04300 04310* 04674* 04698 04699
04702 04706 04708 04747* 04757 04761 04764 04765
04876 04958 05005 05133* 05135* 05136* 05142* 05146*
05147* 05154 05155 05157 05166 05168 05169 05172
05215* 05223* 05274 05276 05673* 05683* 05695* 05696*
05697* 05702 05736* 05744* 05745* 05755* 05768 05778
05923 05981* 06013 06016 06043 06069 06146* 06161*
06169 06179 06180 06182 06192 06197 06200 06205
06208 06209 06672* 06673* 06738* 06740* 06743* 06744*
06755 06759 06764 06768 06769* 07009* 07013* 07016*
07017* 07025* 07030* 07031* 07045 07047 07049 07055
07069 07072 07584 07604 07613 07665* 07674* 07676*
07677* 07683 07845* 07872 07885 07889 07896 07916
08044* 08068 08069 08085 08091 08108 08168* 08175*
08190 08207 08762 08780 08931 08947 08951 08952
08953 09097 09169 09114 09179 09180 09181 09183
09194 09195 09197 09198 09199 09205 09206 09214
09215 09302 09573 09737 09935
TOMACH MUCOSAL INJURIES
00301* 00310* 00819* 01185 01231 02048* 02112 02216*
02982* 03107* 03108* 03120 04168* 05140* 05157 05266
06158* 06159* 06160* 07127* 07994* 08033* 08040* 08081
08168*
TOMACH MUCUS SECRETION
00047* 00398* 00858 00859 00862 01794* 02783* 03694*
03695* 03705* 03757* 03769* 03771 03775 03779 04216
06741* 06750* 06934 07037 07125* 07688
TOMACH NEOPLASMS
00130* 00261 00293* 00300* 00322 00329 00330 00334
00337 00956 01204 01985 02104 02249 03104* 03121
05128* 05903 05994* 07011* 07032 07057 07846* 07929
08080 08096 08762 09098 09185 09194 09206
TOMACH NEOPLASMS,BENIGN
00132* 00292* 00325 00332 00333 00341 01174* 01182*
01233 01234 02047* 02059 02104 02234 03126 03130
03134 03135 04191 04193 04199 05151 06165 06169
06194 07022* 08046 08053 08057 08084 08101 08104
08106 09178 09193 09199 09203 09208 09216 09823
09905
TOMACH NEOPLASMS,MALIGNANT
00132* 00332 01096 01169* 01173* 01176* 01185 01190
01196 01201 01226 01228 01233 01335 01998 02052*
02057* 02082 02085 02086 02088 02092 02093 02108
02113 02242 02258 02885* 02886* 02912* 02947 02975*
02986* 03009 03015 03047 03098* 03099* 03105* 03107*
03110* 03111* 03116* 03117* 03123 03131 03133 03140
03141 04065 04191 04225 05173 05863* 05923 06142*
06148* 06150* 06168 06194 06204 07015* 07023* 07029*
07067 07071 07076 08042* 08044* 08056 08058 08075
08076 08093 08096 08106 08620 09056 09170 09174
09193 09212 09213
TOMACH NEOPLASMS,VILLOUS
09199
TOMACH OBSTRUCTION
01231 04043* 05157 05171 05172 05946 06172 07043
07069 07074 08712 09098 09272 09866
TOMACH PATHOLOGY
00006* 00323 00329 00959 01054 01175* 01197 01203
01204 01208 01220 01226 01232 01241 01243 01326
01335 01915 02031 02067 02070 02108 03063 03130
03133 03141 03213 03985 04171* 04199 04206 04213
04217 05005 05128* 05134* 05135* 05140* 05142* 05154
05161 05168 05171 05172 05177 05903 05945 06158*
06159* 06160* 06194 06197 06198 07008* 07012* 07018*
STOMACH PATHOLOGY
CONTINUED
07030* 07045 07055 07061 07935 07998 08081 08085
08093 08104 08106 08110 09098 09114 09170 09172
09178 09182 09183 09188 09193 09197 09198 09199
09201 09207 09208 09267 09272 09275 09284 09286
09479 09905 09907 09908 09909
STOMACH PEPSIN SECRETION
00041* 00045* 00064* 00275 00297* 00299* 00834* 00837*
00838* 00845* 00846* 00850* 00858 00859 00862 00872
00880 00955 01090 01166* 01296* 01301* 01749* 01754*
01755* 01757* 01758* 01761* 01762* 01767* 01777* 01781
01784 01794* 02697* 02785* 02788* 02789* 02795* 02796*
02801* 02806 02807 02992* 03136 03705* 03760* 03761*
03775 03776 04216 04740* 04756 05742* 05743* 05749*
05754* 05755* 05762* 05764* 05766 05773 06002* 06155*
06166 06258* 06738* 06739* 06740* 06742* 06746* 06748*
06749* 06753* 06754* 06763 06764 06766 06767 06769
06841* 06858* 06902 06934 07063 07446* 07674* 07682*
07684 07688 07854* 08059 08180 08200 09078 09294
STOMACH PERFORATION
00327 00336 01207 01210 01212 01213 01514* 02005
02081 03129 03132 03138 03139 03186* 04027* 05088*
06035 06175 06301 06582* 07056 08034* 08100 08762
08789 09212 09286 09288 09289
STOMACH PERFUSION
00865 01779* 04749* 06160* 06160* 07009* 07674* 07677*
07678* 07682*
STOMACH POLYPS
00332 00337 00340 00341 00342 00343 00920* 00948*
01167* 01174* 01175* 01182* 01256* 02076 03099* 03126
03135 04193 04223 06150* 06169 06191 06195 06843*
07032 07045 07068 07073 07329 07916 08046 08084
08089 08101 08246 08389 08762 09098 09197 09206
09216 09823 09897
STOMACH RADIOLOGY
00292* 00317 00334 00337 00340 00417 00572 00920*
00959 00975 00999 01014 01034* 01035* 01048* 01167*
01168* 01174* 01176* 01178* 01182* 01185 01198 01199
01200 01209 01216 01217 01221 01228 01229 01232
01234 01298* 01319* 01320* 01361 01872* 01909 01915
01952 01985 02052* 02059 02078 02079 02080 02091
02108 02109 02110 02236 02885* 02886* 02905* 02969
03099* 03103* 03126 03135 03889* 03890* 03921* 03927
03935 03940 03945 03996 04004 04019 04176* 04181*
04186 04191 04197 04202 04203 04205 04211 04869
04882 04957 05134* 05138* 05163 05168 05173 05235
05839* 05900 05902 05912 05939 05940 05942 05945
05990* 06013 06016 06143* 06163 06169 06170 06171
06178 06185 06192 06195 06203 06207 06212 06843*
06883* 06912 06920 06936 06941 06963 07020* 07057
07071 07076 07128* 07180 07600 07845* 07846* 07850*
07857* 07875 07888 07896 07900 07912 07916 07929
07982* 08019 08048 08065 08066 08075 08081 08082
08084 08091 08093 08101 08171* 08389 08790 08931
09051 09056 09065 09085 09098 09109 09118 09155
09169 09184 09185 09203 09207 09215 09274 09284
09306 09307 09308
STOMACH SECRETION
00039 00053 00056 00057 00109* 00196* 00223 00307*
00328 00339 00393* 00398* 00797 00833* 00850* 00854
00861 01092 01104 01181* 01194 01209 01299* 01702*
01719* 01727* 01747* 01768* 01769* 01770* 01780 01790
01791 01989 02054* 02071 02087 02093 02392* 02703*
02754* 02761* 02806 02822 02823 02892* 03063 03145
03746* 03762* 03775 03801 03981 04097 04213 04436*
04602* 04674* 04754 04757 04764 05167 05169 05274
05455* 05684* 05740* 05743* 05750* 05751* 05761* 05762*
05768 05839* 06154* 06177 06200 06275 06307 06309
06690 06755 06759 07030* 07031* 07062 07135* 07594*
07681* 07682* 07686 07688 07689 07835 07983* 08006
08033* 08950 08951 08952 09077 09176 09180 09184
09292 09302 09303 09304 09319 09573 09737 09921
STOMACH SECRETION DISORDERS
00040* 00041* 00118* 00188* 00189* 00290* 00291* 00294*
00312* 00320 00383* 00845* 00869 00935* 00970 00974
01034* 01166* 01361 01485* 01872* 02125* 03000* 03109*
03113* 03723* 03749* 03768* 03887* 03945 03974 03978
04031* 04049* 04193 04213 04258* 04989* 05031 05053
05098 05131* 05266 05418 05435* 05626 05764* 05852*
05867* 05879* 05884* 05928 06000* 06014 06018 06145*
06150* 06166 06208 06210 06240* 06252* 06598 06742*
06748* 06766 06858* 06934 06956 06958 07014* 07019*
07027* 07039 07040 07041 07068 07134* 07152 07154
07160 07176 07361 07362 07581 07582 07583 07584
07839* 07846* 07856* 08044* 08166* 08176* 08458 08768
08769 08779 08780 08798 08824 08826 09097 09210
09298 09313 09396 09809 09824 09865
STOMACH SECRETION IN DISEASE
00042* 01090 01933* 02215* 02820 02905* 02947 02948
02980* 02988* 03000* 03036 03193* 03237* 03761* 05141*
05143* 07446* 09194
STOMACH SECRETION STUDY TECHNIQUES
00040* 00049* 00049* 00060 00133 00291* 00392* 00397*
00413 00807 00838* 00847* 00848* 00858 00865 00934*
00974 00993 00995 01001 01090 01194 01198 01199

STOMACH SECRETION STUDY TECHNIQUES
CONTINUED
01200 01308* 01313* 01754* 01755* 01756* 01757* 01770*
01774* 01775* 01781 01794* 01859* 01872* 01912 02094
02235 02236 02697* 02790* 02800* 02801* 02820 02899*
02905* 02909* 03065 03180* 03705* 03748* 03749* 03751*
03755* 03756* 03757* 03758* 03760* 03767* 03773 03887*
03945 03978 04224 04291 04740* 04746* 04759 04881
04914 05217* 05746* 05756* 05759* 05840* 05879* 05884*
05928 06252* 06667* 06750* 06755 06858* 06902 06922
06934 06950 06956 06958 07469* 07674* 07675* 07677*
07678* 07679* 07687 07839* 07845* 07857* 07896 08165*
08168* 09107 09192
STOMACH SECRETION,DRUG EFFECTS ON
00040* 00041* 00043* 00046* 00328 00383* 00384* 00397*
00819* 00831* 00832* 00842* 00846* 00847* 00853 00867
00871 00872 00888 00953* 00986 01172* 01310* 01313*
01314* 01702* 01703* 01748* 01749* 01767* 01768* 01770*
01775* 01778* 01782 01791 02090 02216* 02788* 02789*
02792* 02796* 02799* 02808 02821 03000* 03122 03180*
03190* 03193* 03695* 03747* 03762* 03769* 03778 03796*
04141 04256* 04259* 04756 04764 05140* 05146* 05215*
05218* 05239 05268 05748* 05755* 05763* 06210 06235*
06252* 06258* 06259* 06287 06744* 06752* 06754* 06769*
07675* 07676* 07678* 07682* 07685 07687 08059 08178*
08200 09246 09307
STOMACH SECRETION,HORMONAL CONTROL OF
00046* 00049* 00054 00057 00831* 00832* 00833* 00840*
00841* 00846* 00847* 00848* 00850* 00852* 00859 00862
00870 00871 00873 00993 01310* 01314* 01358 01359
01748* 01749* 01750* 01754* 01755* 01758* 01774* 01775*
01776* 01777* 01782 01789 01790 01791 01810* 01899
02235 02703* 02779* 02780* 02785* 02788* 02792* 02794*
02796* 02800* 02801* 02806 02814 03000* 03693 03755*
03756* 03758* 03760* 03764* 03767* 03768* 03770* 03772
03773 03776 03784 04037* 04267* 04740* 04741* 04745*
04746* 04748* 04749* 04752* 04755 04756 04758 04763
05017 05018 05213* 05229* 05686* 05695* 05696* 05736*
05741* 05742* 05745* 05746* 05753* 05754* 05757* 05758*
05759* 05760* 05763* 05766 05769 05770 05771 05772
05773 05775 05776 06000* 06014 06213 06247* 06255*
06739* 06741* 06742* 06746* 06747* 06749* 06750* 06752*
06756 06757 06758 06760 06761 06767 06768 06769
06922 06956 07127* 07152 07154 07169 07665* 07675*
07676* 07679* 07682* 07683 07684 08176* 08190 08720*
08769 08948 08949 09315
STOMACH SECRETION,NERVOUS CONTROL OF
00043* 00054 00059 00299* 00307* 00324 00831* 00841*
00842* 00843* 00848* 00852* 00856 00857 00859 00862
00867 00868 00871 00872 00873 00874 01358 01359
01362 01749* 01759* 01778* 01794* 02212* 02235 02779*
02779* 02785* 02788* 02789* 02792* 02793* 02794* 02795*
02799* 02806 02809* 02818 02819 02909* 03180* 03705*
03754* 03755* 03757* 03758* 03760* 03776 03784 03978
04086 04256* 04259* 04742* 04748* 04751* 04756 04758
05229* 05250 05258 05679 05748* 05753* 05772 06252*
06258* 06259* 06289 06292 06295 06667* 06738* 06741*
07039 07154 07275* 07604 07675* 07684 08165* 08168*
08172* 08188 08203
STOMACH SURGERY
SEE ALSO GASTRECTOMY
00181* 00261 00288 00297* 00309* 00313* 00319 00319
00324 00337 00363 00379* 00388* 00557 00673 00807
00835* 00970 01038* 01047* 01170* 01171* 01174* 01178*
01182* 01183* 01184* 01186 01187 01193 01210 01211
01213 01215 01218 01219 01222 01225 01227 01233
01298* 01299* 01302* 01304* 01305* 01306* 01307* 01322*
01355 01358 01359 01361 01502 01788 01926* 01946
01952 01996 02011* 02018 02044* 02065 02066 02067
02071 02082 02083 02084 02091 02101 02105 02105
02106 02106 02110 02112 02150 02194* 02199* 02209*
02215* 02221 02235 02236 02237 02247 02250 02250
02259 02261 02407* 02442* 02541 02612 02663* 02780*
02803* 02899* 03004 03009 03012 03058 03101* 03102*
03107* 03111* 03113* 03128 03130 03134 03142 03158
03183* 03185* 03186* 03187* 03188* 03189* 03192* 03194*
03196 03197 03211 03239* 03271 03760* 03776 04040*
04051* 04054 04071 04085 04123 04159 04176* 04177*
04181* 04186 04191 04194 04198 04199 04201 04202
04207 04211 04260* 04261* 04262* 04263* 04264* 04265*
04266* 04269* 04274 04282 04292 04293 04296 04297
04730* 04748* 04773* 04869 04975* 05072 05112 05132*
05135* 05152 05153 05158 05163 05169 05170 05171
05172 05174 05177 05214* 05216* 05217* 05221* 05222*
05235 05236 05237 05240 05244 05245 05247 05253
05256 05262 05756* 05758* 05903 06045 06061 06109*
06143* 06147* 06152* 06157* 06165 06175 06178 06181
06184 06185 06187 06189 06195 06196 06201 06207
06217* 06239* 06254* 06259* 06263* 06264* 06266 06270
06275 06277 06278 06279 06283 06285 06301 06306
06308 06309 06310 06532* 06726* 06873* 06883* 06978
06988 07014* 07024* 07027* 07032 07043 07051 07052
07057 07066 07070 07071 07128* 07138* 07139 07146
07147 07153 07166 07168 07177 07178 07249 07275*
07329 07552 07677* 07680* 07871 07916 07984* 07990*
07995* 07997 07998 08001 08015 08016 08031 08036*
STOMACH SURGERY
CONTINUED
08037* 08046 08048 08049 08050 08051 08053 08055
08057 08060 08061 08062 08064 08065 08074 08084
08094 08097 08098 08099 08101 08102 08104 08105
08108 08111 08118* 08165* 08174* 08177* 08186 08187
08192 08196 08197 08201 08203 08210 08213 08214
08443* 08471 08712 08717* 08762 08779 08793 08816
08835 08845 08859 08950 09040 09098 09155 09157
09162 09169 09170 09182 09186 09187 09196 09201
09202 09203 09205 09207 09208 09215 09248 09252
09256 09261 09272 09274 09278 09284 09289 09296
09301 09304 09306 09309 09441 09484 09781 09811
09865 09912
STOMACH SURGERY COMPLICATIONS
07062 08067
STOMACH SURGERY TECHNIQUES
00800 01136* 01246* 02833 03186* 03228* 03228* 04054
04195 04282 04743* 04759 05051 05137* 05162 05164
05165 05170 05176 05258 05273 05883* 06152* 06167
06168 06174 06186 06190 06231 06246* 06265 06269
06277 06278 06288 06293 06300 07010* 07033 07036
07054 07056 07062 07074 07129* 07132* 07151 07166
07885 08034* 08169* 08182 08184 08276 09076 09189
09200 09268 09271
STOMACH TRANSPLANTATION
01136* 01314* 01705 04147 04192 05051 05136* 06122
06131 06990 07677*
STOMACH TRAUMA
00327 01038* 01108 02009* 02081 03107* 03128 03132
06160* 07677* 08081 08762
STOMACH TREATMENT
02069 04174* 05270 06043 06170 06193 07002 07070
08027 08081 08094 08178* 09183 09273
STOMACH ULCERS
02110 02218* 02255 02256 02256 02258 02267 02269
02272 02982* 03107* 03118 03186* 03190* 03196 03206
03212 03213 03214 03215 03218 03761* 03996 04179*
04190 04202 04276 04292 04300 04765 05220* 05226*
05232* 05235 05242 05244 05245 05250 05266 05273
05275 05764* 05863* 05879* 05894 05945 06149* 06150*
06160* 06208 06239* 06242* 06244* 06250* 06257* 06262*
06296 06298 06302 06307 06582* 06843* 07006 07010*
07017* 07018* 07020* 07062 07072 07125* 07127* 07128*
07133* 07138* 07141 07150 07167 07168 07180 07275*
07396 07656* 07676* 07873 07916 08059 08073 08089
08098 08100 08165* 08168* 08173* 08175* 08196 08197
08199 08200 08208 08209 08214 09045 09048 09076
09077 09098 09183 09186 09188 09201 09205 09212
09251 09252 09254 09257 09259 09261 09262 09263
09264 09267 09270 09272 09273 09275 09276 09277
09278 09279 09280 09281 09282 09283 09285 09286
09287 09288 09289 09290 09292 09293 09295 09301
09302 09306 09307 09308 09309 09319 09728 09908
09927 09935
STOMACH ULTRASTRUCTURE
02057* 03697* 04706 04761 05155 05683* 05684* 06673*
07017* 07613 07665* 08068 08931 09206
STOMACH VARICES
01035* 01117 01926* 01952 05063 05902 06203 06579*
07889 07900 07998 08729* 09185
STOMACH VOLVULUS
04043* 04197 05171 06206 07074 08001
STOMACH,AGE EFFECTS ON
00061 01203 02084 03106* 06695 06739* 06751* 08176*
09208
STOMACH,CLINICAL STUDIES OF
00068* 00306* 00319 00323 00413 00808 01205 01208
02078
STOMACH,CROHNS DISEASE OF
02080 02107
STOMACH,DISEASES ASSOCIATED WITH
00330 00340 00341 00342 00343 01998 03062
STOMACH,DRUG EFFECTS ON
04168* 04874 04978* 05668* 05686* 05744* 06758 07013*
07038 07603 07689 08040* 09195 09214 09282
STOMACH,DRUG TREATMENT OF
07047
STOMACH,HORMONAL CONTROL OF
01701* 01807* 01809* 02196* 03144 03712* 03713* 03765*
05146* 05668* 06672* 06726* 07063 07135* 07659* 07835
09315
STOMACH,NERVOUS CONTROL OF
00861 00886* 00888 00890 02103 02196* 02789* 02894*
03144 03794* 04132 04769* 04773* 05057 05169 05276
05668* 05679 05787* 05883* 06052 06122 06154* 06191
06201 06206 06243* 06297 06298 06726* 07012* 07017*
07049 07145 07659* 07662* 08110 08187 08193 08805
08950
STOMACH,TOXIC EFFECTS ON
00013* 00289* 00305* 00819* 01030* 01231 01762* 02069
02216* 02240 02723 02747 03114* 04168* 05112 06043
06144* 06158* 06159* 06205 06672* 07040 07547* 08033*
09179 09181 09204 09246 09282

RANGULATION
00240 00248 00365 00512 00795 01023* 02185 05032
05195 06090 06991 06999 07103 07117 07542* 07997
08304 08763 09900
RESS FACTORS
00006* 05685* 06008* 08270
RESS FACTORS IN ULCERS
01300* 01711* 01961 02213* 02214* 02244 02245 02251
02254 02273 02705* 02722 03198 03200 04100 04190
04259* 04267* 04279 04284 04290 04300 05261 05264
05266 06251* 06291 07017* 07130* 07131* 07150 07466*
08175* 08179 08180 09195 09294
RICTURE,BILIARY TRACT
01657* 01691 03631* 04488 05642* 05664 07496* 07500*
08058 08666* 08691 08692 08695 08698 08701 08715
09750 09780 09784
RICTURE,ESOPHAGUS
00273 01141* 02010* 02013* 02014* 02043 02113 02990*
03091 03911* 04135, 04139 04145 04147 05092 06116
07002 07006 07989* 08005 08009 08019 08030 08762
09135 09144 09153 09161 09162
RICTURE,SMALL INTESTINE
09232
RONTIUM ABSORPTION
00893* 00894* 01739 02752* 02772 03679 03734* 05856*
07632* 08942
UDIES OF CANCER,CLINICAL
00259 00290* 00344 00945* 01240 02058* 02077 02085
02309* 02310*
UDIES OF CANCER,EXPERIMENTAL
01581 01582 01583 01585 01586 02077
UDIES OF CIRRHOSIS,CLINICAL
00733 00737 00742 00743 00744 00746 00748 01590
01636* 01906 02473* 02628* 02638* 08653
UDIES OF CIRRHOSIS,EXPERIMENTAL
00729* 00730* 00731* 00732* 01633* 01720* 02579* 02656
07433 09589 09704
UDIES OF ESOPHAGUS,CLINICAL
00269 00272 00274 00275 00276 00278 00287 01148
01163 01645 01908 02019 02024 02038
UDIES OF GALLBLADDER,CLINICAL
00767
UDIES OF GASTROINTESTINAL TRACT,CLINICAL
00154 00211 00231 01062 01102 01898 02135 02182
UDIES OF HEPATITIS,CLINICAL
00020 00634 00635 00639 00704 00705 00706 00710
00717 00722 01875* 01894 02593 02607 02611 02614
08523 08604* 08628
UDIES OF HEPATITIS,EXPERIMENTAL
00707 00721 00723 01626 01875* 02623 05442* 06459*
06553* 07345* 07805 08596* 08624 09671
UDIES OF JAUNDICE,CLINICAL
00596* 00633 00637 00643 00679 02599*
UDIES OF LARGE INTESTINE,CLINICAL
00457 00467 00475 00497 00516 00958 01458 01463
01740 02311* 02326* 02330* 02341
UDIES OF LIVER,CLINICAL
00093 00159 00208 00597* 00599* 00608* 00612 00613
00614 00615 00616 00617 00618 00619 00625 00630
00640 00657 00658 00659 00661 00664 00669 00691
00742 01592 01594 01595 02527 02535 02538 02544
02584
UDIES OF LIVER,EXPERIMENTAL
00087* 01609 02527 02558 02573 02582* 02583 02589
UDIES OF PANCREAS,CLINICAL
00193* 00538* 00545 00553 00557 00568 00574* 01486*
01491* 01506 02416 02423
UDIES OF PANCREAS,EXPERIMENTAL
02432
UDIES OF PANCREATITIS,CLINICAL
00583 00584 00586 00587 00592
UDIES OF PARASITIC DISEASES,CLINICAL
00128* 00136 00180* 00256 00463
UDIES OF PEPTIC ULCERS,CLINICAL
00144 00377* 00380* 00398* 00410 00412 00415 00416
02232
UDIES OF SALMONELLOSIS,CLINICAL
00255 01466*
UDIES OF SALMONELLOSIS,EXPERIMENTAL
02384* 08744*
UDIES OF SCHISTOSOMIASIS,CLINICAL
00192* 00205
UDIES OF SCHISTOSOMIASIS,EXPERIMENTAL
00680
UDIES OF SMALL INTESTINE,CLINICAL
00357 00359 00360 02169
UDIES OF STOMACH,CLINICAL
00068* 00306* 00319 00323 00413 00808 01205 01208
02078
UDIES OF ULCERATIVE COLITIS,CLINICAL
00531* 00532* 00536 01469* 01481 02396*
UDIES OF ULCERS,CLINICAL
00103* 00406 00409 00417 01357 01364 02222 02225
02229 02258
STUDY TECHNIQUES,ABSORPTION
00035 00117* 00816* 00818* 01723* 01726* 01746 01870*
01898 01979 02750* 02752* 02759* 02763* 03061 03726*
03886* 04000 04710* 04711* 04717* 04720* 04721* 04728*
04850* 04854* 04950 05288* 05720* 05727 05861* 06219*
06691 06705* 06706* 06715 06881* 06961 07622* 07628*
07643* 07645 07647 07648 07915 07919 08054 08077
08134 08238 08648 08935 08937 08940 09176
STUDY TECHNIQUES,BILE SECRETION
04006 04007 07660*
STUDY TECHNIQUES,CIRCULATION
01239 01590 01591 04899 04935 05667* 05668* 06482
STUDY TECHNIQUES,DYE
SEE ALSO MARKER STUDIES
01871*
STUDY TECHNIQUES,GASTROINTESTINAL CIRCULATION
00798 00799 00897 00903 01977 01984 01989 01990
01994 02036 02112 02121* 02315* 02353 02510 02514
03903* 09036
STUDY TECHNIQUES,PANCREAS SECRETION
03993 06772* 06773*
STUDY TECHNIQUES,RADIOISOTOPE
SEE TRACER STUDIES
STUDY TECHNIQUES,SECRETION
05747* 05752* 05767 06618* 06734* 06880* 06914 06924
07694 07854*
STUDY TECHNIQUES,STOMACH SECRETION
00040* 00049* 00049* 00060 00133 00291* 00392* 00397*
00413 00807 00838* 00847* 00848* 00858 00865 00934*
00974 00993 00995 01001 01090 01194 01198 01199
01200 01308* 01313* 01754* 01755* 01756* 01757* 01770*
01774* 01775* 01781 01794* 01859* 01872* 01912 02094
02235 02236 02697* 02790* 02800* 02801* 02820 02899*
02905* 02909* 03065 03180* 03705* 03748* 03749* 03751*
03755* 03756* 03757* 03758* 03760* 03767* 03773 03887*
03945 03978 04224 04291 04740* 04746* 04759 04881
04914 05217* 05746* 05756* 05759* 05840* 05879* 05884*
05928 06252* 06667* 06750* 06755 06858* 06902 06922
06934 06950 06956 06958 07469* 07674* 07675* 07677*
07678* 07679* 07687 07839* 07845* 07857* 07896 08165*
08168* 09107 09192
SUCROSE ABSORPTION
04688* 08244
SUGAR INTOLERANCE
00065 00153 01589 08237 08495
SULFOBROMOPHTHALEIN
00598* 00621 00713 00992 01535* 01543* 02479* 02602*
02840* 02858 03361* 03362* 03369* 03482* 03532 03642
03660 03811* 04539 04570* 04802* 04806* 04823 04841*
04847* 04892 04899 04902 04904 04908 04909 04910
04911 04912 05423* 05449* 05450* 05637* 05822* 05829
05836* 05842* 05858* 05890 05899 05951 05952 06449*
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07386 07747* 07752* 07754 07788 07851* 07852* 07866*
07941 07969 08441* 08507 08528 08558* 08586 08657*
08990 09003 09042 09775
SURGERY COMPLICATIONS
07993* 08252* 08895 09187 09511
SURGERY COMPLICATIONS,STOMACH
07062 08067
SURGERY TECHNIQUES
01975 02680 02689 02690 04095 04159 04293 04346
04349 04351 04355 04490 04638 04644 04646 04658
04665 04975* 05200 05231* 05259 05279 05311* 05312*
05321 05322 05340 05345 05348 05502 05503 05510
05638* 05657 05662 05663 05983* 06045 06061 06115
06285 06338 06365 06408 06586 06587 06599 06635
06644 06650 06656 06659 06661 06970* 06997 07004
07077* 07099 07104 07118 07206* 07209* 07219 07243
07246 07256 07280* 07298* 07305* 07328 07339* 07340*
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07497* 07508 07545* 07552 07694 07762 07983* 07984*
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08197 08262* 08269* 08275 08278 08282 08293 08301
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08695 08701 08713 08717* 08815 08842 08851 08872
08878 08908 08950 09038 09072 09130 09131 09132
09141 09152 09153 09155 09157 09161 09344 09364
09370 09372 09373 09374 09375 09383 09384 09392
09436 09443 09445 09497 09511 09513 09516 09517
09538 09539 09541 09744 09747 09751 09753 09764
09768 09769 09778 09787 09802 09827 09856 09893
09927 09930
SURGERY TECHNIQUES,STOMACH
00800 01136* 01246* 02833 03186* 03228* 03228* 04054
04195 04282 04743* 04759 05051 05137* 05162 05164
05165 05170 05176 05258 05273 05883* 06152* 06167
06168 06174 06186 06190 06231 06246* 06265 06269
06277 06278 06288 06293 06300 07010* 07033 07036
07054 07056 07062 07074 07129* 07132* 07151 07166
07885 08034* 08169* 08182 08184 08276 09076 09189
09200 09268 09271

SURGERY,ABDOMEN
00240 00246 00248 00462 01039* 01040* 01078 01085
01107 01133 01164 01444 01953 02011* 02128* 02355
02464 03018 03021 03040 04032* 04054 04071 04120
04152 04427 04928 04991 04992 04999 05000 05096
05960 05983* 06023 06024 06034 06046 06050 06291
06963 07000 07118 07217* 07242 07253 07285 07286
07552 07559 07561 07568 07657* 07941 07990* 08117*
08118* 08128 08143 08144 08310 08334 08401 08514
08549* 08566 08568 08748 08752 08772 08829 08832
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09790 09812 09817 09820 09846 09856 09857 09868
09885 09886 09901 09922 09925 09965 09987
SURGERY,ANORECTAL
01382* 01390* 01407* 01408* 01462 08310 08345
SURGERY,ANUS
01386* 01419 02313* 02364 04351 05312* 05316* 05325
05326 05330 06328* 07242 07243 07245 07260 08257*
08291 08292 08293 08294 08345 08351 09358 09361
09362 09372 09373 09418
SURGERY,APPENDIX
09347
SURGERY,BILIARY TRACT
00161 00351* 00563 00752* 00756* 00757* 00758* 00759
00760 00771 00775 00776 00777 00780 00962 01022*
01038* 01342 01509* 01510* 01548* 01566 01587 01652*
01654* 01657* 01658* 01659* 01680 01682 01684 01692
01901 01942 01948 01975 02412* 02442* 02664* 02665*
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03411 03475 03626* 03629* 03631* 03634 03635 03654
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06477 06483 06507 06516 06618* 06624* 06628* 06629*
06630* 06633 06635 06642 06644 06645 06647 06654
06655 06656 06659 06661 06662 06665 06668* 06878*
06914 06964 07302* 07339* 07346* 07366 07389 07390
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07501* 07508 07513 07518 07520 07521 07522 07527
07528 07531 07532 07534 07537 07538 07539 07540
07552 07582 07762 07878 07879 07938 07964 08152
08156 08405 08434* 08465 08467 08469 08470 08510
08522 08567 08640 08656* 08658* 08660* 08661* 08663*
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08696 08698 08699 08700 08701 08702 08705 08707
08708 08710 08712 08713 08714 08793 09034 09062
09072 09220 09221 09474 09478 09484 09558 09594
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09751 09752 09753 09754 09755 09763 09767 09768
09769 09773 09774 09776 09777 09780 09781 09782
09783 09785 09786 09798 09802 09807 09868
SURGERY,CANCER
00260 00346* 00446* 00551 00624 00632 00929* 01017*
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01942 02008* 02039 02044* 02055* 02056* 02058* 02095
02304* 02306* 02309* 02310* 02329* 02340 02367 02369
02371 02407* 02495 02665* 03075* 03117* 03262* 03276
03292 03333* 03625* 04124 04126* 04140 04146 04151
04156 04194 04198 04201 04203 04208 04244 04246
04327* 04333* 04348 04355 04390* 04975* 04991 04999
05081* 05089 05107 05130* 05132* 05153 05158 05189
05212 05222* 05310* 05313* 05327 05328 05329 05339
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06199 06202 06344 06355 06364 06367 06370 06403
06410 06447* 06478 06635 06882* 06936 06963 06972*
06990 06996 06997 07003 07007* 07026* 07029* 07034
07035 07052 07054 07065 07066 07120 07146 07207*
07208* 07209* 07232 07240 07247 07257 07260 07291
07520 08015 08017 08037* 08039* 08064 08070 08071
08074 08086 08109 08111 08125 08155 08257* 08260*
08261* 08262* 08265* 08274 08275 08296 08302 08309
08310 08314 08320 08322 08325 08326 08355 08383
08433* 08510 08514 08517 08529 08535 08675 08717*
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09364 09371 09377 09384 09392 09437 09440 09443
09457 09463 09493 09516 09562 09569 09743 09749
09753 09796 09803 09831 09892
SURGERY,CHOLELITHIASIS
00753* 00775 01028* 01651* 01658* 02668* 02670 03627*
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05647* 05649* 05651 05657 05659 05665 06628* 06633
06635 06650 06652 06656 06661 06662 06663 07491*
07511 07522 07525 07531 07534 08519 08661* 08672
08685 08693 08695 08696 08699 08702 09220 09221
09495 09741 09752 09757 09763 09769 09780
SURGERY,DUODENUM
02001 03168 03185* 03201 03324 04244 04254 05237
06153* 06224 06225 06227 06301 06306 07108 07110
07277* 07496* 07539 08045* 08061 08138 08181 08210
SURGERY,DUODENUM
CONTINUED
08212 08394 08420 08678 08679 08691 08692 08713
08762 09169 09222 09226 09233 09252 09269 09296
09314 09436 09769 09782 09803 09912
SURGERY,ENTEROCOLITIS
09993
SURGERY,ESOPHAGUS
00262* 00265* 00270 00273 00274 00277 00285 00286
00473 00673 01059 01085 01135* 01136* 01137* 01140*
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01159 01160 01162 01163 02008* 02011* 02012* 02015*
02016* 02017 02018 02028 02029 02032 02034 02037
02039 02042 03009 03069* 03070* 03073* 03084 03086
03087 03093 03094 03096 03205 04135 04136 04137
04138 04139 04140 04141 04142 04144 04146 04147
04149 04151 04154 04156 04157 04160 04161 04162
04163 04164 04165 04975* 05082* 05084* 05088* 05089
05090 05091 05092 05093 05098 05107 05108 05110
05112 05113 05114 05115 05116 05117 05118 05137*
05164 06112 06115 06117 06122 06123 06124 06126
06127 06130 06131 06132 06134 06135 06137 06696
06942 06945 06969* 06970* 06972* 06974* 06976* 06977
06978 06981 06983 06986 06988 06990 06993 06995
06996 06997 06998 06999 07000 07003 07985* 07986*
07988* 07989* 07991* 07993* 07995* 07996* 07997 08000
08003 08005 08006 08009 08014 08015 08019 08023
08028 08029 08030 08032 08056 08079 08118* 08816
09130 09131 09133 09134 09135 09136 09138 09139
09141 09142 09145 09146 09150 09151 09152 09153
09154 09155 09157 09158 09161 09162 09164 09165
09167 09591 09811
SURGERY,GALLBLADDER
00765 00766 01651* 01658* 01661* 01676 02668* 02675
02919* 03332* 03649 04628* 04632* 04638 04653 05634*
05642* 05649* 05653 05663 05665 06008* 06217* 06426*
06532* 06621* 06628* 06629* 06641 06642 06647 06650
06656 06659 06663 06879* 07491* 07493* 07497* 07506
07510 07522 07528 07531 07532 07878 08058 08118*
08469 08655* 08663* 08668 08668 08671 08672 08681
08686 08692 08702 08705 08707 08709 08714 09560
09745 09754 09757 09762 09770 09776 09778 09781
09783 09787 09788 09792 09806
SURGERY,GASTROINTESTINAL
SEE ALSO GASTROINTESTINAL TREATMENT
SEE ALSO ROUX-EN-Y GASTROENTEROSTOMY
00143 00184* 00199* 00221 00261 00262* 00286 00294*
00338 00348* 00363 00381* 00440* 00800 00939* 00973
01023* 01028* 01029* 01033* 01045* 01049 01056 01057
01059 01084 01085 01086 01087 01096 01342 01401*
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02083 02152 02210* 02349 02743 02924 02974* 03010
03013 03018 03027 03040 03345 04027* 04032* 04043*
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04113 04241 04365 04446* 04972* 04986* 04993 04995
04996 04999 05000 05009 05027 05029 05035 05050
05150* 05165 05321 05873* 05983* 06007* 06024 06027
06029 06032 06034 06056 06059 06077 06078 06689
06964 07093 07505 07552 08035* 08105 08215 08401
08717* 08763 08773 08782 08785 08815 08823 08825
08828 08832 08842 08852 08854 08858 08878 09161
09490 09754 09774 09802 09834 09854 09900 09991
SURGERY,LARGE INTESTINE
00207 00349* 00441* 00445* 00446* 00449* 00455* 00465
00478 00479 00480 00481 00483 00485 00486 00487
00488 00491 00492 00493 00494 00495 00500 00512
00520 00523 00939* 01047* 01058 01091 01379* 01381*
01387* 01389* 01392* 01394* 01396* 01397* 01400* 01403*
01411 01422 01434 01437 01449 01450 01475 01940*
02276* 02300* 02301* 02303* 02306* 02307* 02308* 02309*
02310* 02311* 02313* 02314* 02316* 02318* 02321* 02322*
02326* 02329* 02330* 02331* 02332* 02334 02340 02343
02347 02362 02374 02387* 02390* 02395* 02399 02401
02473* 02637* 02996* 03026 03091 03155 03251* 03252*
03253* 03258* 03261* 03264* 03276 03278 03280 03282
03289 03297* 03306 03955 04095 04096 04327* 04332*
04336* 04337* 04339 04340 04341 04342 04344 04346
04347 04348 04349 04350 04351 04354 04355 04358
04360 04366 04370* 04371* 04928 04975* 05030 05310*
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05339 05340 05343 05345 05347 05351 06049 06061
06067 06137 06318 06327* 06328* 06329* 06331* 06337
06338 06339 06340 06343 06344 06345 06346 06348
06352 06354 06355 06357 06363 06365 06366 06367
06368 06369 06371 06372 06374 06385 06393 06605
06882* 07078* 07098 07099 07183* 07205* 07208* 07209*
07211* 07212* 07217* 07218 07219 07220 07227 07231
07232 07234 07238 07239 07240 07241 07242 07244
07246 07247 07248 07250 07251 07252 07256 07259
07267 07273 07565 07817* 08030 08087 08144 08163
08249* 08251* 08252* 08254* 08255* 08265* 08269* 08271
08272 08274 08277 08278 08281 08282 08284 08291
08292 08296 08301 08307 08308 08311 08312 08313
08314 08315 08316 08317 08319 08323 08327 08332
08335 08337 08341 08353 08355 08357 08361 08362
08369 08370 08371 08717* 08743* 08784 08851 08901

RY,LARGE INTESTINE
TINUED
916 09146 09151 09158 09187 09344 09348 09349
350 09355 09358 09361 09363 09367 09368 09369
370 09374 09375 09378 09379 09382 09383 09384
385 09389 09395 09398 09409 09413 09416 09417
418 09421 09427 09428 09764 09847 09857 09868
888 09893 09901 09926 09930 09985
RY,LIVER
087* 00649 00663 00673 00735 00736 01049 01059
531* 01532* 01546* 01548* 01549* 01550* 01551* 01565
569 01570 01593 01600* 01602 01607 01652* 01844
478* 02480* 02484* 02485* 02493 02507 02515 02517
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423 03426 03457 03465 03467 03475 03593* 04435*
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503 04551 04616* 05424* 05427* 05428* 05443* 05469
485 05490 05499 05502 05503 05510 05517 05525
564 05565 05620 05859* 06447* 06464* 06478 06516
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395 07400 07410 07414 07435 07470* 07550* 07601
730* 07731* 08434* 08435* 08437* 08441* 08457 08465
480 08481 08488 08491 08510 08512 08514 08517
519 08520 08529 08533 08535 08536 08566 08567
568 08590 09087 09489 09506 09509 09511 09513
516 09526 09554 09557 09561 09562 09569 09622
627 09632 09659 09778 09781 09990
RY,MESENTERY
219 01246* 01276 01394* 01927* 06066 08878 09815
822 09875 09888
RY,PANCREAS
161 00550 00551 00558 00560 00562 00563 00564
571 00573 01021* 01036* 01049 01059 01092 01134
170* 01268 01489* 01490* 01496 01501 01502 01505
509* 01526 01942 01952 02412* 02415 02425 02433
442* 02457 02468 02781* 03004 03310* 03322 03324
326 03328 03332* 03333* 03352 04037* 04039* 04308*
386* 04387* 04388* 04391* 04393 04395 04396 04398
400 04401 04405 04409* 04414 04420 04687* 04722*
791* 05014 05037 05039 05382* 05384* 05389 05393
394 05396 05398 05399 05402* 06398* 06403 06404
406 06408 06410 06411 06413 06416 06426* 06439
444 06642 07275* 07277* 07290 07291 07293 07294
295 07297 07302* 07325 07326 07328 07550* 07583
694 07868* 08045* 08376 08377 08380 08381 08392
394 08403 08404 08412 08413 08421 08678 08748
779 08856 09073 09431 09433 09434 09436 09438
439 09440 09441 09442 09443 09444 09445 09448
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898
RY,PANCREATITIS
524 00562 00581 00588 00591 01510* 01519 01658*
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390* 04409* 04413* 04419 04420 05398 05414 05421
638* 06422* 06439 07291 07298* 07299* 07300* 07305*
314 07327 07328 08397* 08403 08413 08418 08419
470 09471 09482
RY,PEPTIC ULCER
109* 00297* 00307* 00311* 00312* 00377* 00379* 00381*
387* 00388* 00395* 00400 00405 00406 00411 00855
934* 00970 01004 01028* 01036* 01097 01126 01166*
173* 01178* 01183* 01190 01191 01192 01193 01195
200 01206 01211 01214 01260* 01278 01298* 01299*
302* 01303* 01304* 01305* 01306* 01307* 01308* 01321*
322* 01324 01325 01326 01327 01328 01329 01330
331 01333 01334 01336 01337 01338 01339 01340
343 01345 01347 01348 01349 01351 01352 01353
355 01358 01359 01362 01363 01673 01749* 01859*
956 02011* 02195* 02199* 02203* 02209* 02215* 02226
228 02231 02235 02236 02237 02259 02264 02270
663* 02780* 02793* 02909* 03009 03117* 03182* 03183*
184* 03185* 03186* 03187* 03188* 03189* 03192* 03196
197 03201 03205 03206 03207 03212 03221 03222
223 03224 03776 04040* 04051* 04085 04086 04158
166* 04178* 04181* 04194 04198 04201 04208 04210
256* 04260* 04261* 04262* 04263* 04264* 04265* 04266*
269* 04274 04275 04281 04282 04283 04292 04293
296 04297 04299 04742* 04743* 04748* 04758 04773*
031 05034 05037 05053 05130* 05152 05153 05216*
217* 05220* 05221* 05222* 05225* 05227* 05229* 05231*
233* 05234 05236 05237 05240 05244 05245 05246
247 05248 05249 05251 05253 05255 05257 05258
259 05261 05262 05265 05267 05273 05695* 05758*
840* 06018 06153* 06157* 06168 06186 06239* 06241*
246* 06248* 06251* 06254* 06263* 06264* 06265 06266
269 06270 06271 06272 06275 06277 06278 06279
280 06283 06285 06286 06288 06289 06293 06295
296 06297 06299 06300 06303 06304 06305 06306
308 06309 06310 06311 06312 06726* 06883* 06950
010* 07026* 07062 07115 07128* 07129* 07132* 07138*
139 07140 07145 07146 07147 07149 07151 07153
155 07163 07164 07166 07168 07177 07178 07329
849* 07885 08050 08082 08141 08165* 08169* 08170*
172* 08173* 08174* 08177* 08180 08181 08182 08183
184 08185 08186 08187 08188 08192 08193 08194
SURGERY,PEPTIC ULCER
CONTINUED
08197 08201 08202 8203 08205 08209 08210 08211
08214 08215 08694 08717* 08729* 08730* 08741* 08769
08779 09076 09093 09093 09200 09247 09248 09249
09256 09258 09259 09261 09266 09267 09268 09269
09271 09278 09285 09289 09291 09293 09300 09301
09304 09306 09308 09309 09313 09314 09318 09865
SURGERY,RECTUM
00440* 00443* 00444* 00446* 00447* 00466 01058 01385*
01398* 01409* 01419 01425 01438 01448 01456 02304*
02306* 02309* 02310* 02316* 02367 02380 03259* 03282
04332* 04355 04371* 04975* 05312* 05314* 05322 05327
05338 05339 05340 05348 05351 06327* 06328* 06331*
06338 06341 06353 06364 06367 06369 06370 06680*
06882* 07206* 07242 07243 07257 08273 08274 08275
08279 08296 08297 08310 08315 08327 08345 08351
08352 08362 08370 09199 09341 09350 09361 09372
09376 09386 09392 09401
SURGERY,SALIVARY GLAND
00185* 02910* 09831 09931
SURGERY,SMALL INTESTINE
00201 00202 00207 00351* 00352* 00358 00360 00365
00366 00368 00424 00434* 00524 00563 00573 00780
00787* 01038* 01058 01091 01134 01248* 01249* 01254*
01258* 01268 01271 01276 01277 01278 01281 01284
01293 01294 01307* 01369 01526 01652* 01975 02115*
02119* 02123* 02127* 02133 02141 02144 02146 02157
02277 02290 02399 02665* 02780* 02802* 02860 02893*
02914* 02974* 02996* 03036 03066 03091 03149* 03150*
03162 03166 03171 03172 03174 03176 03178 03185*
03186* 03194* 03205 03227* 03230* 03271 03328 03333*
03339* 03633* 03756* 03768* 03800 03955 04039* 04051*
04054 04095 04096 04124 04232* 04240 04242 04243
04247 04249 04296 04513 04666 04849* 04928 04975*
04977* 05014 05039 05137* 05186* 05187* 05189 05190
05193 05195 05197 05199 05200 05204 05206 05208
05209 05210 05211 05212 05214* 05279 05280 05283
05389 06045 06152* 06219* 06227 06231 06319 06415
06477 06635 06836* 06977 07042 07077* 07078* 07081*
07083* 07087* 07088 07091 07098 07099 07102 07103
07104 07107 07111 07113 07114 07119 07124 07129*
07181* 07183* 07184 07193* 07508 07520 07565 07657*
08003 08030 08056 08074 08077 08101 08112* 08114*
08115* 08117* 08118* 08122 08125 08126 08127 08132
08133 08141 08143 08144 08150 08157 08159 08163
08182 08201 08212 08213 08221 08230* 08346 08362
08403 08469 08692 08717* 08743* 08776 08784 08835
08901 08916 09084 09151 09177 09182 09232 09237
09241 09247 09261 09434 09746 09755 09761 09847
09868 09875 09893 09912 09930
SURGERY,STOMACH
SEE ALSO GASTRECTOMY
00181* 00261 00288 00297* 00309* 00313* 00319 00319
00324 00337 00363 00379* 00388* 00557 00673 00807
00835* 00970 01038* 01047* 01170* 01171* 01174* 01178*
01182* 01183* 01184* 01186 01187 01193 01210 01211
01213 01215 01218 01219 01222 01225 01227 01233
01298* 01299* 01302* 01304* 01305* 01306* 01307* 01322*
01355 01358 01359 01361 01502 01788 01926* 01946
01952 01996 02011* 02018 02044* 02065 02066 02067
02071 02082 02083 02084 02091 02101 02105 02105
02106 02106 02110 02112 02150 02194* 02199* 02209*
02215* 02221 02235 02236 02237 02247 02250 02250
02259 02261 02407* 02442* 02541 02612 02663* 02780*
02803* 02899* 03004 03009 03012 03058 03101* 03102*
03107* 03111* 03113* 03128 03130 03134 03142 03158
03183* 03185* 03186* 03187* 03188* 03189* 03192* 03194*
03196 03197 03211 03239* 03271 03760* 03776 04040*
04051* 04054 04071 04085 04123 04159 04176* 04177*
04181* 04186 04191 04194 04198 04199 04201 04202
04207 04211 04260* 04261* 04262* 04263* 04264* 04265*
04266* 04269* 04274 04282 04292 04293 04296 04297
04730* 04748* 04773* 04869 04975* 05072 05112 05132*
05135* 05152 05153 05158 05163 05169 05170 05171
05172 05174 05177 05214* 05216* 05217* 05221* 05222*
05235 05236 05237 05240 05244 05245 05247 05253
05256 05262 05756* 05758* 05903 06045 06061 06109*
06143* 06147* 06152* 06157* 06165 06175 06178 06181
06184 06185 06187 06189 06195 06196 06201 06207
06217* 06239* 06254* 06259* 06263* 06264* 06266 06270
06275 06277 06278 06279 06283 06285 06301 06306
06308 06309 06310 06532* 06726* 06873* 06883* 06978
06988 07014* 07024* 07027* 07032 07043 07051 07052
07057 07066 07070 07071 07128* 07138* 07139 07146
07147 07153 07166 07168 07177 07178 07249 07275*
07329 07552 07677* 07680* 07871 07916 07984* 07990*
07995* 07997 07998 08001 08015 08016 08031 08036*
08037* 08046 08048 08049 08050 08051 08053 08055
08057 08060 08061 08062 08064 08065 08074 08084
08094 08097 08098 08099 08101 08102 08104 08105
08108 08111 08118* 08165* 08174* 08177* 08186 08187
08192 08196 08197 08201 08203 08210 08213 08214
08443* 08471 08712 08717* 08762 08779 08793 08816
08835 08845 08859 08950 09040 09098 09155 09157
09162 09169 09170 09182 09186 09187 09196 09201

SURGERY,STOMACH
CONTINUED
09202 09203 09205 09207 09208 09215 09248 09252
09256 09261 09272 09274 09278 09284 09289 09296
09301 09304 09306 09309 09441 09484 09781 09811
09865 09912
SURGERY,ULCER
00181* 01311* 01365 01962 02255 02259 02261 02266
08216 09253 09255 09281 09373
SURGERY,ULCERATIVE COLITIS
01379* 01468* 01471* 01472* 01475 01480 01482 02387*
02396* 02397 02399 02401 03297* 04370* 04371* 04375*
04379 05343 05374 05377 05379 06384 06385 06388
06393 06395 07267 07273 08254* 08361 08362 08371
09421
SURGERY,VASCULAR
00221 05065 06594 07353 07359 07473 07554 08154
08180 08496 09060 09517 09523
SURGERY,VOLVULUS
04653 06343 07074 07103 07241 08264* 08357 09383
SURGICAL APPARATUS
00143 00161 00776 00777 00962 01133 02974* 05162
05165 05170 05983* 06447* 07077* 07298* 07990* 08034*
SURGICAL APPARATUS AND TECHNIQUES
02974* 03143 03178 03185* 03222 03289 03306 03308
03328 03379* 03465 03625* 03627*
SURGICAL TECHNIQUES,EXPERIMENTAL
02152 02317* 02429 02573 04078 04163 04242 04461
04477 04623 04631* 04640 04742* 04743* 05084* 05424*
05458 06219* 06327* 06413 06447* 06618* 06679* 07024*
07087* 07331* 07353 07403 07500* 07677* 07730* 07988*
08006 08009 08034* 08114* 08489 08658* 09350
SURGICAL TREATMENT OF BLEEDING
00217 00264* 00395* 00607* 00656 01032* 01089 01218
01272 01363 01600* 01642* 01926* 01956 02045* 02199*
02205* 02217* 02263 02318* 03009 03044 03046 03102*
03149* 03206 03355* 03949 04177* 04212 04220 04262*
04269* 04273 04285 04292 04298 04342 04371* 04490
04598* 04599* 04606* 04997 05011 05038 05042 05043
05100 05221* 05231* 05249 05256 05265 05267 05981*
06022 06064 06096 06124 06254* 06274 06283 06578*
06579* 06583* 06587 06596 06597 06601 06970* 06973*
06995 07390 07406 07429 07465* 07468* 07470* 07473
07483 07486 07562 07871 07984* 07994* 07996* 08109
08173* 08193 08482 08632* 08813 08816 08842 08845
08873 09139 09156 09168 09285 09288 09296 09304
09497 09539 09613 09703 09927
SURVIVAL RATES,CANCER
01936* 02058* 02097 02482* 03072* 03075* 03281 04332*
04333* 05081* 05087* 05106 05139* 05313* 05384* 05387*
05640* 06108* 06398* 06464* 06972* 07007* 07026* 07029*
07052 07086* 07120 07207* 07209* 07338* 08037* 08039*
08064 08071 08086 08090 08260* 08261* 08262* 08265*
08296 08310 08314 08320 08325 09173 09353 09364
09770
SYNDROME,BLIND LOOP
08145
SYNDROME,BUDD-CHIARI
00607* 00610 00645 01538* 01558 01885 01930* 05848*
06489 06842* 08454 08530 09544 09545 09585 09733
SYNDROME,CARCINOID
SEE ALSO CARCINOIDS
SEE ALSO SEROTONIN
00089* 00370 00541* 00570 01071 02057* 02368 02977*
03006 03015 03067 03121 03227* 04117 04246 05017
05023 05314* 06078 06368 07258 08266* 08285 08498
08877 09446 09828 09832 09892
SYNDROME,CRIGLER-NAJJAR
03390 09620
SYNDROME,CUSHINGS
00541*
SYNDROME,DUBIN-JOHNSON
01665* 03369* 08478 09492 09584
SYNDROME,DUMPING
01184* 08174*
SYNDROME,GARDNERS
00519 01940* 05346 09419
SYNDROME,GASTRECTOMY-INDUCED AFFERENT LOOP
00326 03142 04201 04204 04224 04299 05150* 06266
07036 07042 08049 08058 08185 08195
SYNDROME,GASTRECTOMY-INDUCED DUMPING
00186* 00302* 00303* 00314 00787 00791 01100 01211
01322 01331 02011* 02062 02106 03009 03100* 03142
03188* 03189* 03194* 04189 04204 04285 04296 04299
05198 05885* 06147* 06152* 06157* 06173 06231 06266
06277 07036 07060 07062 07129* 07132* 07177 08035*
08049 08058 08062 08182 08185 09093 09175 09196
09271 09300 09314
SYNDROME,MALLORY-WEISS
01108 01154 01959 05042 05109 08018 08845 09139
SYNDROME,PEUTZ-JEGHERS
00364 01256* 02133 02305* 02343 04082 04334* 04340
05194 08123 08140 08147 08337
SYNDROME,PLUMMER-VINSON
09160
SYNDROME,SJOGRENS
00540* 09446
SYNDROME,ZIEVES
00648 00738 02470* 04458* 05432* 06501 07480 08411
SYNDROME,ZOLLINGER-ELLISON
00188* 00195* 00541* 00815* 00970 01021* 01049 01051
01090 01092 01121 01485* 01487* 01890 01899 02053*
02235 02237 02290 02988* 02993* 03004 03052 03068
03784 03918* 04037* 04039* 04048* 04049* 04167* 04290
04755 04855* 05018 05030 05031 05034 05037 05053
05071 05075 05076 05078 05764* 05928 06000* 06014
06016 06018 06251* 06902 07036 07160 07275* 07297
07581 07582 07583 07584 07835 07862* 07954 08720*
08730* 08768 08769 08779 08780 08824 08826 08856
08871 09300 09809 09824 09865

THERAPY,DIETETIC
00396* 00425* 00429* 00978 00984 01026* 01250* 01638*
01857* 01926* 02197* 02205* 02262 02286* 02395* 03097*
03330* 03538 03556 03614 04015 04074 04273 04281
04288 04314 04379 04494 04509 04517* 04584 04586
04595* 04605* 04648 04939 05238 05284* 05286* 05308
05461 05463 05598* 05613 05632* 05986* 05997* 06007*
06211 06216* 06236* 06276 06476 06491 06567 06576
06613 06641 07094 07194* 07195 07494* 07502* 07523
07535 07563 07823 08222 08245 08607 08820 08891
09260 09266 09278 09320 09326 09328 09816 09880
09997
THROMBOSIS
02464 03663 04096 04118 04374* 04490 06527 06815
07254 07375 07565 07879 07922 08118* 08321 08380
08402 08489 08501 08840 08850 08853 09059 09493
09565 09707 09730 09790
THROMBOSIS,HEPATIC VEIN
SEE BUDD-CHIARI SYNDROME
THYROID GLAND
01963 02458 05815* 06003* 06176 06742* 06786* 06809
07063 07281 07720* 08475 08644 08877 09195 09209
09735
THYROID GLAND DISEASE
00189*
TOLERANCE TESTS
04730* 04837* 04841* 05985* 05986* 06004* 07277* 08225*
TOLERANCE TESTS IN MALABSORPTION DIAGNOSIS
00129* 00153 00426* 00428* 00435* 02281* 02913* 03230*
04000 04314 04442* 04880 06836* 08247 09323
TOXIC EFFECTS OF ALCOHOL
09731
TOXIC EFFECTS OF RADIATION
SEE ALSO TOXIC EFFECTS ON ABDOMEN
SEE ALSO TOXIC EFFECTS ON ABSORPTION
SEE ALSO TOXIC EFFECTS ON BILIARY TRACT
SEE ALSO TOXIC EFFECTS ON ESOPHAGUS
SEE ALSO TOXIC EFFECTS ON GASTROINTESTINAL TRACT
SEE ALSO TOXIC EFFECTS ON LARGE INTESTINE
SEE ALSO TOXIC EFFECTS ON LIVER
SEE ALSO TOXIC EFFECTS ON PANCREAS
SEE ALSO TOXIC EFFECTS ON RECTUM
SEE ALSO TOXIC EFFECTS ON SALIVARY GLANDS
SEE ALSO TOXIC EFFECTS ON SMALL INTESTINE
SEE ALSO TOXIC EFFECTS ON STOMACH
00016 00810 00906 01054 01181* 01287 01404* 01718
01737* 01833* 01935* 02134 02158 02159 02160 02161
02162 02163 02166 02167 02168 02171 02172 02173
02174 02175 02176 02177 02178 02179 02240 02297
02417 02430 02549 02718 02719 02720 02723 02724
02725 02726 02727 02744 02835 02836 02983* 02985*
03031 03032 03033 03034 03368* 03443 03674* 03734*
03844 04253 04519* 04544 04735 04736 04987* 05083*
05188* 05465 05552* 05561 05563 05568 05958 06010
06047 06144* 06215* 06336 07002 07080* 07419* 07436
07565 07736* 07814* 07819 07882 08256* 08302 08454
08559* 08577 08722* 08870 08968 09225 09932
TOXIC EFFECTS ON ABDOMEN
01054 02317* 02720 04056
TOXIC EFFECTS ON ABSORPTION
02171 02172 02297 04453*
TOXIC EFFECTS ON BILIARY TRACT
04679* 07514 07664
TOXIC EFFECTS ON ESOPHAGUS
00266* 00287 03091 05083* 05112 06043 06724* 07999
08011
TOXIC EFFECTS ON GALLBLADDER
07514
TOXIC EFFECTS ON GASTROINTESTINAL TRACT
00182* 00218 01113 02158 02159 02160 02161 02162
02163 02176 02177 02718 02719 02720 02724 02725
02726 02744 02747 03031 03032 03033 03034 05728
06039 06047 07542* 07664 07681* 07831 08722* 08740*
08805 09070 09198 09204 09394 09400 09851 09879
09894 09910

C EFFECTS ON LARGE INTESTINE
0471 01257* 01469* 02747 02983* 03263* 04378 04735
5058 05317* 05318* 06010 06336 07210* 07216* 08256*
3259* 08302 09040
C EFFECTS ON LIVER
0010 00014* 00603* 00606* 00629 00631 00654 00683*
0684* 00685* 00686* 00688 00692 00693 00694 00702
0715 00906 00927* 00934* 01079 01179* 01539* 01540*
1541* 01556 01558 01559 01588 01601 01603 01604
1605 01606 01608 01633* 01641* 01720* 01833* 01863*
2492 02498 02545 02549 02576* 02578* 02580* 02594
2595 02596 02599* 02639 02875 02877 03368* 03374*
3383* 03389 03432 03433 03435 03440 03443 03460
3475 03479* 03480* 03482* 03483* 03484* 03489 03490
3491 03492 03496 03497 03498 03499 03501 03505
3507 03511 03512 03513 03514 03516 03521* 03549
3585* 03599* 03602 03653 03716 03719 03813* 03814*
3815* 03821* 03834 03844 03855 03874 03954 04009
4012 04094 04446* 04469 04473 04486 04506 04514
4515 04518* 04519* 04520* 04521* 04522* 04523* 04524*
4527* 04528* 04529* 04531 04532 04533 04534 04535
4536 04537 04538 04539 04540 04541 04542 04543
4544 04545 04546 04547 04548 04549 04550 04552
4553 04554 04555 04556 04582 04616 04633* 04705
4719* 04794* 04796* 04834 04845* 04848* 04892 04954
5077 05437* 05442* 05447* 05449* 05460 05465 05481
5500 05527* 05528* 05532* 05533* 05534* 05537* 05538*
5539* 05540* 05541* 05542* 05543* 05544* 05545* 05546*
5547* 05548* 05549* 05550* 05552* 05553* 05554 05555
5557 05558 05561 05562 05563 05580 05596* 05704
5812* 05813* 05820* 05824 05827 06247* 06459* 06460*
6471 06489 06490 06520 06521 06522 06523 06525
6533* 06534* 06535* 06536* 06538* 06540 06541 06542
6543 06544 06546 06548 06549 06550 06589 06692
6781* 06784* 06787* 06792* 06797* 06803 06804 06811
6868* 06884 06933 07187* 07380 07385 07419* 07420*
7421* 07423* 07424 07426 07428 07430 07431 07432
7433 07434 07437 07438 07440 07441 07587 07681*
7706* 07716* 07721* 07722* 07725* 07736* 07737* 07748*
7749* 07752* 07755 07758 07771 07804 07807 07808
7864* 07870 08450 08454 08492 08539* 08540* 08544*
8547* 08550* 08552* 08553* 08554* 08555* 08556* 08559*
8560* 08561* 08563 08564 08574 08575 08576 08577
8579 08587 08588 08591 08616 08654 08956 08958
8975 08976 08977 08979 08987 09000 09004 09102
9533 09536 09537 09543 09588 09589 09601 09624
9626 09628 09641 09642 09643 09646 09650 09654
9660 09662 09704 09731 09972
C EFFECTS ON PANCREAS
0579* 00589 01500 01753* 02417 02430 03315* 04383*
4424 04669* 04684* 04687* 06401* 06771*
C EFFECTS ON RECTUM
1404* 05058
C EFFECTS ON SALIVARY GLANDS
0801 00802
C EFFECTS ON SMALL INTESTINE
0016 00022* 00372 01079 01257* 01287 01718 01734*
1935* 01937* 02134 02165 02166 02167 02168 02173
2174 02175 02179 02317* 02747 02983* 03674* 03717
4245 04253 04323 04714* 04735 04781* 05180* 05188*
5202 05715* 06010 06215* 06703* 06727 07080* 07082*
7085* 07187* 07189* 07639* 07812* 07814* 07819 08134
8135 08559* 09225
C EFFECTS ON STOMACH
0013* 00289* 00305* 00819* 01030* 01231 01762* 02069
2216* 02240 02723 02747 03114* 04168* 05112 06043
6144* 06158* 06159* 06205 06672* 07040 07547* 08033*
9179 09181 09204 09246 09282
C HEPATITIS
ALSO TOXIC EFFECTS ON LIVER
0927*
NS OF MICROORGANISMS,METABOLIC
4108 05185* 06214* 06533* 06803 06804 06831 07082*
7542* 07748* 07755 08547* 08734*
NS,BACTERIAL
5532*
NS,CHEMICAL
ALSO TOXIC EFFECTS ON ABDOMEN
ALSO TOXIC EFFECTS ON BILIARY TRACT
ALSO TOXIC EFFECTS ON ESOPHAGUS
ALSO TOXIC EFFECTS ON GASTROINTESTINAL TRACT
ALSO TOXIC EFFECTS ON LARGE INTESTINE
ALSO TOXIC EFFECTS ON LIVER
ALSO TOXIC EFFECTS ON PANCREAS
ALSO TOXIC EFFECTS ON RECTUM
ALSO TOXIC EFFECTS ON SALIVARY GLANDS
ALSO TOXIC EFFECTS ON SMALL INTESTINE
ALSO TOXIC EFFECTS ON STOMACH
00010 00014* 00022* 00176 00287 00289* 00628 00629
00683* 00685* 00692 00693 00694 01257* 01469* 01601
01633* 01734* 02069 02545 02580* 02594 02595 02596
02747 03479* 03501 03513 03514 03516 03874 04633*
04731* 05442* 05533* 05534* 05541* 05542* 05544* 05549*
05553* 05555 05820* 06205 06536* 06549 07424 07431
07432 07514 07587 07706* 07716* 08134 08135 08560*
08563 08956 08975 08976 08987 09536 09537 09543
TOXINS,CHEMICAL
CONTINUED
09650 09662
TOXINS,METABOLIC
02637* 03668* 04678 05077
TRACE ELEMENT AND MINERAL ABSORPTION
00019 00022* 00026* 00030 00031 00032 00039 00311*
00396* 00425* 00439 00773 00893* 00894* 00978 01037*
01192 01315* 01530* 01640* 01719* 01720* 01726* 01728*
01730* 01732* 01737* 01739 01744 02260 02291 02629*
02738 02750* 02751* 02752* 02753* 02754* 02756* 02802*
02920* 03017 03077* 03234* 03240* 03241* 03301* 03500
03588* 03592* 03679 03723* 03724* 03726* 03733* 03734*
03736 03739 03742 03986 04219 04240 04309* 04310*
04384* 04436* 04602* 04717* 04719* 04724* 04725* 04726*
04727* 04731* 04732 04733 04950 05076 05127* 05207
05289* 05291* 05444* 05629 05729 05733 05846* 06029
06704* 06709 06712 06713 06714 06715 06716 07197
07632* 07641* 07642* 07645 08036* 08110 08236 08525
08531 08937 08942 08951 08988 09106 09330 09534
09709 09845
TRACE ELEMENT AND MINERAL DEFICIENCIES
01829* 05076 05289* 06441
TRACE ELEMENT AND MINERAL EXCRETION
03676* 03915*
TRACE ELEMENT AND MINERAL METABOLISM
05233* 05289* 05456* 05681* 06307 07281 07467* 07787
08577 09611 09861
TRACE ELEMENT METABOLISM,LIVER
00756* 01541* 01640* 01842 02474* 02630* 03597* 03812*
03814* 03855 03857 04447* 04516* 04633* 04915 05426*
05453* 06451* 06515 06595 06816 07397 07477 07893
08425* 09491 09572
TRACER STUDIES
00013* 00022* 00024* 00026* 00028* 00033 00063* 00064*
00077* 00103* 00113* 00114* 00115* 00137 00148 00157
00182* 00184* 00303* 00345* 00539* 00611 00696* 00730*
00808 00810 00812* 00830 00893* 00894* 00897 00903
00925* 00941* 00945* 00951* 00956 00961 00963 00983
01010 01261* 01529* 01640* 01726* 01730* 01745 01811*
01826* 01892 01897 01906 01916 01937* 01979 02121*
02122* 02127* 02164 02285* 02320* 02427 02519 02605*
02651 02763* 02888* 02892* 02903* 02922 02968 03099*
03526* 03585* 03586* 03592* 03594* 03601* 03676* 03710*
03823* 03915* 03924* 04695* 04706 04718* 04724* 04725*
04726* 04727* 04728* 04729* 04731* 04808* 04809* 04824
04825 04827 04828 04829 04836 04838* 04839* 04840*
04854* 04903 04932 04942 04947 04948 04949 04950
04951 04952 04953 04954 04955 04956 04957 04983*
05074 05300 05302 05362* 05454* 05457* 05505 05537*
05552* 05554 05601* 05612* 05667* 05668* 05704 05710*
05713* 05717* 05720* 05721* 05729 05804* 05818* 05833
05837* 05843* 05855* 05856* 05857* 05861* 05876* 05878*
05882* 05910 05913 05916 05917 05918 05921 05925
05927 05944 05965 05974 05976 05977 06044 06315*
06332* 06379* 06452* 06484 06535* 06539 06581* 06677*
06698* 06704* 06705* 06708* 06709 06711 06715 06735*
06744* 06779* 06783* 06785* 06793* 06798* 06821 06842*
06844* 06873* 06875* 06876* 06903 06913 06938 06940
06961 06964 07083* 07085* 07188* 07536 07583 07594*
07622* 07629* 07633* 07634* 07635* 07641* 07642* 07643*
07644* 07655 07666* 07667* 07669 07670 07672 07697*
07699* 07703* 07704* 07706* 07711* 07715* 07716* 07718*
07727* 07728* 07729* 07733* 07735* 07741* 07749* 07759
07765 07767 07774 07776 07777 07779 07784 07786
07787 07792 07799 07800 07811* 07814* 07815* 07816*
07821 07828 07848* 07854* 07857* 07890 07892 07907
07908 07915 07919 07925 07928 07931 07943 07947
07948 07949 07950 07955 07967 08077 08228* 08230*
08236 08238 08246 08493 08525 08581 08648 08649
08760 08905 08923 08926 08937 08940 08942 08947
08969 08981 08997 09036 09039 09043 09058 09080
09109 09429 09494 09593 09775
TRANSPLANTATION
SEE ALSO IMMUNOLOGY
02028 02083 05051
TRANSPLANTATION,BILIARY TRACT
01792 07500*
TRANSPLANTATION,ESOPHAGUS
06993 09150
TRANSPLANTATION,GASTROINTESTINAL
09835
TRANSPLANTATION,LARGE INTESTINE
00473 01144 01147 01151 02016* 02042 04138 04141
04156 05051 06131 06137 06990 07989* 08815 09133
09151 09158 09164
TRANSPLANTATION,LIVER
00087* 00647 00663 00809* 01546* 01550* 01551* 01565
01570 01593 01705 02475* 02517 02518 02520 02527
02558 02573 02575 02735* 02844 03360* 03382* 03413
03428 03852 03924* 04433* 04435* 04440* 04448* 04459*
04477 04481 04500 05424* 05428* 05429* 05445* 05458
05470 05510 05536* 06514 06531* 06798* 07336* 07384
07550* 07738* 07750* 08423* 08435* 08439* 08445 08481
08488 08489 08504 09518 09526 09557 09828

TRANSPLANTATION,PANCREAS
00571 01505 01705 01990 02409* 02429 03310* 03898*
04388* 05051 05388 06399* 06413 07277* 07280* 07550*
TRANSPLANTATION,SMALL INTESTINE
00787* 01151 01273 01276 01705 01990 02116* 02127*
04203 05051 05084* 06123 06131 06977 06997 07024*
07078* 07277* 08003 08815 08867 09151 09164 09350
TRANSPLANTATION,STOMACH
01136* 01314* 01705 04147 04192 05051 05136* 06122
06131 06990 07677*
TRANSPORT,AMINO ACID
07649 09967 09970
TRANSPORT,ION
00021* 00031 00305* 00814* 01733* 01734* 01744 01847
02126* 02320* 02325* 02768 02769 02770 02775 02776
02777 03041 03042 03260* 03367* 03600* 03669* 03674*
03722* 03736 03738 03740 03742 03745 03747* 03751*
03759* 03814* 03913* 03955 04023* 04070 04180* 04331*
04453* 04674* 04703 04711* 04713* 04716* 04835 04863*
04981* 05368 05410 05445* 05682* 05724* 05726 06701*
06708* 06743* 06784* 06791* 06813 07009* 07065 07543*
07575 07623* 07624* 07627* 07630* 07631* 07633* 07636*
07637* 07638* 07641* 07642* 07688 07765 07784 08055
08231* 08372* 08516 08874 08939 09022 09464 09737
TRAUMA IN CHILDREN,ABDOMEN
01490* 01602 02996*
TRAUMA,ABDOMEN
00234 00327 00475 00652 01038* 01039* 01057 01207
01435 01598* 02005 02915* 03132 04120 04225 04401
04498 05068 05069 05096 05399 05871* 06050 06999
07095 07108 07113 07118 07293 07435 07918 08002
08026 08126 08752 08822 08907 09039 09066 09226
09404 09433 09638 09856 09925 09987
TRAUMA,BILIARY TRACT
00652 00961 01944 03654 04643 04644 04645 04662
05490 05642* 06629* 06665 07389 08567 08692 09630
09638 09856
TRAUMA,DUODENUM
07017* 07108 07118
TRAUMA,ESOPHAGUS
00274 00285 01108 01146 02023 02023 03092 04133
04158 05093 05108 05112 05113 06137 06985 07993*
09145
TRAUMA,GALLBLADDER
00769 01683 03910* 06629* 09807
TRAUMA,GASTROINTESTINAL
00573 00976 01927* 01944 02005 03345 04073 04113
06482 08852 09928
TRAUMA,LARGE INTESTINE
00164 00512 01435 01436 02996* 05845* 07256 08026
08353 09398 09404
TRAUMA,LIVER
00649 00652 00656 00687 00691 00961 01069 01352
01529* 01557 01598* 01600* 01602 01607 01610 01944
02563 02584 03467 03475 03810* 03910* 04441* 04450*
04462 04551 04833 05516 05564 05565 05566 06894
07350* 07435 08050 08533 08536 08566 08568 09039
09559 09623 09627 09630 09632 09638
TRAUMA,PANCREAS
01038* 01489* 01490* 02442* 03325 03326 03341* 03910*
05394 06404 06427* 07294 07295 09433 09456 09488
TRAUMA,RECTUM
01069 03278 05350
TRAUMA,SMALL INTESTINE
00365 01038* 01069 01251* 02996* 03171 04238 04245
04254 04284 05211 07095 07107 07108 07113 07117
08026 08114* 09226 09235
TRAUMA,STOMACH
00327 01038* 01108 02009* 02081 03107* 03128 03132
06160* 07677* 08081 08762
TRIGLYCERIDE ABSORPTION
00028* 00826 01372* 03741 03886* 04673* 04719* 04854*
04859* 04939 04949 05296 05308 05551* 05850* 06815
06820 06822 06824 07188* 07439 07629* 07634* 07640*
07704* 08233* 08239
TROPICAL SPRUE
00438 01177* 01374* 02991* 03229* 03242* 05290* 06323
07192* 07194* 07198 07576 08229* 09325
TRYPSIN
04680 07416
TRYPSIN INHIBITION
01509* 02781* 03330* 03336* 03343* 03349 03787 03788
03972 04417 04418 04421 04422 04425 04960 05995*
06399* 06422* 06445 06446 06572 07295 07301* 07307*
07315 07316 07319 07320 07323 07496* 07953 08061
08395* 08404 08406 08407 08412 08659* 08659* 08972
09046 09469 09472 09484
TRYPSIN SECRETION,PANCREAS
00058 00119* 00804 00805 00836* 00860 00930* 01762*
01765* 01771* 01935* 02407* 02442* 02448 02454 02787*
02804 03330* 03343* 04389* 04412* 04684* 04851* 06429*
06924 07692* 07693* 07954 08400* 09210
TUBERCULOSIS
00243 00244 00499 01236 01592 02068 02544 02649
03089 03232* 03923* 04034* 04103 05578 06026 06036
06067 06486 06496 06547 06636 06890 07096 07228
TUBERCULOSIS
CONTINUED
07441 08099 08157 08218* 08266* 08521 08758 08808
09102 09198 09204 09329 09341 09661 09739 09933
TYPHOID FEVER
SEE ALSO SALMONELLOSIS
01945 04026* 05021 06832 06870* 06955 07317 08745
08749 09864 09975
TYPHOID FEVER CARRIER STATE
06856* 07905 08803 09848
TYPHOID FEVER COMPLICATIONS
04083 04840* 04974* 08875 09849 09951
TYPHOID FEVER DIAGNOSIS
04974* 07905 08744* 08875 09808 09951
TYPHOID FEVER EPIDEMIOLOGY
02991* 06057 08797 08803 08875 09848 09882 09883
09951
TYPHOID FEVER IMMUNOLOGY
01052 04907 07905 08736* 08787
TYPHOID FEVER IN CHILDREN
08736* 08787 08875
TYPHOID FEVER PATHOLOGY
08875 09808
TYPHOID FEVER PREVENTION
04907 08736* 09951
TYPHOID FEVER TREATMENT
04974* 06829 07560 08744* 08765 08787 08803 08875
09808 09951

ULCER COMPLICATIONS,PEPTIC
00182* 00186* 00218 00284 00297* 00387* 00395* 00400
00414 00416 01016 01032* 01036* 01051 01089 01103
01168* 01270 01297* 01302* 01306* 01307* 01312* 01321
01325 01328 01335 01342 01345 01352 01360 01363
01652* 01655* 01855* 01956 01958 02045* 02194* 02195
02198* 02201* 02203* 02205* 02224 02227 02230 02231
02232 02237 02239 02253 02257 02263 03004 03044
03182* 03202 03203 03206 03207 03408 03910* 03949
04060 04063 04073 04212 04217 04223 04255* 04260
04261* 04262* 04269* 04270* 04273 04277 04278 04284
04285 04289 04292 04295 04298 04889 04985* 04997
05076 05124 05214* 05215* 05220* 05221* 05231* 05234
05237 05240 05241 05242 05243 05244 05245 05246
05249 05251 05252 05253 05256 05259 05265 05267
05995* 06002* 06064 06069 06239* 06241* 06243* 06246
06254* 06257* 06265 06269 06272 06274 06280 06283
06290 06296 06301 06304 06309 07018* 07056 07072
07128* 07133* 07136* 07141 07159 07162 07165 07166
07178 07396 07397 07561 07562 07968 08042* 08166
08173* 08177* 08186 08189 08193 08194 08199 08202
08209 08214 08669 08700 08730* 08779 08816 08832
08836 08871 09048 09175 09186 09248 09249 09252
09258 09275 09277 09285 09286 09288 09289 09295
09296 09301 09304 09728 09812 09912 09935
ULCER DIAGNOSIS
02213* 02219* 03172 08895 09253 09255 09281
ULCER DIAGNOSIS,DUODENUM
09211
ULCER DIAGNOSIS,ENDOSCOPY IN PEPTIC
00132* 09254
ULCER DIAGNOSIS,PEPTIC
00053 00374 00382* 00404 00407 00417 00834* 00837
00922* 00956 00965 00966 01014 01081 01090 01288
01307* 01308* 01332 01357 01362 01797* 01855* 01858
01859* 01867* 02211* 02221 02222 02235 02237 02899
03099* 03182* 03215 03748* 03749* 03887* 03907* 03918
03921* 03944 03945 03996 04004 04105 04258* 04281
04291 04889 05230* 05232* 05246 05248 05261 05263
05265 05879* 05912 05945 06016 06268 06280 06281
06305 06305 06311 06313 06841* 06843* 06850* 06860
06891 06904 06918 06919 06927 06956 06979 07019
07020* 07128* 07140 07158 07163 07839* 07846* 07855
07882 07888 07935 08205 08217 08871 09045 09211
09256 09257 09260 09276 09277 09283 09285 09308
09312 09317
ULCER DIAGNOSIS,RADIOLOGY IN PEPTIC
00132* 00920* 00975 01013 02205* 03191* 04855* 05253
06151* 06251* 06253* 06294 06313 07020* 07125* 07160
07882 07910 08217 09254
ULCER DISEASE,BLOOD COAGULATION IN
02229 03203 05234
ULCER DISEASE,IMMUNOLOGY IN
02943 03109* 03111* 03113*
ULCER EPIDEMIOLOGY
02233
ULCER EPIDEMIOLOGY,PEPTIC
00414 00415 00419 01055 01081 01301* 01323 01360
01925* 02010* 02011* 02115* 02200* 02230 02235 02991
03219 04272* 04277 04280 04281 04287 04294 04634
05221* 05224* 05252 05254 05260 05264 05272 05626
06238* 06242* 06256* 06261* 06268 06753* 07133* 07134
07136* 07140 07158 07159 08166* 08171* 08194 08204
09257 09265 09299 09317

ETIOLOGY
52 00270 00310* 00385* 00401 00408 00409 00787*
35* 01311* 02213* 02214* 02219* 02244 02245 02273
35 02705* 03172 03181* 03223 03769* 04048* 04129*
72* 04190 04192 04202 04245 04257* 04259* 04271*
76 04279 04280 04287 04300 04613 05219* 05235
31* 09373
ETIOLOGY,PEPTIC
09* 00382* 00389* 00393* 00398* 00407 00410 00415
19 00861 00936* 00953* 01021* 01036* 01168* 01171*
47* 01277 01298* 01301* 01303* 01314* 01316* 01317*
41 01362 01366 01751* 01760* 01781 01899 01925*
74 02196* 02198* 02200* 02215* 02235 02240 02261
04 03119 03180* 03197 03198 03768* 03918* 04037*
00 04212 04255* 04267* 04268* 04270* 04277 04281
90 04922 04972* 05018 05028 05066 05178* 05187*
13* 05215* 05223* 05224* 05225* 05230* 05246 05250
52 05260 05261 05263 05264 05266 05271 05272
74 05626 05757* 06000* 06014 06208 06238* 06240*
42* 06243* 06244* 06245* 06247* 06248* 06250* 06256*
60* 06261* 06262* 06268 06281 06287 06288 06291
98 06309 06311 06582* 06755 07039 07127* 07130*
33* 07134* 07135* 07141 07148 07150 07151 07152
54 07159 07167 07169 07179 07275* 07396 07581
82 07583 07584 07689 08158 08165* 08167* 08168*
72* 08175* 08179 08180 08189 08190 08200 08204
05 08720* 08769 08804 08824 08826 09246 09257
58 09264 09267 09279 09280 09283 09294 09297
98 09299 09300 09301 09315 09809 09824 09828
HISTOLOGY,PEPTIC
26*
IMMUNOLOGY,PEPTIC
48*
PATHOLOGY
11* 01357 02222 02245 02258 02260 02705* 03213
19* 09253 09255
PATHOLOGY,PEPTIC
75* 01179* 01317* 02074 02271 03061 05142* 06179
13 07126* 07167 07168 07545* 07968 08207 08948
57 09262 09279 09306 09312 09316 09317 09318
PERFORATION
11*
PERFORATION,PEPTIC
76* 00400 00414 01005 01036* 01051 01103 01121
02* 01306* 01312* 01321* 01325 02195* 02209* 02224
27 02230 02231 02232 02237 02257 03182* 03207
10* 04255* 04260* 04262* 04270* 04277 04284 04289
89 05214* 05215* 05220* 05221* 05231* 05234 05237
44 05245 05246 05251 05253 05259 05995* 06239*
46* 06265 06272 06296 06301 06304 07056 07141
65 07178 08173* 08189 08194 08202 08209 08779
32 08871 09248 09249 09286 09288 09289 09301
12
SECRETION,PEPTIC
99* 00383* 03768* 05764* 07274*
SURGERY
81* 01311* 01365 01962 02255 02259 02261 02266
16 09253 09255 09281 09373
SURGERY,PEPTIC
09* 00297* 00307* 00311* 00312* 00377* 00379* 00381*
87* 00388* 00395* 00400 00405 00406 00411 00855
34* 00970 01004 01028* 01036* 01097 01126 01166*
73* 01178* 01183* 01190 01191 01192 01193 01195
00 01206 01211 01214 01260* 01278 01298* 01299*
02* 01303* 01304* 01305* 01306* 01307* 01308* 01321*
22* 01324 01325 01326 01327 01328 01329 01330
31 01333 01334 01336 01337 01338 01339 01340
43 01345 01347 01348 01349 01351 01352 01353
55 01358 01359 01362 01363 01673 01749* 01859*
56 02011* 02195* 02199* 02203* 02209* 02215* 02226
28 02231 02235 02236 02237 02259 02264 02270
63* 02780* 02793* 02909* 03009 03117* 03182* 03183*
84* 03185* 03186* 03187* 03188* 03189* 03192* 03196
97 03201 03205 03206 03207 03212 03221 03222
23 03224 03776 04040* 04051* 04085 04086 04158
66* 04178* 04181* 04194 04198 04201 04208 04210
56* 04260* 04261* 04262* 04263* 04264* 04265* 04266*
69* 04274 04275 04281 04282 04283 04292 04293
96 04297 04299 04742* 04743* 04748* 04758 04773*
31 05034 05037 05053 05130* 05152 05153 05216*
17* 05220* 05221* 05222* 05225* 05227* 05229* 05231*
33* 05234 05236 05237 05240 05244 05245 05246
47 05248 05249 05251 05253 05255 05257 05258
59 05261 05262 05265 05267 05273 05695* 05758*
40* 06018 06153* 06157* 06168 06186 06239* 06241*
46* 06248* 06251* 06254* 06263* 06264* 06265 06266
69 06270 06271 06272 06275 06277 06278 06279
80 06283 06285 06286 06288 06289 06293 06295
96 06297 06299 06300 06303 06304 06305 06306
08 06309 06310 06311 06312 06726* 06883* 06950
10* 07026* 07062 07115 07128* 07129* 07132* 07138*
39 07140 07145 07146 07147 07149 07151 07153
55 07163 07164 07166 07168 07177 07178 07329
49* 07885 08050 08082 08141 08165* 08169* 08170*
72* 08173* 08174* 08177* 08180 08181 08182 08183
84 08185 08186 08187 08188 08192 08193 08194

ULCER SURGERY,PEPTIC
CONTINUED
08197 08201 08202 08203 08205 08209 08210 08211
08214 08215 08694 08717* 08729* 08730* 08741* 08769
08779 09076 09093 09093 09200 09247 09248 09249
09256 09258 09259 09261 09266 09267 09268 09269
09271 09278 09285 09289 09291 09293 09300 09301
09304 09306 09308 09309 09313 09314 09318 09865
ULCER TREATMENT
01311* 01365 02214* 02218* 02219* 02225 02241 02243
02255 02256 02259 02266 02269 03107* 03161 03190*
03210 03212 03214 04202 04259* 05266 05275 07131*
08855 09253 09255 09281 09373
ULCER TREATMENT,PEPTIC
00109* 00309* 00313* 00377* 00379* 00384* 00386* 00388*
00390* 00391* 00392* 00393* 00394* 00396* 00397* 00405
00406 00953* 00986 01024* 01025* 01036* 01296* 01302*
01313* 01318* 01319* 01320* 01328 01344 01350 01354
01356 01758* 02195* 02197* 02202* 02204* 02205* 02206*
02223 02227 02228 02235 02237 02252 02257 02264
02894* 02897* 03184* 03202 03204 03207 03208 03221
03223 03982 04268* 04275 04276 04281 04286 04288
04290 04297 04883 04922 04982* 05005 05053 05099
05216* 05218* 05228* 05229* 05231* 05238 05239 05243
05248 05251 05254 05257 05261 05262 05263 05267
05268 05269 05270 05273 05763* 06000* 06105 06149*
06151* 06182 06211 06236* 06237* 06247* 06248* 06249*
06250* 06251* 06255* 06258* 06259* 06265 06267 06268
06269 06270 06271 06273 06274 06276 06280 06281
06284 06289 06292 06294 06297 06302 06305 06310
754* 06763 07038 07064 07115 07125* 07142 07143
07144 07148 07149 07153 07155 07156 07157 07160
07161 07163 07170 07171 07172 07173 07174 07175
07176 07177 07180 07675* 08169* 08174* 08178* 08188
08191 08194 08197 08198 08202 08209 08210 08211
08729* 08741* 08837 08871 08879 09248 09251 09254
09256 09259 09260 09261 09262 09266 09270 09273
09274 09278 09284 09285 09291 09298 09301 09303
09304 09308 09309 09311 09313 09318 09816 09842
09894 09911 09927
ULCER,CURLINGS
03182* 04100 04972* 07131* 08173*
ULCER,GASTRECTOMY-INDUCED RECURRENT
02899*
ULCERATIVE COLITIS
00497 00526 00773 01104 01115 01440 01470* 01479
01871* 02281* 02290 02339 02400 02402 02403 02404
02901* 03253* 03265* 03300* 03305 03306 03307 03308
04111 04361 05342 05342 06021 07183* 07266 07268
08246 08250* 08289 08313 08339 08758 08909 09082
09982
ULCERATIVE COLITIS COMPLICATIONS
00186* 00535 01466* 01467* 01471* 02390* 02395* 03295*
03296* 03297* 03300* 03301* 03303 04060 04370* 04376
04378 05357 05364 05365 05366 05368 05373 05375
05376 05379 06326* 06391 08254* 08288 08359* 08368
08369 09426 09429
ULCERATIVE COLITIS DIAGNOSIS
00149 00532* 00536 01453 01472* 01474 02381* 02387*
02391* 03295* 03304 03305 03997 03999 04356 04929
05366 05367 05374 05376 05378 06042 06378* 06383
06384 06388 06394 06395 06880* 06887 06921 07263*
07265 07267 07270 07895 08359* 08361 08363 08366
09083 09421 09422 09423 09424 09426 09429
ULCERATIVE COLITIS DIAGNOSIS,BIOPSY IN
01865* 04375* 06396 09423
ULCERATIVE COLITIS DIAGNOSIS,RADIOLOGY IN
00532* 01013 01464* 01472* 02382* 02383* 03888* 04370*
04375* 09423
ULCERATIVE COLITIS EPIDEMIOLOGY
01055 01481 01925* 02385* 04304 05371 05375 05378
06383 06388 08224 09424
ULCERATIVE COLITIS ETIOLOGY
00533 00537 01169* 01367* 01466* 01468* 01470* 01477
02381* 02384* 02386* 02389* 02600* 03307 04369* 04379
05066 05360* 05363* 05370 05371 05374 05376 05378
06011 06360 06376* 06377* 06379* 06381* 06384 06386
06390 06394 06395 06397 07185 07254 07261* 07262*
07264* 07270 07271 08360* 08361 08364 08367 09423
09424 09425 09426 09428 09835
ULCERATIVE COLITIS IMMUNOLOGY
06376* 06377* 06380* 06381* 06390 06394 06395 06397
07261* 07263* 07264* 09835
ULCERATIVE COLITIS IN CHILDREN
03049 03305 04371* 04379 05371 06388 09422
ULCERATIVE COLITIS PATHOLOGY
00456 00532* 00534 01451 01471* 01473* 01474 01478
01865* 02383* 02387* 02388* 02390* 02394* 02406 03301*
03304 03307 03888* 04379 04929 05361* 05362* 05367
05373 05376 06011 06326* 06366 06376* 06378* 06389
06390 06392 06396 06882* 07267 07269 07271 07557
07895 08263* 08359* 08361 08363 08364 08365 08366
08368 09422 09423 09425 09427 09428 09429

ULCERATIVE COLITIS SURGERY
01379* 01468* 01471* 01472* 01475 01480 01482 02387*
02396* 02397 02399 02401 03297* 04370* 04371* 04375*
04379 05343 05374 05377 05379 06384 06385 06388
06393 06395 07267 07273 08254* 08361 08362 08371
09421
ULCERATIVE COLITIS TREATMENT
00536 01024* 01367* 01464* 01468* 01469* 01476 01477
01925* 02276* 02381* 02386* 02392* 02393* 02395* 02396*
02397 02399 02401 02405 02894* 02976* 03294* 03299*
03305 03306 03308 04370* 04371* 04372* 04373* 04375*
04376 04377 04379 05364 05369 05372 05374 05376
05377 05378 05379 05380 06314* 06360 06377* 06382*
06384 06385 06388 06391 06392 06393 06395 07272
07273 08362 08367 08371 08820 09344 09354 09421
09424 09426 09427 09880
ULCERATIVE COLITIS,AGE FACTORS IN
09421
ULCERATIVE COLITIS,BIOCHEMICAL DIAGNOSIS OF
06380*
ULCERATIVE COLITIS,CIRCULATION IN
00535 05373 07266
ULCERATIVE COLITIS,CLINICAL STUDIES OF
00531* 00532* 00536 01469* 01481 02396*
ULCERATIVE COLITIS,DISEASES ASSOCIATED WITH
00531* 00535 01466* 01481 01482 01925* 02385* 02390*
02391* 02392* 02393* 02396* 02976* 03295* 03296* 03297*
03300* 03302 04373* 04374* 05361* 05365 05366 05375
06387 06391 07238 07265 08224 08359* 09354 09422
09430
ULCERATIVE COLITIS,DRUG TREATMENT OF
00946* 01464* 01469* 01471* 02386* 02397 03298* 04370*
04371* 04372* 04376 04377 04379 05369 05372 05374
05380 06382*
ULCERATIVE COLITIS,IMMUNOLOGY IN
00533 00537 01466* 01465* 01470* 01471* 01871* 02389*
02398 02405 04379 05363* 05370 09425
ULCERATIVE COLITIS,METABOLIC CHANGES IN
00531* 02365 02391* 05368 09429
ULCERATIVE COLITIS,MICROORGANISMS IN
01367* 01466* 02391* 02398 05363* 06377* 06381* 07262*
09423
ULCERS
SEE ALSO ANTACIDS
SEE ALSO ANTICHOLINERGIC AGENTS
SEE ALSO DUODENUM
SEE ALSO GASTRECTOMY
SEE ALSO ULCERATIVE COLITIS
00195* 00307* 00389* 00402 01982 02100 02249 02612
02912* 03066 03220 03222 03224 04172* 04982* 07131*
08021 08892 09287
ULCERS IN CHILDREN
02219* 03216 04982*
ULCERS IN CHILDREN,PEPTIC
00382* 00387* 01328 01361 01363 02225 03049 04281
06250* 06268 06272 07159 07165 07882 08194 08203
08214 08217 08729* 09301
ULCERS,AGE FACTORS IN
02225 04272* 09289
ULCERS,AGE FACTORS IN PEPTIC
01028* 01312* 01317* 01347 01354 01360 01925* 02200*
04255* 05264 06241* 06248* 06257* 07136* 07150 09295
09311
ULCERS,ANASTOMOTIC
08049
ULCERS,ANASTOMOTIC PEPTIC
01005 05213* 06248* 07128* 07160 07164 09274
ULCERS,BIOCHEMICAL DIAGNOSIS OF PEPTIC
00144 01129 03918* 08206 09077 09078
ULCERS,BLEEDING PEPTIC
00182* 00395* 00416 01016 01032* 01297* 01312* 01321*
01328 01360 01956 01958 01961 02045* 02194* 02195*
02198* 02205* 02213* 02217* 02237 02239 02263 02998*
03004 03044 03182* 03202 03203 03206 03408 03949
04063 04073 04212 04255* 04260* 04261* 04262* 04269*
04270* 04273 04277 04285 04292 04295 04298 05221*
05249 05256 05265 05267 06002* 06177 06241* 06243*
06246* 06254* 06257* 06274 06283 07133* 07396 07968
08193 08424* 08448 08779 08813 08816 08832 08836
08842 08845 09048 09252 09275 09285 09295 09296
09479 09905 09907 09908 09909 09912 09927 09935
ULCERS,CIRCULATION IN PEPTIC
00396*
ULCERS,CLINICAL STUDIES OF
00103* 00406 00409 00417 01357 01364 02222 02225
02229 02258
ULCERS,CLINICAL STUDIES OF PEPTIC
00144 00377* 00380* 00398* 00410 00412 00415 00416
02232
ULCERS,DISEASES ASSOCIATED WITH PEPTIC
00284 00385* 01034* 01168* 01242 02201* 02235 02253
02998* 03047 04280 05131* 05144* 05221* 05241 06195
06242* 06256* 06286 06290 06508 06757 07134* 07136*
07137* 07152 07154 07155 07162 07179 07180 07274*
07304* 07327 07545* 07580 07689 08166* 08167* 08171*
08179 08196 08199 09728
ULCERS,DRUG TREATMENT OF
01758* 02195* 02202* 02204* 02205* 02235 02237 02243
02252 02268 02272 03200 03208 03982 04276 04286
04288 04290 05239 05243 05269 05275 05744* 06149*
06235* 06237* 06247* 06249* 06255* 06258* 06267 06271
06284 06292 06294 06763 06766 07020* 07038 07064
07125* 07142 07143 07144 07156 07157 07160 07161
07163 07170 07171 07172 07173 07174 07175 07176
08191 08879 09250 09251 09260 09270 09273 09278
09282 09298 09307 09308 09319 09894
ULCERS,DRUG TREATMENT OF PEPTIC
03190* 03195 03198 08198
ULCERS,DRUG=INDUCED
00304* 00401 01442 01925* 01982 02196* 02207* 02235
02273 02722 03151* 03172 03200 03768* 04172* 04257*
04257* 04279 05215* 05219* 06159* 06247* 06250* 06255*
06262* 06298 06582* 06758 06766 06769* 07127* 07148
07179 07397 08059 08172* 08179 08189 08190 08791
09246 09297 09301 09894 09910
ULCERS,DUODENUM
00181* 00399 00405 01310* 01312* 01365 01768* 01962
01982 02211* 02213* 02214* 02215* 02217* 02218* 02219*
02228 02234 02238 02243 02246 02248 02250 02251
02252 02256 02266 02267 02268 02269 02905* 02982*
02998* 03107* 03185* 03188* 03189* 03190* 03191* 03201
03206 03210 03214 03216 03761* 04038* 04245 04256*
04258* 04260* 04261* 04262* 04264* 04266* 04270* 04271*
04272* 04273 04274 04276 04280 04282 04284 04288
04291 04292 04293 04297 04613 04765 04982* 05014
05018 05028 05098 05216* 05217* 05220* 05223* 05224*
05225* 05226* 05227* 05228* 05232* 05236 05237 05242
05244 05245 05250 05253 05257 05262 05764* 05840*
05863* 05879* 05994* 06008* 06149* 06150* 06177 06235*
06236* 06237* 06239* 06240* 06242* 06244* 06245* 06248*
06250* 06251* 06253* 06254* 06257* 06258* 06259* 06260*
06268 06270 06272 06284 06285 06289 06292 06302
06303 06304 06305 06308 06309 06313 06532* 06843*
06883* 07006 07010* 07062 07071 07115 07125* 07128*
07133* 07134* 07138* 07140 07149 07151 07152 07158
07162 07164 07165 07177 07178 07180 07231 07285
07397 07684 07839* 07873 08059 08073 08108 08165*
08173* 08176* 08181 08187 08196 08197 08199 08201
08203 08204 08208 08209 08210 08212 08216 08729*
08730* 08842 09045 09048 09076 09077 09078 09201
09205 09248 09249 09252 09256 09257 09258 09259
09260 09262 09263 09269 09270 09271 09273 09275
09276 09279 09280 09281 09283 09285 09287 09290
09291 09292 09293 09295 09300 09303 09307 09311
09312 09313 09317 09318 09574 09728 09865 09908
09927
ULCERS,ESOPHAGUS
00473 01021* 01946 02016* 04129* 04141 04157 05119
07131* 08011 08730* 08779
ULCERS,EXPERIMENTAL
00310* 00397* 00401 00408 00419 00868 01300* 01303*
01314* 01357 01366 02196* 02207* 02384* 02705* 02799*
03197 03198 04190 04259* 04267* 04268* 04279 04742*
05166 05213* 05215* 05275 05360* 06247* 06255* 06287
06288 06307 07127* 07135* 07580 07582 07656* 07676*
08168* 08175* 08200 09195 09264 09319
ULCERS,FREEZING TREATMENT OF PEPTIC
01356 04216 05270 06160* 09911
ULCERS,GASTRECTOMY=INDUCED PEPTIC
00377*
ULCERS,GASTRECTOMY=INDUCED RECURRENT
00132* 00312* 00400 00413 01315* 02221 03183* 03187*
03188* 03189* 03205 04204 04260* 04265* 04275 04283
04299 05233* 05255 05263 05840* 06248* 06293 06297
06310 06311 06841* 07036 07128* 07132* 07164 07177
07855* 08049 08058 08185 08730* 08769 08871 09253
09258 09284 09300 09309
ULCERS,GASTRODUODENAL
00404 02213* 02218* 02244 04279 08780 09183 09292
09297
ULCERS,HORMONAL CONTROL OF PEPTIC
00410 01319* 01344 01758* 01925* 02202* 02235 02240
04289 05264 05271 09315
ULCERS,LARGE INTESTINE
02326* 02327* 03161 03257* 04970* 06354 07131* 07210
08333 09378
ULCERS,MEDICATIONS IN TREATMENT OF PEPTIC
00380* 00391* 00392* 00394* 00404 00409 00412 01296
01300* 01318* 01319* 01320*
ULCERS,METABOLIC CHANGES IN PEPTIC
00103* 00144 00298* 01315* 01932* 02260 06743* 07016
07019* 07135* 07274* 08206 09263
ULCERS,NONSPECIFIC
01123 01257* 01442 02326* 02335 06391
ULCERS,NONSPECIFIC SMALL INTESTINE
01080 01283 01311* 02252 02914* 03151* 03162 03172
03173 04245 05219* 07114 07141 08131 09255
ULCERS,PEPTIC
00195* 00773 00843* 00846* 01100 01129 01172* 01310
01312* 01346 01932* 01982 02016* 02074 02207* 02208
02220 02238 02241 02242 02247 02249 02251 02265
02271 02957 03061 03192* 03199 03209 03219 04025

ERS,PEPTIC
NTINUED
04979* 05134* 05276 05795* 05961 06021 06238* 06251*
06252* 06282 07000 07015* 07073 07131* 07453* 08054
08102 08158 08206 08207 08207 08208 08208 08213
08217 08725* 08856 08909 09290 09305 09310 09845
09922
ERS,RECTUM
02335 08756
ERS,RECURRENT
01344 05220* 07142 08165* 09254
ERS,SIMPLE NONSPECIFIC
E NONSPECIFIC ULCERS
ERS,SMALL INTESTINE
06251* 08223 08730* 08779
ERS,STOMACH
02110 02218* 02255 02256 02256 02258 02267 02269
02272 02982* 03107* 03116 03186* 03190* 03196 03206
03212 03213 03214 03215 03218 03761* 03996 04179*
04190 04202 04276 04292 04300 04765 05220* 05226*
05232* 05235 05242 05244 05245 05250 05266 05273
05275 05764* 05863* 05879* 05894 05945 06149* 06150*
06160* 06208 06239* 06242* 06244* 06250* 06257* 06262*
06296 06298 06302 06307 06582* 06843* 07006 07010*
07017* 07018* 07020* 07062 07072 07125* 07127* 07128*
07133* 07138* 07141 07150 07167 07168 07180 07275*
07396 07656* 07676* 07873 07916 08059 08073 08089
08098 08100 08165* 08168* 08173* 08175* 08196 08197
08199 08200 08208 08209 08214 09045 09048 09076
09077 09098 09163 09186 09186 09201 09205 09212
09251 09252 09254 09257 09259 09261 09262 09263
09264 09267 09270 09272 09273 09275 09276 09277
09278 09279 09280 09281 09282 09283 09285 09286
09287 09288 09289 09290 09292 09293 09295 09301
09302 09306 09307 09308 09309 09319 09728 09908
09927 09935
ERS,STRESS FACTORS IN
01300* 01711* 01961 02213* 02214* 02244 02245 02251
02254 02273 02705* 02722 03198 03200 04100 04190
04259* 04267* 04279 04284 04290 04300 05261 05264
05266 06251* 06291 07017* 07130* 07131* 07150 07466*
08175* 08179 08180 09195 09294
RASOUND IN DIAGNOSIS
04844* 04888 04901 04906 05914 06837* 06925
RASTRUCTURE
08531
RASTRUCTURE,ANUS
07605
RASTRUCTURE,BILIARY TRACT
05703 09789
RASTRUCTURE,DUODENUM
03707* 07614 08137 08146
RASTRUCTURE,ESOPHAGUS
07610
RASTRUCTURE,GALLBLADDER
05643* 06671* 08704
RASTRUCTURE,GASTROINTESTINAL
02163 02295 02328* 02740 07073 07617 07670 07818
08863 09019
RASTRUCTURE,LARGE INTESTINE
04363 05367 05690* 06688 07210* 08246 08299 08934
09357 09920
RASTRUCTURE,LIVER
02538 02594 02735* 03378* 03387* 03447 03456 03488*
03505 03516 03517 03522* 03524* 03568 03592* 03620
03696* 03698* 03706* 03709* 03716 03719 03813* 03834
03865 03866 04432* 04460* 04473 04516* 04519* 04555
04633* 04690* 04700 04793* 04803* 04809* 04834 04835
05447* 05449* 05453* 05456* 05460 05472 05481 05484
05542* 05544* 05545* 05574* 05575* 05581 05669* 05680*
05694* 05699* 05704 05820* 05827 05834 05954 06455*
06525 06534* 06551 06623* 06682 06683 06692 06697
06780* 06784* 06818 06819 06820 06821 06823 07017*
07187* 07334* 07335* 07420* 07421* 07430 07432 07436
07438 07445* 07455* 07477 07598* 07607 07615 07618
07619 07708* 07718* 07721* 07721* 07758 07765 07767
07769 07775 07777 07782 07784 07790 07793 07795
07809 07809 08430* 08431* 08449 08451 08460 08497
08539* 08542* 08543* 08545* 08546* 08547* 08551* 08552*
08554* 08557* 08574 08591 08595* 08615 08921 08922
08933 08958 08965 08966 08968 08969 08974 08983
08999 09000 09001 09006 09011 09492 09555 09556
09563 09566 09582 09595 09601 09612 09642 09648
09650 09656 09658 09660 09687 09697 09698 09789
RASTRUCTURE,PANCREAS
01500 02427 02430 02440 03314* 03696* 03699* 03704*
04399 04692* 05708 06414 07665* 08720* 08780 08924
08925
RASTRUCTURE,PERITONEUM
04984*
RASTRUCTURE,RECTUM
08298 08338
RASTRUCTURE,SALIVARY GLAND
01110 05381* 07665*
ULTRASTRUCTURE,SMALL INTESTINE
02193 03707* 03718 03989 04226* 04229* 04234 04710*
04868 05184* 05196 05285* 05687* 05691* 05707 06215*
06686 06687 06691 06692 06881* 07079* 07080* 07082*
07187* 07196 07597* 07602 07609 07611 07612 07629*
07819 07832 08119* 08134 08135 08139 08223 08233*
08243 08246 08926 08934 09330 09920
ULTRASTRUCTURE,STOMACH
02057* 03697* 04706 04761 05155 05683* 05684* 06673*
07017* 07613 07665* 08068 08931 09206
UREA METABOLISM,LIVER
01829* 01830* 02523 02549 03870 04064 04546 04620
04676* 04794* 04812 04977* 05810* 07700* 08971

VAGOTOMY
00002* 00059 00309* 00313* 00324 00379* 00386* 00395*
00772 00787* 00791 00792 00841* 00842* 00855 00866
00934* 01004 01038* 01161 01211 01298* 01299* 01302*
01303* 01304* 01305* 01306* 01307* 01308* 01322* 01324
01326 01333 01337 01338 01339 01358 01359 01362
01703* 01711* 01749* 01763* 01768* 01774* 01778* 01806*
01946 02011* 02045* 02102 02103 02194* 02196* 02199*
02209* 02210* 02212* 02217* 02226 02235 02236 02237
02243 02250 02256 02407* 02663* 02779* 02793* 02800*
02899* 02909* 02919* 02930 03004 03102* 03117* 03148*
03166 03180* 03182* 03183* 03185* 03187* 03188* 03189*
03192* 03197 03211 03221 03757* 03776 03784 03794*
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04177* 04256* 04260* 04261* 04262* 04263* 04264* 04265*
04266* 04274 04275 04280 04282 04283 04292 04293
04296 04297 04298 04390* 04730* 04742* 04743* 04748*
04751* 04758 04773* 04869 05086* 05152 05153 05169
05216* 05217* 05221* 05227* 05229* 05236 05237 05240
05247 05250 05253 05255 05256 05257 05258 05262
05273 05354 05668* 05753* 05756* 05758* 05760* 05763*
05840* 05928 06029 06052 06122 06201 06239* 06246*
06248* 06254* 06258* 06259* 06263* 06264* 06265 06270
06275 06279 06283 06285 06288 06289 06292 06295
06297 06298 06299 06300 06308 06309 06310 06312
06667* 06718* 06726* 06741* 06834 06883* 06950 07014*
07027* 07043 07115 07128* 07129* 07130* 07139 07145
07151 07153 07163 07166 07177 07178 07180 07275*
07604 07657* 07675* 07680* 07684 07685 07686 08035*
08079 08141 08165* 08169* 08172* 08174* 08177* 08183
08187. 08188 08192 08193 08197 08201 08202 08203
08210 08214 08215 08216 08439* 08443* 08835 08854
09076 09139 09167 09248 09256 09258 09259 09261
09269 09271 09274 09278 09291 09304 09309 09313
09865
VALVE,ILEOCECAL
00009* 00498 01292 02142 03174 03795* 06316* 06944
09926
VARICES,ESOPHAGUS
00226 00264* 00268 00281 00282 00370 00607* 00620
00942* 01011 01034* 01035* 01057 01112 01596 01642*
01645 01926* 01930* 01934* 02010* 02032 02033 02036
02473* 02484* 02628* 02645 02957 02994* 03046 03049
03073* 03083 03096 03355* 03413 03936 03949 04063
04073 04128* 04137 04285 04490 04598* 04599* 04603*
04606* 04616 04617 04621 04846* 04916 04926 05063
05099 05100 05511 05513 05602* 05603* 05607* 05617
05629 05897 05994* 06002* 06467* 06579* 06583* 06596
06597 06601 06610 06840* 06848* 06960 06968* 06970*
06973* 06980 06992 06995 07046 07341* 07368 07451*
07464* 07465* 07468* 07470* 07858* 07889 07900 07984*
07996* 07998 08013 08387 08428* 08448 08471 08473
08482 08526 08530 08632* 08642 08645 08729* 08813
08816 08836 08845 08900 09060 09098 09130 09236
09310 09479 09496 09539 09565 09613 09703 09708
09724 09726 09730
VARICES,GASTROINTESTINAL
08817
VARICES,STOMACH
01035* 01117 01926* 01952 05063 05902 06203 06579*
07889 07900 07998 08729* 09185
VASCULAR DISEASES
08777
VASCULAR DISEASES,MESENTERY
00973 01028* 01248* 01268 01927* 01929* 01953 02997*
04096 04240 04849* 04938 04995 05040 05063 05064
05065 05201 05518 06023 06386 06886 06896 07084*
07115 07553 07554 07555 07556 07887 08154 08738*
08763 08831 08850 08853 08911 09091 09232 09428
09493 09857 09888 09922
VASCULAR SURGERY
00221 05065 06594 07353 07359 07473 07554 08154
08180 08496 09060 09517 09523
VILLOUS GASTROINTESTINAL NEOPLASMS
00461 00524
VILLOUS LARGE INTESTINE NEOPLASMS
00446* 01049 01144 02320* 02325* 02337 04334* 05319*
06330* 06357 06949 07242 08317 09399
VILLOUS RECTUM NEOPLASMS
00440* 00443* 00444* 00446* 00457 00525 02337 02363
05319* 05351 06341 09199

VILLOUS SMALL INTESTINE NEOPLASMS
04042*
VILLOUS STOMACH NEOPLASMS
09199
VIRAL HEPATITIS
00696* 00697* 00707 00708 00728 00773 01530* 01611*
01614* 01618* 01627 01632 01659* 01720* 01875* 02492
02609 02611 02615 02616 02617 02620 02622 02625
02684* 03378* 03440 03518* 03537 03561 03568 03569
03577 03580 03582 03583 03835 04009 04561* 04576
05440* 05475 05481 05533* 06461* 07450* 07778 07891
08500 08601* 08617 08618 08620 08621 08623 08625
08626 08627 09079 09546 09688 09689 09691
VIRAL HEPATITIS COMPLICATIONS
06556* 06559* 06571 06865* 09574
VIRAL HEPATITIS DIAGNOSIS
00107* 00711 00712 00713 00715 00721 00725 00727
01555 01611* 01613* 01615* 01616* 01629 01631 01852*
01863* 01886 01894 02598* 02606 02608 02959 02967
03521* 03523* 03531 03532 03533 03536 03538 03548
03549 03550 03553 03555 03557 03559 03560 03562
03575 03919* 03976 04559* 04562* 04577 04581 04582
04583 04872 04905 05572* 05579 05587 05588 05591
05837* 05870* 06465* 06554* 06555* 06557* 06561 06562
06573 06852* 06854* 06865* 06867* 06868* 06869* 06939
06957 07278* 07442* 07444* 07454* 07457 07459 07840
07867* 07883 07890 07893 07904 08474 08523 08592*
08593* 08595* 08598* 08610 08612 09029 09046 09081
09089 09664 09667 09673 09677 09684 09685 09686
09691 09693 09695
VIRAL HEPATITIS EPIDEMIOLOGY
00603* 00695* 00705 01613* 01615* 01617* 01623 01624
01628 01629 01630 01631 02598* 02599* 02604* 02619
02624 02959 02991* 03383* 03519* 03547 03551 03559
03566 03567 03583 03919* 04565* 04566* 04567* 04575
04577 04579 04582 04583 04585 04589 05462 05569*
05572* 05577 05580 05588 05591 06554* 06560 06563
06566 06570 06573 06574 07418* 07447* 07452* 07457
07461 07463 07890 08592* 08596* 08599* 08599* 08600*
08602* 08608 08611 08613 08622 08624 08629 08630
09089 09665 09666 09672 09685 09690 09700
VIRAL HEPATITIS ETIOLOGY
07890
VIRAL HEPATITIS IMMUNOLOGY
00723 01613* 01626 01630 01875* 02611 03521* 03525*
03533 03551 03553 03567 03914* 04562* 04569* 04577
05590 05870* 06554* 06868* 07411 07443* 08598* 08599*
08610 09029 09665 09666 09678 09684 09685 09690
VIRAL HEPATITIS IN CHILDREN
01613* 01615* 01617* 01622 02601* 02604* 02619 02631*
03049 03527* 03531 03539 03547 03548 03554 03558
03564 03573 03574 03575 03576 03577 03578 03582
04566* 04579 04583 04589 05576* 05577 05584 06556*
06560 06562 06563 06575 06576 06577 06852* 06854*
06869* 07278* 07442* 07444* 07448* 07452* 07459 07462
07484 07883 08463 08524 08593* 08594* 08597* 08598*
08599* 08600* 08602* 08604* 08605 08606 08607 08609
08610 08616 08619 08629 08630 09540 09664 09670
09676 09677 09679 09690 09695 09697 09700
VIRAL HEPATITIS PATHOLOGY
05442* 05543* 05574* 05575* 05582 06556* 06561 06572
06865* 07134* 07442* 07445* 07458 08595* 08615 08616
08621 09669 09671 09673 09679 09681 09682 09684
09686 09698
VIRAL HEPATITIS PROPHYLAXIS
00718 01613* 01615* 01623 01624 01628 01629 01630
01631 02598* 03519* 03525* 03529* 03552 03569 04566*
04577 04579 05572* 05590 06563 07461 08611 08621
08629 09665 09666
VIRAL HEPATITIS TREATMENT
00700 00706 00711 00713 00714 00716 00720 01568
01596 01613* 01615* 01621 01622 01624 01631 02604*
02605* 02621 03383* 03523* 03535 03538 03539 03540
03541 03543 03544 03545 03555 03556 03559 03564
03565 03576 03582 04577 04578 04584 04586 05573*
05576* 05578 05579 05587 05588 05590 05644* 06495
06556* 06560 06562 06564 06565 06567 06575 06576
06577 06865* 06867* 07278* 07462 07963 08594* 08598*
08606 08607 08619 08621 08625 08626 08627 09540
09668 09671 09674 09677 09680 09685 09691 09695
09699
VIRAL HEPATITIS,ACUTE
00603* 00704 00720 00726 00927* 01612* 01621 02603*
02614 03356* 03383* 03408 03416 03520* 03521* 03530
03555 03557 03563 03576 04442* 04556 04557* 04558*
04578 04580 04584 04586 05534* 05571* 05581 05583
05586 05588 05589 06556* 06558* 06567 06568 06569
06571 06572 06579* 06787* 06957 07443* 07454* 07455*
07456 07963 08463 09610 09676 09681
VIRAL HEPATITIS,CHRONIC
02604* 04443* 04444* 05571* 05585 05587 06558* 07454*
09573 09681 09687
VIRAL HEPATITIS,DISEASES ASSOCIATED WITH
00720 02615 02620 02625 03402 03527* 03554 03580
03590* 04559* 04580 04590 04594 05488 05570* 05573*
05578 05584 05587 05589 06430 06553* 06558* 06559*
VIRAL HEPATITIS,DISEASES ASSOCIATED WITH
CONTINUED
06564 06566 06570 06573 06575 07446* 07449* 07451*
07453* 07454* 07458 07460 07462 07484 08609 08613
08614 08618 09675 09682 09684
VIRAL HEPATITIS,METABOLIC CHANGES IN
09491 09668 09669
VITAMIN A ABSORPTION
SEE ALSO CAROTENE ABSORPTION
02283* 04310* 04473 05779* 06735* 06813 07635* 07644*
07774 08229* 08377 08891
VITAMIN ABSORPTION
00036 00311* 00321 00430* 00816* 00822 00825 00845*
01230 01375 01856* 02170 02171 02283* 02289 02291
02291 02664* 02749* 02761* 03104* 03109* 03230* 03237*
03242* 03247 03500 03589* 03688 03737 04060 04213
04219 04221 04240 04242 04301* 04305* 04306* 04310*
04311 04323 04710* 04724* 04850* 04950 05024 05031
05145* 05285* 05287* 05301 05302 05302 05382* 05392
05710* 05730 05779* 05867* 06029 06707* 06844* 07031*
07190* 07194* 07197 07199 07201 07635* 07644* 08041*
08077 08116* 08229* 08230* 08242 08377 08940 08961
09082 09176 09327 09329 09330 09926
VITAMIN B COMPLEX
00901 01080 05463 06808 07404 07687 07780 08891
VITAMIN B12
00308* 00320 01170* 01863* 02813 03000* 03415 04031*
04087 04091 04173* 05145* 05875* 07194* 07196
VITAMIN B12 ABSORPTION
00036 00311* 00321 00430* 00822 00825 00828 00829
00845* 01230 01375 01856* 02170 02171 02289 02664*
02749* 02761* 03104* 03109* 03230* 03237* 03247 03589*
03737 04213 04242 04310* 04311 04323 04710* 04850*
04950 05031 05285* 05392 05710* 05730 05867* 06707*
06844* 07031* 07190* 07199 07201 07594* 08077 08229*
08230* 08242 08483 08746* 08940 09176 09329 09429
09926
VITAMIN B12 IN LIVER
02739 03851 06791*
VITAMIN B12 IN MALABSORPTION TREATMENT
04201 08746*
VITAMIN C
00904 02756* 02864 03857 03858 04025* 04168* 05179*
06267 06778* 06809 08741* 08891 08900
VITAMIN C ABSORPTION
04060 04724* 05024
VITAMIN D
01842 04941 05071 05306 07289 07597* 07632* 08041*
08103 08891
VITAMIN D ABSORPTION
02285* 03688 04219 04221 08103
VITAMIN DEFICIENCIES
07768 08593*
VITAMIN DEFICIENCIES IN MALABSORPTION
00425* 00427* 00543 04046* 04091 04201 04204 04321
04941 05144* 05145* 05179* 05301 05309 05867* 06808
07190* 07192* 07194* 07201 08242 08248 08377
VITAMIN E
00427* 00543 04046* 04535 05179* 05464 07143 07646
VITAMIN K
00112* 03868 07664 07963
VITAMIN METABOLISM
05454*
VITAMIN METABOLISM,LIVER
05815* 07774
VITAMINS
SEE ALSO ABSORPTION
SEE ALSO LIVER
SEE ALSO MALABSORPTION
00831* 01643 02402 03668* 03812* 03933 04091 04305*
04306* 04815 06517 07686 07792 07793 09084 09264
09916
VOLVULUS
01042* 08021
VOLVULUS SURGERY
04653 06343 07074 07103 07241 08264* 08357 09383
VOLVULUS,GALLBLADDER
04645 07504
VOLVULUS,LARGE INTESTINE
00445* 01125 01463 01493* 02300* 02334 02351 03268
04339 04351 05311* 06026 06343 07241 07259 08251*
08264* 08357 09383 09403 09409
VOLVULUS,SMALL INTESTINE
00326 01125 03058 04322 06226 07098 08346 08776
08835 09219
VOLVULUS,STOMACH
04043* 04197 05171 06206 07074 08001
VOMITING
00197* 01322* 01950 01972 02105 02114* 03288 04052*
04057 04169* 05187* 05199 05787* 05788* 05946 06019
06051 06091 06107 06120 06431 07121 07600 07918
07994* 08008 08019 08107 08154 08195 08751 08883
09139 09181 09192 09277 09460 09602 09859 09899

OMITING IN CHILDREN
07902
ON WILLEBRANDS DISEASE
08855

ATER ABSORPTION
00814* 00815* 00817* 00818* 00823 01412 01723* 01803*
02170 02290 02320* 02750* 02753* 02758* 02774 02777
02829* 02893* 03730* 03745 04343 04711* 04713* 04716*
05185* 05724* 06701* 06710 07197 07216* 07575 07622*
07623* 07624* 07627* 07633* 08231* 08255* 09954
ATER METABOLISM IN CIRRHOSIS
02512 02658 03617 05609* 05613 08634*
ATER METABOLISM,GASTROINTESTINAL
00441* 01049 02129* 02290 02730* 02924 02978* 03041
03774 03955 04070 04180* 04376 04863* 04981* 05604*
06017 06020 07065 07124 07139 07543* 07633*
HIPPLES DISEASE
01451 02352 02989* 03986 04003 04061 04319 04326
05070 05184* 05690* 06325 07089 07196 08233* 08235
08246 08338 09335 09338
ILLEBRANDS DISEASE,VON
08855
ILSONS DISEASE
SEE HEPATOLENTICULAR DEGENERATION

YLOSE ABSORPTION
00025* 02283* 02284* 04091 05309 05722* 05985* 06701*
07112 07446* 09083
YLOSE ABSORPTION,D
09242
YLOSE IN MALABSORPTION DIAGNOSIS,D
00926* 04000 04314 04959 05309 05867* 06836* 08229*
08247 09074 09083 09323 09326 09327

IEVES SYNDROME
00648 00738 02470* 04458* 05432* 06501 07480 08411
INC ABSORPTION
01640* 05291* 08236 08942 09491 09709
INC METABOLISM
07281
INC METABOLISM,LIVER
01640* 02474* 02630* 09709
OLLINGER-ELLISON SYNDROME
00188* 00195* 00541* 00815* 00970 01021* 01049 01051
01090 01092 01121 01485* 01487* 01890 01899 02053*
02235 02237 02290 02988* 02993* 03004 03052 03068
03784 03918* 04037* 04039* 04048* 04049* 04167* 04290
04755 04855* 05018 05030 05031 05034 05037 05053
05071 05075 05076 05078 05764* 05928 06000* 06014
06016 06018 06251* 06902 07036 07160 07275* 07297
07581 07582 07583 07584 07835 07862* 07954 08720*
08730* 08768 08769 08779 08780 08824 08826 08856

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00147 00686* 04909 04911
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